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The Burden of Exposure–Related Diffuse Lung Disease

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Abstract

Estimating the burden of exposure-related diffuse lung disease in terms of health effects and economic burden remains challenging. Labor statistics are inadequate to define the scope of the problem, and few studies have analyzed the prevalence of exposure-related illnesses and the subsequent health care cost. Well-defined exposures, such as those associated with coal mines, asbestos mines, and stonecutting, have led to more accurate assessment of prevalence and cost. As governmental regulation of workplace exposure has increased, the prevalence of diseases such as silicosis and coal workers' pneumoconiosis has diminished. However, the health and economic effects of diseases with long latency periods, such as asbestosis and mesothelioma, continue to increase in the short term. Newer exposures, such as those related to air pollution, nylon flock, and the World Trade Center collapse, have added to these costs. As a result, estimates of cost for occupational diseases, including respiratory illnesses, exceed \$26 billion annually, and the true economic burden is likely much higher.

Keywords

Burden; occupational exposure; pneumoconiosis; interstitial lung disease

Risk of occupational injury or illness exists for virtually every job held by the American workforce, an estimated nearly 140 million people.¹ However, despite a large at-risk population, few published studies have tried to estimate the incidence of these injuries and illnesses or their ultimate cost in terms of health expenditure and lost revenue. Exposure-related diffuse lung disease has long been recognized as an occupational hazard of specific trades: coal mining, asbestos mining, manufacturing, construction, shipyard work, stonecutting, farming, and the like. (Table 1). Awareness of the dangers of exposure has prompted agencies such as the Occupational Safety and Health Administration (OSHA) to mandate the use of personal protective equipment and set limits on acceptable exposure levels. As a result of these changes, exposure-related health effects have become less prevalent, thereby reducing the financial burden in terms of disability and health care costs. However, environmental exposure via particulate air pollution continues to plague the U.S. health care system by causing significant morbidity. Previously unrecognized agents are also emerging as potential causes of exposure-related diffuse lung disease, such as nylon flock worker's lung or diacetyl-associated popcorn-worker lung. Unprecedented massive exposure to workers and residents alike after the catastrophic World Trade Center bombing left us with health consequences that are only now being recognized.

ESTIMATES OF ECONOMIC COST

Occupational exposure-related injuries and illnesses create significant morbidity and mortality among the United States' 139 million workers. In 2005, the Bureau of Labor Statistics (BLS) documented 5734 fatal work injuries and 4.2 million nonfatal injuries; 1.2 million of these cases required days away from work.² The economic impact was compounded by both missed production days and needs for social insurance, such as workers' compensation. Workers' compensation programs in the 50 states, the District of Columbia, and federal programs paid over \$53 billion to injured workers in 2002, up from \$50 billion in 2001. Employer costs were nearly \$73 billion. In contrast, Social Security and Medicare pay benefits to workers with long-term disabilities of any cause, when those disabilities preclude work. The Social Security Administration paid an additional \$65 billion to disabled workers in 2002, and Medicare paid \$33 billion for health care for disabled persons under age 65.³ Using data from 1992, Leigh et al culled information from 14 primary sources (e.g., census data, U.S. Bureau of Labor Statistics) and over 200 secondary sources (e.g., estimates in the literature) from private and public agencies to attempt to quantify the economic effects of occupational injury and illness. This analysis estimated the total costs of occupational injury and illness to be over \$171 billion annually, including direct costs of medical expense and workers' compensation as well as indirect costs such as productivity losses and higher consumer prices. Nearly 1 million reported occupational exposure-related illnesses, including respiratory diseases, cost an estimated \$26 billion annually.^{1,4}

