

Figure 1. (Continued). sectioning revealed diffuse centrilobular ground-glass attenuations suggestive of bronchiolitis. (C–E) Pathologic findings of a representative case of humidifier disinfectant-associated interstitial lung disease in humans. (C) The typical bronchiolocentric destruction with obliteration was observed corresponding to centrilobular, ground-glass opacities seen on CT. Along the bronchovascular bundle were seen inflammation and fibrosis without definite granulomas (hematoxylin and eosin [H&E] stain, original magnification, $\times 100$). (D) Some terminal bronchioles revealed bronchial epithelial denudation with squamous metaplasia (H&E stain, original magnification, $\times 200$). (E) In areas with advanced disease, most of the airspaces had disappeared and were filled with extensive fibrosis and interstitial thickening (H&E stain, original magnification, $\times 100$). (F and G) Pathologic examination of PHMG-induced lung injury in rats. (F) Inflammation and fibrosis were observed along bronchovascular bundles and resulted in bronchiolar obliteration (*inset*, H&E stain, original magnification, $\times 100$). Squamous metaplasia was also seen in the left upper area of the slide (H&E stain, original magnification, $\times 40$). (G) The bronchiolar epithelium was denudated, and foamy histiocyte accumulation was observed along the lining of the bronchiolar epithelial cell denudation. Flattened epithelial cells also replaced the normal ciliated cuboidal epithelium (asterisk) (H&E stain, original magnification, $\times 400$).

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Resurgence of a Debilitating and Entirely Preventable Respiratory Disease among Working Coal Miners



To the Editor:

For more than 40 years, the National Institute for Occupational Safety and Health (NIOSH) has monitored trends in coal workers' pneumoconiosis, including progressive massive fibrosis (PMF). PMF is an advanced, debilitating, and lethal form of coal workers' pneumoconiosis with limited, primarily palliative treatment options and no cure. As part of ongoing surveillance efforts, NIOSH administers the Coal Workers' Health Surveillance Program (CWHSP), which offers underground coal miners periodic chest radiographs and confidentially informs them of their pneumoconiosis status (1). Just 15 years ago, PMF was virtually eradicated, with a prevalence of 0.08% among all CWHSP participants and 0.33% among active underground miners with at least 25 years of mining tenure. Since that time, the national prevalence of PMF identified through the CWHSP has increased; the rate of increase in the central Appalachian states of Kentucky, Virginia, and West Virginia has been especially pronounced (Figure 1). Excessive inhalation of coal mine dust is the sole cause of PMF in working coal miners, so this increase can only be the result of overexposures and/or increased toxicity stemming from changes in dust composition (2). During 1998 to 2012, NIOSH identified 154 cases of PMF among CWHSP participants, 125 of whom were long-tenured underground coal miners in central Appalachia. In 2012, the prevalence of PMF in this group of working miners reached 3.23% (5-year moving average), the highest level since the early 1970s. At the same time, NIOSH documented cases of PMF among surface coal miners with little or no underground mining tenure (3).

Each of these cases is a tragedy and represents a failure among all those responsible for preventing this severe disease. This year marks the 45th anniversary of the Federal Coal Mine Health and Safety Act. In that legislation, Congress enacted enforceable dust standards to reduce the incidence of coal workers' pneumoconiosis and eliminate PMF among underground coal miners (4). Despite readily available dust control technology and best practices

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

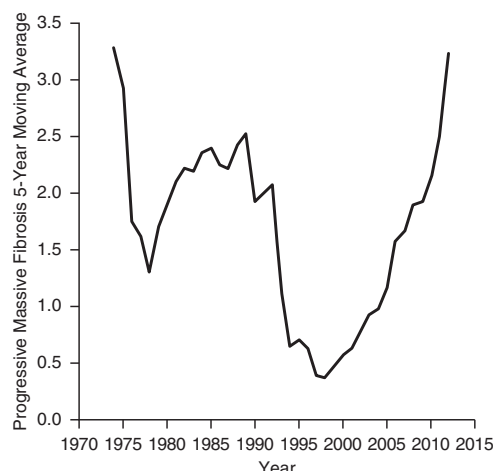


Figure 1. Prevalence of progressive massive fibrosis among working underground coal miners with 25 or more years of underground mining tenure (1974–2012) in Kentucky, Virginia, and West Virginia, according to the Coal Workers' Health Surveillance Program. Data are 5-year moving average (e.g., data plotted for 1974 = $\frac{\sum [PMF_{1970} + PMF_{1971} + PMF_{1972} + PMF_{1973} + PMF_{1974}]}{\sum \text{Participants}_{1970-1974}}$; surveillance is conducted on a 5-year national cycle).

guidance (5), recent findings suggest dust exposures have not been adequately controlled and that a substantial portion of U.S. coal miners continue to develop PMF. On August 1, 2014, NIOSH issued an interim final rule modifying existing regulations to include surface coal miners in the CWHSP (6). In addition, the interim final rule expands medical surveillance beyond occupational history and chest radiography to include respiratory symptom assessment and spirometry testing for the recognition of undiagnosed chronic obstructive pulmonary disease among all working coal miners. We believe that expanded medical surveillance is an important part of ensuring success in efforts to protect U.S. coal miners from this deadly but entirely preventable disease. ■

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Trends in Infection Source and Mortality among Patients with Septic Shock



To the Editor:

We read with interest the article by Leligdowicz and colleagues (1) describing associations between source of infection and mortality among patients with septic shock. The authors aptly point out the relevance of their findings to clinical trial design and prognostic scoring systems. We were intrigued by the demonstration that adjusted mortality risk differs widely between different sites of infection during septic shock and wondered whether this finding may help inform the interpretation of recent studies demonstrating declining severe sepsis mortality rates (2–4). For example, if rates of lower-risk septic shock resulting from urinary tract infections are rising over time, whereas rates of higher-risk septic shock caused by bowel ischemia are declining (perhaps because of improvements in cardiovascular prevention), then large improvements in severe sepsis mortality attributed to improved processes of care may partly be explained by changing secular trends in infection sites. Did Leligdowicz and colleagues observe changing incidence of infection sites over the 19-year course of their study? ■

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