Coal mining remains a sizable industry, with millions of working and retired coal miners worldwide. This article provides an update on recent advances in the understanding of respiratory health issues in coal miners and focuses on the spectrum of disease caused by inhalation of coal mine dust, termed coal mine dust lung disease. In addition to the historical interstitial lung diseases (coal worker's pneumoconiosis, silicosis, and mixed dust pneumoconiosis), coal miners are at risk for dust-related diffuse fibrosis and chronic airway diseases, including emphysema and chronic bronchitis. Recent recognition of rapidly progressive pneumoconiosis in younger miners, mainly in the eastern United States, has increased the sense of urgency and the need for vigilance in medical research, clinical diagnosis, and exposure prevention. Given the risk for disease progression even after exposure removal, along with few medical treatment options, there is an important role for chest physicians in the recognition and management of lung disease associated with work in coal mining.

Keywords: pneumoconiosis; silicosis; pulmonary fibrosis; chronic obstructive pulmonary disease; coal mining

In recent years, the country has been riveted by stories of tragic disasters in our nation's coal mines, including the explosions and fires at the Sago and Upper Big Branch mines in West Virginia. Less attention has been paid to the increase in prevalence and severity of the chronic lung disease that results from coal mine dust exposure. This article provides a review of the spectrum of disease caused by prolonged inhalation of mine dust, "coal mine dust lung disease" (CMDLD). CMDLD includes the historical interstitial lung diseases (coal worker's pneumoconiosis [CWP], silicosis, and mixed dust pneumoconiosis) but also includes the more recently described entity of dust-related diffuse fibrosis (DDF). Coal mine dust also causes chronic obstructive pulmonary disease, including chronic bronchitis and emphysema, which often are not recognized as being related to this exposure.

CONTEMPORARY COAL MINING

Coal mining remains a sizable industry due to its important role in energy production. Modern mining technology has improved productivity, with the capability to pulverize thousands of tons of coal per shift. These tasks generate clouds of respirable dust particles with toxic radicals on their surfaces (1). The number of coal miners in the United States declined steadily after World War II as the industry became more mechanized. This trend reversed in 2003, and by 2011, 143,437 miners worked in 1,973 underground and surface coal operations, providing about 20% of all domestic energy and more than 40% of the electricity (2). With 28% of the world's recoverable coal reserves, mining employment in the United States is likely to remain high for many years (3). Many countries engage in coal mining: China alone employs more than 6 million miners (4). Due to the potential for a large burden of lung disease, the respiratory health of coal miners remains an important consideration for physicians in the United States and worldwide (5).

RESURGENT PNEUMOCONIOSIS—EVIDENCE FOR INCREASES IN PREVALENCE AND SEVERITY

Although many physicians believe these diseases are only of historical interest, evidence published during the last few years from several independent data sources points toward an ongoing increase in both the prevalence and severity of CMDLD.

U.S. Coal Miner Chest Radiograph Surveillance Programs

Starting in about 2000, surveillance of working U.S. coal miners began to show an unexpected increase in the proportion of miners with chest radiographic changes consistent with pneumoconiosis, ascertained by at least two qualified physicians reading independently, using the International Labor Office (ILO) pneumoconiosis classification (available at http://www.ilo.org/safework/info/publications/WCMS_168260/lang--en/index.htm) (6, 7) (Figure 1). This increase followed 30 years of decline after enactment of the Federal Coal Mine Health and Safety Act of 1969 (8). That act established enforceable limits for respirable dust exposure in coal mines and was followed by significant reductions in exposure. U.S. coal miners are also offered a health surveillance program administered by the National...
Coal Mine Disaster Investigation

Additional independent confirmation of a high risk for dust disease among contemporary coal miners came from the West Virginia Governor’s Panel investigating the 2010 explosion that killed 29 miners at the Upper Big Branch mine (16). The state medical examiners obtained sufficient lung tissue for post mortem examination in 24 of the 29 victims, and 71% (17 of 24) were noted to have pathologic findings of CWP. The 17 miners whose lungs showed CWP ranged in age from 25 to 61 years, including five who had less than 10 years’ mining experience; nine had been miners for more than 30 years. Of note, 16 of the 17 miners with CWP had started working after the modern dust limits were put into effect.

COAL MINE DUST CAUSES A BROAD SPECTRUM OF LUNG DISEASE

The surveillance results described above are certainly troubling, but they track only one manifestation of coal mine dust toxicity, that of radiographic pneumoconiosis. There are also new insights about the diffuse interstitial fibrosis and airways disease caused by this exposure.