LUNG CANCER

Methods have been developed to estimate disease burdens from specific occupational exposure, as well as to determine the contribution of certain exposures to the development of cancer.⁴ One cohort study analyzed eight known lung carcinogens caused by occupational exposure to determine the relative risk from these agents. The combined relative risk of lung cancer for the eight carcinogens ranged from 1.31 to 3.69. These occupational carcinogen exposures could account for up to 10,000 cases of lung cancer in U.S. men and 1900 cases in U.S. women annually. These cases represent 9% of all U.S. lung cancer deaths in men and 2% in women annually.⁵ Globally, 20 to 30% of the male and 5 to 20% of the female working-age population have work-related exposure to lung carcinogens, including asbestos, arsenic, beryllium, cadmium, chromium, diesel, nickel, and silica⁶ (Fig. 1). The worldwide attributable fraction of lung cancer due to occupational exposure is similarly estimated to be 10% for men and 5% for women.⁷

REACTIVE AIRWAYS DISEASE

Occupational exposures contribute significantly to non-malignant respiratory disease, particularly diseases of the airways such as asthma and chronic obstructive pulmonary disease (COPD). Occupational asthma is defined as variable airflow limitation or bronchial hyper-reactivity caused by particular materials specifically found in the workplace environment.⁸ Other exposures, such as organic dust, can mimic occupational asthma with the development of reactive airways dysfunction syndrome. Occupational asthma is the most common form of occupational lung disease in the developed world, accounting for 1 in 10 cases of adult-onset asthma. More than 250 agents have been identified that cause sensitization and subsequent occupational asthma. The most frequently noted include inorganic and organic dusts, grains, fur, fungi, wood, metals, welding fumes, and so forth.

Models estimating burden must take into account multiple simultaneous or sequential exposures, duration of exposure, and latency period to disease state. One such model estimates that globally, 38,000 deaths annually and 1.6 million disability adjusted life-years

(DALYs) result from occupational asthma. The attributable fraction of asthma mortality from occupational exposure ranges from 11 to 20% worldwide, with developing countries shouldering the highest burden. COPD associated with workplace exposures (particularly toxic fumes and secondhand smoke) causes an estimated 318,000 deaths and 3.7 million DALYs annually around the world; the attributable fraction was 12%⁹ (Figs. 2 and 3). Certain industries and regions have markedly higher prevalence of occupational asthma. Western red cedar is a powerful airway irritant; in British Columbia, where these trees are cut and processed, workers have a much higher rate of occupational asthma (occupational asthma accounts for 52% of all respiratory disease).⁹

ASBESTOS-RELATED LUNG DISEASE

Asbestos exposure is linked to malignant mesothelioma, lung cancer, pleural disease, and pulmonary asbestosis.¹⁰ Benign pleural effusions are often the initial clinical manifestation of asbestos exposure, followed by pleural plaques, interstitial fibrosis, bronchogenic carcinoma, and malignant mesothelioma. However, any of these presentations may be the first or only manifestation. The clinical effects of asbestos exposure depend on cumulative dose and latency period, often in excess of 30 to 40 years from initial exposure.^{11,12} In the early 1980s, labor union campaigns and government regulating agencies curtailed the industrial use of asbestos, effectively limiting exposure in the workplace.

Between 1990 and 1999, there were over 10,000 occupational asbestosis deaths reported by the National Institute for Occupational Safety and Health (NIOSH), accounting for one third of all pneumoconiosis deaths and displacing coal workers' pneumoconiosis as the most frequent type of pneumoconiosis death. States with the highest age-adjusted asbestosis-associated death rates include Delaware, West Virginia, Mississippi, Washington, and New Jersey (Fig. 4). The construction industry accounted for 25% of these deaths; other affected industries included plumbers, pipefitters, and steamfitters. Industries with the highest cumulative exposure (manufacturing workers, shipbuilders, boiler-makers, insulation workers) had proportionate asbestosis mortality 15 to 20 times higher than all other industries. In addition to excess mortality, asbestos exposure was associated with 2686 malignant mesothelioma deaths in 2004 alone.¹³ Due to the long latency period for asbestos-related lung disease, the peak incidence of asbestosis and malignant mesothelioma is expected in the years 2010 to 2030,¹⁴ despite the declining exposure since 1979 due to OSHA regulation.¹⁵ A study of 18,211 U.S. sheet metal workers between 1986 and 2004 found a lower prevalence of nonmalignant asbestos-related disease among those with first exposure after 1970, likely reflecting government regulation, when compared with workers with first exposure before 1949.¹⁶ However, the NIOSH data show that as the cohort of early- and heavily exposed asbestos workers ages, the latency period increases, leading to an increased prevalence of lung disease (Fig. 5). Patterns of disease may also be changing because asbestos-related lung disease causing obstructive rather than restrictive dysfunction appears more frequently in those who smoke. In a cohort of 3383 asbestos-exposed workers referred for medical evaluation, 42% had normal pulmonary function, 25% had obstructive dysfunction, 19% had restrictive dysfunction, and 6% showed a mixed pattern.¹⁷

