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, ×100). (*D*) Some terminal bronchioles revealed bronchial epithelial denudation with squamous metaplasia (H&E stain, original magnification, ×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, ×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, ×100). Squamous metaplasia was also seen in the left upper area of the slide (H&E stain, original magnification, ×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, ×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.

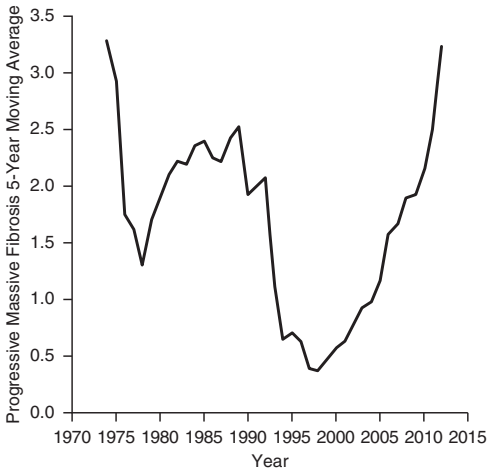

Figure 1. Prevalence of progressive massive fibrosis among working underground coal miners with 25 or more years of underground mining tenure (1974–2012) in Kentucky, Virginia, and West Virginia, according to the Coal Workers' Health Surveillance Program. Data are 5-year moving average (e.g., data plotted for 1974 = $\frac{\sum[PMF_{1970} + PMF_{1971} + PMF_{1972} + PMF_{1973} + PMF_{1974}]}{\sum Participants_{1970-1974}}$; 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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Age of Systemic Lupus Erythematosus Onset and Risk for Asthma

To the Editor:

In their nationwide population study, Dr. Shen and coworkers report there was a significantly higher incidence of asthma in patients with systemic lupus erythematosus (SLE) than in the general population. Of interest is that in their study, asthma risk associated with SLE was gradually decreased in older participants (1).

To our knowledge, childhood-onset SLE is characterized by a high degree of morbidity compared with adult-onset SLE (2). Pediatric patients are at higher risk of developing lupus renal disease, malar rashes, pericarditis, and hematologic alterations (3). In addition, they usually have a greater use of prednisone and additional immunosuppressive therapies to attenuate SLE activity (2). In contrast, patients with adult-onset SLE have a higher risk of developing pulmonary disease and may be associated with another autoimmune disease (4).

In a longitudinal study by Hersh and colleagues, 43.3% of participants with adult-onset SLE had pulmonary involvement during a 13.4-year follow-up compared with 23.6% of participants with childhood-onset SLE during 16.5 years of follow-up (4). The relative risk of pulmonary involvement between adult-onset SLE and childhood-onset SLE could be roughly estimated to be 2.26. In the present study, the relative risk for incident asthma between those who were certificated with SLE at greater than 20 years of age and those younger than 20 years was estimated to be 2.64, which is close to the risk seen in the study by Hersh and colleagues. Therefore, the occurrence of asthma could be caused by lupus pulmonary involvement, including interstitial pneumonitis or pleuritis in people with adult-onset SLE, although some other risk factors for asthma, such as cigarette smoking, may coexist with SLE.

In contrast to children, adults may suffer asthma-like bronchial spasms from smoking-related chronic obstructive pulmonary disease or cardiogenic origins such as heart failure. According to the 2012 National Center for Health Statistics surveys and the Vital Statistics System, in the United States, the prevalence of asthma was 9.3% for children and 8.0% for adults (5). However, the incidence of asthma in adults was much higher than in children in the control group in the present study, implying that some comorbid diseases in adults were misclassified to asthma. In