Classic Pneumoconiosis—Not So Classic

Contrary to the popular belief that the fibrotic scars seen on chest radiographs of coal miners are almost always rounded, both rounded and irregular opacities have been described as manifestations of CMDLD. Several studies have demonstrated a clear relationship between both of these types of opacities and measures of coal mine dust exposure (17–19). Irregular radiographic opacities in a miner may also reflect a syndrome of DDF, a less common variant of both CMDLD and silicosis (20). In a study of 357 Welsh coal miners, irregular opacities were associated with reduced diffusion capacity and restrictive changes on pulmonary function (21). Pathologically, DDF may manifest as bridging fibrosis connecting the macular, nodular, or PMF lesions of CWP or silicosis, often with pigmented interlobular septal thickening (22, 23) (Figure 3). Absent careful consideration of the occupational history, DDF may be mistaken for idiopathic pulmonary fibrosis, with important implications for both disease management and prognosis. In the Welsh coal miners with findings of DDF, age at presentation was younger (55.5 ± 7 yr) and mean years of survival more favorable (11.4 ± 5 yr) compared with patients with IPF (22). Prevalence of DDF ranges between 15 and 20% in autopsy studies of coal miners (22), substantially higher than IPF prevalence reported in the general population (24).

Radiographic Distribution of Opacities—Not Always Upper Lobe Predominant

Although conventional teaching has been that the radiographic finding of upper lung zone—predominant small rounded opacities is the sine qua non of CWP, there is surprisingly little published evidence to support this assertion. A recent study from 2,476 underground U.S. coal miners showed that small radiographic opacities were approximately equally distributed throughout the lung zones (25). In this study, small opacities were considered primarily rounded in a majority of the miners but were mostly irregular in a relatively large proportion (37.9%) (25). The study found no meaningful differences in age, mining tenure, or smoking between miners with rounded versus irregular opacities, although data on tobacco use were limited.

Chronic Obstructive Lung Disease in Coal Miners

There is increasing recognition that occupational exposure as well as tobacco smoke plays a significant role in the development of COPD, and observations of COPD in non-smoking coal miners suggest that cigarette smoke may synergize with coal mine dust to increase the risk of COPD. A recent study of 4,2517 miners (both smoking and non-smokers) found that annual airway function measurements were reduced in former miners with radiographic findings of pneumoconiosis, with the greatest reduction in forced expiratory volume in 1 second (FEV1) observed in the former miners with grade 1 pneumoconiosis (26). In a study of 2,464 underground coal miners, the risk of COPD was increased in former miners with radiographic pneumoconiosis (27). These findings, along with the increasing recognition of COPD among coal miners, underscore the importance of early identification and intervention to prevent the progression of COPD in this high-risk group.
of chronic obstructive lung disease (26). Perhaps the most compelling evidence comes from the multiple studies of coal miners, confirming a strong, consistent, and dose-dependent relationship between respirable coal mine dust exposure and chronic airway diseases (including chronic bronchitis and emphysema) (7, 27). Obstructive lung disease often occurs in miners without pneumoconiotic opacities on routine chest radiographs (28). Symptoms of cough, sputum production, shortness of breath, and wheezing are all associated with cumulative exposure to respirable coal mine dust, with prevalence of chronic bronchitis in U.S. miners estimated at 35% (29). Miners tend to suffer larger declines in lung function shortly after beginning work, after which losses continue, but at a slower pace (30, 31). The onset of bronchitic symptoms is associated with large early FEV₁ declines, suggesting both may result from inflammation and narrowing in the larger airways (32). Studies of FEV₁ decline in coal miners have demonstrated essential equivalence between 1 year of work in a job at the coal face and 1 year of cigarette smoking; no disproportionate effect of smoking compared with mine dust has been observed (33–35).

Emphysema seen in CMDLD is most commonly the centriacinar type; pathologically, it is referred to as focal emphysema when a coal dust macule is also present (22). Bullous and panacinar emphysema have also been described and are part of the continuum of disease that may follow either smoking or coal mine dust exposure (22). Emphysema severity in miners is related to cumulative coal mine dust exposure and lung dust content (21, 36). In a landmark study of 722 autopsied coal miners and nonminers, cumulative exposure to respirable coal mine dust, as well as the weight of dust retained in the lungs, were both significant predictors of emphysema severity, after accounting for cigarette smoking, age at death, and race (37). The contributions of coal mine dust exposure and cigarette smoking were similar in predicting emphysema severity (37). Additional evidence for the toxicity of coal mine dust comes from mortality studies, which show increased death rates from COPD associated with cumulative coal mine dust exposure, after accounting for smoking (38).

**Progression and Severity of Disease**

Several risk factors associated with severe forms of CMDLD have been identified. A survey of 264 underground coal miners identified several mine environment variables that contributed to the risk of excessive decline in lung function over 11 years. Accelerated declines in FEV₁ (defined in that study as a loss of 60 ml per year more than a matched referent working miner) were associated with work in roof bolting, lack of use of respiratory protection, exposure to explosive blasting fumes, and use of stored mine water (which can be contaminated with organic materials) for dust suppression sprays (39). As noted previously, rapid radiographic CWP progression has been observed in certain U.S. mining regions (10, 15). Elevated respirable silica exposures, work in smaller mines, and mining of thin coal seams or coals with high carbon content have all been implicated as factors in the observed regional differences in disease severity (40). It is important to note that the functional and radiographic abnormalities of CMDLD may both progress after exposure ceases, likely due to ongoing inflammatory effects of retained mineral dust (41–44).