As workplace asbestos exposure waned, attention turned toward risk among community members in heavily asbestos-exposed industries. The Environmental Protection Agency (EPA) developed a model to study exposure risk to women living in a chrysotile asbestos-mining town in Quebec between 1970 and 1989. The model incorporated measurements of ambient asbestos dust and asbestos fibers and extrapolated known occupational risks to populations with environmental exposure. Seventy percent of the women in the study lived in a household with at least one asbestos worker; only 5% of the women had worked in the asbestos industry or had direct occupational contact with asbestos. No measurable excess

lung cancer mortality was noted in this study, but there were significant excess numbers of death among nonminers due to pleural cancer and asbestosis compared with a reference population.¹³

Environmental asbestos risk is clearly illustrated in the case of Libby, Montana. A working-class town of ~10,000 residents, Libby's primary industry from the 1920s to the 1990s was vermiculite mining. Libby's vermiculite mines provided 80% of the world's supply of vermiculite, an ore used in cinder blocks, wallboard, and insulation. In the 1970s and 1980s, the medical community noted elevated rates of asbestos-related lung diseases among Libby miners. The EPA and Agency for Toxic Substances and Disease Registry (ATSDR) became involved in the investigation in the late 1990s and determined that the vermiculite was contaminated with tremolite, actinolite, and other forms of the amphibole series of asbestos ("Libby asbestos"). Despite the closing of the mine in 1990, significant community exposure remained in the form of contaminated building materials, insulation, waste rock, and soil. The ATSDR reviewed Libby's mortality data and found that for the period 1979 to 1998, Libby had a 40- to 80-fold higher asbestosis mortality than expected, and 20 to 30% higher lung cancer mortality than expected. Nearly 7000 residents underwent medical screening, including chest radiographs; 18% of the general population and 51% of former mine workers had pleural abnormalities.^{18,19} In 2003, the U.S. District Court of Montana ordered the owners of the vermiculite mine to reimburse the federal government \$54 million for the costs of investigation and cleanup of asbestos contamination.

SILICOSIS

Silicosis is a preventable occupational disease caused by the inhalation of crystalline silica dust that over several months can lead to silicosis, lung cancer, and COPD; silicosis has also been associated with higher rates of tuberculosis and autoimmune disease.²⁰ The dangers of silica came to light in the 1930s during the excavation of Hawk's Nest Tunnel in West Virginia. Nearly 800 workers out of 5000 died from silicosis. By 1937, 46 states had passed laws regulating occupational silica exposure. Between 1968 and 2002, a total of 16,305 death certificates listed silicosis as an underlying or contributing cause of death. The age-adjusted mortality rate has dropped from 1157 to 148 from 1968 to 2002, with an average of 30 deaths per year since 1995²¹ (Fig. 6).

In 1991, NIOSH estimated that at least 1.7 million U.S. workers were potentially exposed to respirable crystalline silica through various industries, including construction, sandblasting, tunneling, foundry work, and mining. These data were based on survey data from the National Occupational Exposure Survey in 1983 and Bureau of the Census County Business Patterns 1986.²² Considerable progress has been made toward the elimination of silicosis via industry regulation and engineered exposure control; however, overexposure remains widespread, particularly in masonry and foundry workers. OSHA air-sampling data from suspected high-exposure industries suggest that 121,000 workers were exposed to the NIOSH recommended exposure limit (REL), and 23,000 were exposed to 10 times the maximum REL.²³ Other high-risk industries include Italian jewelry makers, Chinese tin and tungsten miners, Brazilian stone carvers, and Norwegian slate workers, all of whom have demonstrated an increased prevalence of silicosis on high-resolution chest computed tomography (CT).²⁴⁻²⁷ In the United States, the highest mortality rates from silicosis occurred in construction, metal mining, coal mining, and steelworks.¹⁶ States with the highest age-adjusted death rates include West Virginia, Utah, Colorado, Pennsylvania, and New Mexico (Fig. 7).

COAL WORKERS' PNEUMOCONIOSIS

“Black lung,” or coal workers’ pneumoconiosis (CWP), once accounted for nearly half of all pneumoconiosis-related deaths. There has been a steady decline in the mortality rate attributable to CWP, from over 2500 deaths annually in the early 1980s to 1000 deaths in the late 1990s (Fig. 8). The bulk of those deaths occurred in Pennsylvania, Kentucky, West Virginia, Virginia, and Ohio (Fig. 9). The prevalence of radiographically evident CWP among active underground miners continues to decline, down from over 10% 30 years ago, to less than 2% now.¹⁶ Despite a decline in morbidity and mortality, coal dust exposure continues to impact health and the economy. Federal Black Lung Benefits are administered through the Division of Coal Mine Workers’ Compensation in the US Department of Labor. Over 102,000 beneficiaries and 18,000 dependents received benefits in excess of \$700 million in 2004.²⁸

AIR POLLUTION

The World Health Organization (WHO) 2002 report on worldwide major health risks considers urban air pollution as one of the largest worldwide health threats of the twenty-first century. Air pollution is the result of the combustion of fossil fuels for transportation and power generation; the combustion process creates a complex mixture of pollutants including primary emissions as well as products of atmospheric transformation (e.g., ozone). Six major classes of pollutants have been described: airborne particulate matter, ozone, sulfur dioxide, nitrogen oxides, carbon monoxide, and all others. Particulate matter appears to be the primary constituent associated with lung disease, including cancer. A study of air pollution in 20 US cities between 1987 and 1994 demonstrated excess cardiorespiratory mortality in cities with elevated ambient levels of coarse particulate matter less than 10 μm in aerodynamic diameter (PM_{10}); for each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} level, the relative death rates increased by 0.68%.²⁹ Ambient air pollution is estimated to cause 5% of trachea, bronchus, and lung cancer; 2% of cardiorespiratory mortality; and 1% of respiratory infections mortality worldwide. These cases amount to nearly 1 million deaths and 7.9 million DALYs.³⁰ Ozone has also been shown to have a significant dose-related mortality effect even after short-term exposure. Bell et al estimated that a 10 parts per billion (ppb) increase in the previous week’s ozone level would cause an additional 319 annual premature deaths in New York City and nearly 4000 premature deaths annually among 95 urban communities.³¹ Further analyses suggest that ozone’s mortality effect is separate from the effect of particulate matter on mortality.³²

EMERGING THREATS

Nylon Flock-Workers’ Lung Disease

Flocking is a process whereby continuous synthetic fiber (primarily nylon, but also rayon or polyester) is cut into shorter pieces and applied to an adhesive-coated fabric to create a plush material that is used for clothing or upholstery. The cut fibers are not a respirable size (0.2 to 5.0 mm), but smaller particles that are created during the processing of nylon flock are. These small nylon shards are thought to cause a nonspecific interstitial pneumonia (NSIP)-like disease.

The disease first came to attention in 1990–91, when five workers from an Ontario nylon flock processing plant developed progressive cough and dyspnea.³³ Four more workers from the same plant later developed the disease, a total of nine cases among 90 workers. In 1995, two nylon flock workers in Rhode Island developed similar symptoms of cough and dyspnea initially attributed to hypersensitivity pneumonitis. Further investigation by the Brown University Program in Occupational Medicine and NIOSH revealed seven cases among 165

workers, and aggregate biopsy results suggested that this was a new entity rather than hypersensitivity pneumonitis.^{34,35} In 1996, two more workers from two different nylon flock processing plants in Massachusetts presented with similar findings. Five more cases were described in 2000, four from the Ontario and Rhode Island plants as well as the first case from North Carolina.³⁶

In January 1998, a NIOSH-hosted workshop reviewed tissue samples from 15 of 20 suspected cases of nylon flock-associated lung disease (five workers did not undergo biopsy). The pathologists reviewing the biopsies described the common underlying lesions as a lymphocytic bronchiolitis and peribronchiolitis with lymphoid hyperplasia represented by the presence of lymphoid aggregates. Variable features included acute alveolar injury in a diffuse alveolar damage pattern and bronchiolitis obliterans with organizing pneumonia. Granulomas, giant cells, and fibrosis were not predominant characteristic.^{37,38}

The clinical syndrome of these 25 cases included a spectrum of illness, manifested by insidious onset of dry cough and dyspnea with inspiratory crackles on exam. Chest radiographs were either normal or had diffuse patchy infiltrates; high-resolution CT scans showed patchy ground-glass opacities, diffuse micronodular disease, or peripheral honeycombing. Pulmonary function tests (PFTs) often revealed restrictive patterns with decreased diffusing capacity. The clinical course was variable, but several cases required hospitalization or even mechanical ventilation. Response to steroids was also variable, but all cases showed marked improvement clinically and radiographically upon leaving work, often relapsing upon return to the factory. Recovery occurred after weeks to months or even years of leaving the workplace, and not all cases returned to their baseline.^{35,37}

Animal studies suggested that dust containing ultrafine respirable fragments of cut flock were responsible for the acute inflammatory process, demonstrated in rats after a single intratracheal instillation.³⁹ Other possible etiologic agents include dyes, a finishing coat that contains tannic acid, acrylic latex adhesives, backing fabrics, and aerosolized mycotoxins; cigarette smoking may be a cofactor.³⁶ Based on its investigations, NIOSH's recommendations to reduce lung toxicity from nylon flock processing include dust control measures, improved ventilation, personal respiratory protective equipment, lung disease screening, and ongoing medical surveillance.³⁷

Popcorn Worker's Lung

In 2000, an outbreak of bronchiolitis obliterans among workers at Missouri microwave popcorn plants led to epidemiological investigations implicating diacetyl, a ketone with butter-flavor characteristics. The clinical syndrome included exertional dyspnea, cough, and wheeze; PFTs demonstrated an obstructive pattern without bronchodilator response. Among the original Missouri cohort, workers in the plant had 2.6 times higher than expected rates of chronic cough and dyspnea, and twice the expected rates of asthma and bronchitis. Eight workers were diagnosed with full-blown bronchiolitis obliterans.⁴⁰ Seven workers in California plants were later diagnosed with the syndrome.⁴¹ Three patients have died.⁴² While the numbers of people working in microwave popcorn plants may be small, the syndrome is illustrative of the fact that healthy workers can become ill from a chemical exposure previously assumed safe.

World Trade Center Lung

The destruction of the World Trade Center towers on September 11, 2001, led to the immediate release of a complex amalgam of airborne pollutants. With two 110-story buildings pulverized followed by prolonged combustion, massive amounts of particulate matter were released, exposing tens of thousands of people to myriad toxic effects.

Respiratory protection was not available immediately after the collapse, and its use was suboptimal during subsequent rescue and cleanup efforts, even among firefighters.⁴³

The initial collapse produced primarily coarse, medium-sized [median mass aerodynamic diameter (MMAD) 2.5 to 10 μm] and large-sized (MMAD > 10 μm) respirable particulates of alkaline pH. Analysis of the particulate matter showed a complex mixture of building debris fibers such as fiberglass, asbestos, aluminum and calcium silicates, and polycyclic hydrocarbons.⁴⁴ Structural fires (from fuel from ignited aircraft, transformers, and automobiles) persisted for 3 months after the catastrophe, further polluting the air with combustion-related fine particulate matter (MMAD < 2.5 μm). The daunting cleanup effort involved mechanical removal of 1.5 billion kg of rubble, which stirred up additional small particulate matter.⁴⁵ Normal upper airway defenses usually prevent large, coarse alkaline particulate from entering the lung (potentially damaging upper airway mucosa instead); fine particles are more likely to damage the lower respiratory tract. However, induced sputum from WTC firefighters 10 months later still contained significantly larger particles (> 2 μm) than control firefighters from Tel Aviv, with the composition of these particles matching that of settled material at Ground Zero. This suggests that the massive exposure overwhelmed the nasopharyngeal filtering, mucociliary clearance, and alveolar macrophage defense mechanisms.⁴⁶

The majority of clinical syndromes associated with WTC dust exposure are related to aerodigestive tract mucosal damage. Prezant et al described WTC cough among exposed firefighters; the syndrome was defined as a persistent postexposure cough accompanied by respiratory symptoms severe enough to require medical leave for at least 4 consecutive weeks. The cough was often accompanied by nasal congestion or drip, pharyngitis, gastroesophageal reflux, wheeze, dyspnea, and chest tightness. Of the ~11,000 NYC firefighters who worked at Ground Zero, 332 fit the criteria for WTC cough. There was a significant dose-dependent relationship: 8% of those with high levels of exposure (present during the actual collapse) developed WTC cough; 3% of those with moderate exposure (within first 2 days) and 1% of those with low exposure (within 3 to 7 days) developed WTC cough. Furthermore, 23% of firefighters with high exposure and 8% of those with moderate exposure demonstrated new bronchial hyper-reactivity on methacholine challenge.⁴⁷ Another study of pulmonary function among WTC workers pre- and post-9/11 showed a mean forced expiratory volume in 1 second (FEV₁) loss of 372 ml in the first year, equal to 12 years of aging-related FEV₁ decline.⁴⁴

Firefighters are routinely monitored for respiratory injury via serial radiographs and pulmonary function testing, so the effects of 9/11 are more easily quantifiable than the sequelae for the lay rescue/cleanup workers at Ground Zero or downtown residents. The WTC Worker and Volunteer Medical Screening Program has registered over 9000 rescue/recovery workers (including first responders, construction workers, utility workers, and lay volunteers). Between 2002 and 2004, 69% reported new or worsening respiratory symptoms while working at Ground Zero; 59% of these reported persistent symptoms. Twenty-eight percent had abnormal spirometry (21% with low FVC, 5% with obstructive patterns), abnormalities that persisted at least 1 year after the exposure.⁴⁸ A WTC Health Registry has been established to try to track the estimated 410,000 persons exposed; when enrollment closed in November 2004, 71,000 people had registered.^{49,50} Several Manhattan hospitals have dedicated WTC clinics that are collecting data from thousands of symptomatic WTC-exposed persons who present with persistent cough or dyspnea and evidence of small airways disease on oscillometry.

In addition to the well-described airways damage from WTC dust exposure, several cases of interstitial disease have been identified. The first case was a firefighter with heavy dust

exposure who presented 13 days after the WTC collapse with cough productive of blackish sputum, fatigue, fever, myalgias, and hypoxic respiratory failure. His CT scan showed patchy ground-glass opacities and airway thickening. Bronchoalveolar lavage demonstrated a pleocytosis with 70% eosinophils, as well as activated CD4+ T cells and elevated interleukin (IL)-5 levels. Asbestos, chromium, and silicon fibers as well as fly ash, degraded glass, and metal particles were identified in alveolar macrophages from the lavage fluid. A diagnosis of acute eosinophilic pneumonia was made. The firefighter was treated with intravenous steroids with rapid improvement in clinical, physiological, and radiographic parameters.⁵¹

The second case of interstitial disease related to WTC exposure involved an engineer who worked one block from Ground Zero. He experienced high levels of exposure on 9/11, returned to the site on 9/14 for one day, then resumed work on 10/3. On 10/16, he developed cough, wheeze, pharyngitis, and dyspnea; chest radiography showed diffuse miliary nodules in all lung fields. CT confirmed the presence of innumerable discrete nodules < 10 mm without evidence of adenopathy, pleural fluid, or cardiac enlargement. Exam, laboratories, and pulmonary function testing were all unremarkable. He underwent lung biopsy via video-assisted thoracoscopy, with pathology demonstrating noncaseating granulomas distributed along bronchovascular bundles. Polarizing microscopy showed no birefringent material; energy-dispersive radiograph analysis showed silica, silicates, and calcium oxalate. Kveim testing and microbiology were negative. His CT abnormalities did not improve after a 4-week course of high-dose prednisone. He was diagnosed with granulomatous pneumonitis, thought to be most likely due to inhaled dust containing heavy metals.⁵²

The persistence of symptoms among exposed individuals, now 7 years after the WTC tragedy, suggests that both health effects and financial burden will continue to rise for the short term. Many of the inhaled toxins include potential carcinogens (e.g., polycyclic aromatic hydrocarbons, asbestos) with long latency periods. Earlier this year, a NYC police officer died with accelerated pulmonary fibrosis, and WTC dust exposure was considered contributory. Other cases of pulmonary fibrosis have been discovered among rescue and cleanup workers, although no definitive association has been made. A utility repairman with extensive Ground Zero exposure died of IPF; his family has brought a wrongful death suit against the city of New York for failing to provide appropriate respiratory protection. Similar class action suits have been filed. The City of New York reported \$392 million in annual costs of WTC-associated health care; merely maintaining the existing major programs offered through the Fire Department of New York and the WTC clinics at Bellevue and Mt. Sinai Hospitals will cost over \$153 million annually.⁵³ Federal and state monetary compensation is earmarked to the sum of \$1 billion, although as of May 2007 only 14,000 of the 100,000 eligible had filed for worker's compensation. The filing deadline was extended until August 2008.⁵⁴

CHALLENGES OF MEASURING BURDEN

The epidemiology and the economic burden of exposure-related diffuse lung disease remain difficult to elucidate. Few studies have successfully estimated the national incidence or prevalence of exposure-related disease. Even less is known about the economic burden imposed on both the health care system and the national workforce. As a result, we are hard pressed to adequately assess risk and determine cost. Changing definitions and classifications (particularly for interstitial lung disease) over the past decades have made data difficult to aggregate and compare. Multiple contributing factors and long latency further complicate analyses. The costs of occupational injuries and illnesses are large compared with other diseases, likely due to the large American workforce that is at risk.¹ Furthermore, when physicians unfamiliar with occupational lung disease fail to elicit a

thorough exposure history, valuable data are not captured. The data that do exist (e.g., disability statistics, death certificates, hospitalization admission diagnoses) may not be accurate or complete.⁵⁵ Thus the true burden of exposure-related lung disease is likely significantly underestimated.

CONCLUSIONS

The true burden of exposure-related diffuse lung disease remains unknown. Estimates of the cost of occupational diseases exceed \$26 billion annually, with future costs likely higher due to ongoing exposures to toxic respirable substances in the workplace, as well as poor compliance with protective measures on the part of businesses and individual workers. The Bureau of Labor Statistics conducted a survey in 2001 of private industry respirator use. The survey found that only 10% of private industry workplaces used respirators; nearly half of these were on a voluntary basis only; 12% used them only when required for emergencies. Less than half of mining, construction, and manufacturing industries reported mandatory nonemergency use of respirators, usually while working with paint vapor, dust, and solvents.⁵⁶ In an OSHA April 2007 report to Congress, the agency named improved respiratory protection as one of its top goals.⁵⁷ The requested OSHA 2008 budget of \$490 million, with over 2000 full-time employees, will help educate workers and employers about respiratory protection and enforce compliance with maintaining safe levels of exposure. Yet even while progress is being made toward reducing occupational illness in the United States, the globalization of industry has led to an “export of hazards,” where industries relocate to developing countries where industry regulations are not as strict. The burden of disease, still astronomical in the United States, will eventually shift toward the developing world.⁵⁸

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Figure 1.

Lung cancer relative risk relative to cumulative exposure to various agents. Reproduced with permission from Steenland K. One agent, many diseases: exposure-response data and comparative risks of different outcomes following silica exposure. *Am J Ind Med* 2005;48:16–23.

Figure 2. Global deaths from asthma, COPD, silicosis, asbestosis and coal workers' pneumoconiosis (CWP) in the year 2000. Reproduced with permission from Driscoll T, Nelson DI, Steenland K, et al. The global burden of non-malignant respiratory disease due to occupational airborne exposures. *Am J Ind Med* 2005;48(6):432–445.

Figure 3. DALYs due to asthma, COPD, silicosis, asbestosis and coal workers' pneumoconiosis (CWP) in the year 2000. Reproduced with permission from Driscoll T, Nelson DI, Steenland K, et al. The global burden of non-malignant respiratory disease due to occupational airborne exposures. *Am J Ind Med* 2005;48(6):432–445.

Figure 4.

Asbestosis: age-adjusted death rates by state, U.S. residents age 15 and over, 1993–2002.

From Centers for Disease Control, National Institute for Occupational Safety and Health

Web site <http://www2.cdc.gov/drds/WorldReportData/UpdateDetails.asp?UpdateDateID=2>.

Figure 5.

Asbestosis: number of deaths, crude and age-adjusted death rates, U.S. residents age 15 and over, 1968–2002. From Centers for Disease Control, National Institute for Occupational Safety and Health Web site <http://www2.cdc.gov/drds/WorldReportData/UpdateDetails.asp?UpdateDateID=2>.

Figure 6. Silicosis: number of deaths, crude and age-adjusted death rates, U.S. residents age 15 and over, 1968–2002. From Centers for Disease Control, National Institute for Occupational Safety and Health Web site <http://www2.cdc.gov/drds/WorldReportData/UpdateDetails.asp?UpdateDateID=2>.

Figure 7.
Silicosis: age-adjusted death rates by state, U.S. residents age 15 and over, 1993–2002.
From Centers for Disease Control, National Institute for Occupational Safety and Health
Web site <http://www2.cdc.gov/drds/WorldReportData/UpdateDetails.asp?UpdateDateID=2>.

Figure 8.

Coal workers' pneumoconiosis: number of deaths, crude and age-adjusted death rates, U.S. residents age 15 and over, 1968–2002. From Centers for Disease Control, National Institute for Occupational Safety and Health Web site <http://www2.cdc.gov/drds/WorldReportData/UpdateDetails.asp?UpdateDateID=2>.

Figure 9. Coal workers' pneumoconiosis: age-adjusted death rates by state, U.S. residents age 15 and over, 1993–2002. From Centers for Disease Control, National Institute for Occupational Safety and Health Web site <http://www2.cdc.gov/drds/WorldReportData/UpdateDetails.asp?UpdateDateID=2>.

Table 1**Selected Agents and Industries with Potential for Respiratory Hazard Exposure**

Agent	Industry	Estimated Number of Persons Exposed	Chronic Respiratory Disease	Estimated Risk
Asbestos	Construction, manufacturing, and mining of asbestos and related materials	~250,000 primary processing 3×10^6 in secondary processing	Pulmonary fibrosis, obstructive airway disease, lung cancer	Asbestosis: 10–50%, lung cancer: 1–10%
Silica	Mining, stone cutting, construction, farming, quarrying	1.2×10^6 in nonagriculture	Pulmonary fibrosis, silicosis	0–13%
Cotton dust	Cotton mills	800,000	Chronic bronchitis, reduced pulmonary function, bysionosis	2–30%
Coal dust	Coal mining	200,000	Pulmonary fibrosis, pneumoconiosis	4–46%
Known chemical and biological sensitizers	Wide variety of industries	$> 9 \times 10^6$	Occupational asthma	Up to 28% of adult asthma
Grain dust	Elevator agents, dock workers, longshoremen, mill workers, bakers	500,000	Occupational asthma, COPD chronic bronchitis	Up to 30% in non-smokers, perhaps up to 60% in smokers

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COPD, chronic obstructive pulmonary disease.