**Coal Mining and Lung Cancer**

Coal miners are potentially exposed to a number of lung carcinogens, including respirable silica (quartz), radon progeny, and diesel exhaust, the latter introduced into U.S. underground coal mines in the 1950s. Despite these exposures, earlier cohort mortality studies of coal miners suggested that coal mining may be protective for lung cancer (45). These studies were criticized due to short follow-up and failure to adequately account for healthy worker effects, including the prohibition against smoking in coal mines (46). Recent investigations suggest that coal miners may show excess lung cancer mortality. Underground U.S. miners showed a nonsignificant excess of lung cancer mortality after 23 years of follow-up (standardized mortality ratio, 1.07; confidence interval, 0.95–1.19) (47). A study of British miners linked lung cancer risk with exposure to quartz but not coal mine dust (38). Further investigation of lung cancer risk in coal miners, with careful control for tobacco smoking, is warranted.

**BIOLOGIC MECHANISMS OF COAL MINE DUST TOXICITY**

Coal dust has been shown to stimulate release of cytokines that are important in lung inflammation and fibrosis, including tumor necrosis factor-α (TNF-α) and IL-1 (48). Compared with nonminers, miners with CWP have elevations of both serum and bronchoalveolar lavage cytokines, including IL-1β, IL-6, and TNF-α (49). Additionally, miners with CWP, with or without PMF, showed elevated bronchoalveolar lavage markers of oxidant injury, including superoxide dismutase, glutathione peroxidase, and catalase, compared with control subjects. In a study of miners with early CWP demonstrated...
on chest high-resolution computed tomography (CT), plasma levels and activities of antioxidant enzymes (malondialdehyde, superoxide dismutase, and glutathione peroxidase) were elevated compared with control subjects (50), suggesting a role for oxidative stress due to increased free radical formation. Oxidant injury from coal dust exposure has also been suggested in animal models of lung inflammation (51).

The level of bioavailable iron in coal dust samples has been associated with the ability of dust samples to initiate lipid peroxidation, activate alveolar protein-1, and down-regulate transferrin receptor gene expression in alveolar type II cells in culture (52, 53). Prevalence of CWP in workers from different mining regions has been correlated with levels of bioavailable iron in coal (54).

Genetic susceptibility to CMDLD has been investigated, but no significant markers have been found. In a case-control study that included 304 underground coal miners with PMF, investigators found that single nucleotide polymorphisms within genes involved in inflammatory and fibrotic processes (including IL-1, IL-6, TNF-α, transforming growth factor-β, vascular endothelial growth factor, intercellular adhesion molecule-1, and matrix metalloproteinase-2) were not differentially distributed between the PMF and unaffected control subjects (55). A study of CMDLD progression over 4 years suggested certain polymorphisms of IL-18 may be protective (56). Further study is needed to identify useful clinical biomarkers of exposure, effect, and susceptibility to CMDLD (57).

DIAGNOSIS OF CMDLD

The diagnosis of coal mine dust–related lung disease is based on a detailed history of exposures and respiratory symptoms combined with lung imaging and pulmonary function testing. A surgical lung biopsy showing pigmented inflammatory and fibrotic lesions with associated emphysema, typically located in the walls of the respiratory bronchioles, can be quite specific for CWP (Figure 3A) (58). In the setting of a coal miner with at least 10 years of exposure and findings and clinical course typical for CMDLD, neither bronchoscopy nor a lung biopsy is required for diagnosis (5, 59). In contrast, although recent studies have shown progression from normal to massive fibrosis in as little as 5 to 7 years (15), miners with a rapid course should be carefully evaluated for alternative explanations, such as mycobacterial infection, silica-associated granulomatosis with polyangiitis (Wegener’s), or other interstitial pneumonias. Physiologic findings contribute to the diagnosis by determining disease patterns and are particularly helpful in evaluating severity and progression as well as the impact of these disorders on work capacity and impairment.

History

The exposure history is a critical component in the diagnosis. The risk of CMDLD is increased with tasks at the coal face, including extracting (mining), loading, and transporting coal. Silica exposure can accelerate progression and occurs when mining equipment disturbs or cuts into rock at the mine roof or floor as well as during drilling and bolting the mine roof to prevent cave-ins. Clinicians evaluating coal miners require detailed information on the intensity and duration of exposure, including the various factors discussed in this review that have been recognized to affect the risk and severity of occupational lung disease. However, respirable dust levels reported for purposes of compliance with regulations have not closely tracked disease risks for individual miners (11).

Chest Imaging

Although lung imaging has been historically central to the recognition of pneumoconiosis, routine chest radiography has low sensitivity for the early changes of CWP (59) and for dust-related airways disease and emphysema. The International Labor Office (ILO) provides a classification system, including a set of standard radiographs, which can facilitate an accurate and consistent approach to recognizing and categorizing the radiographic changes associated with dust exposure. ILO categories correlate reasonably well with pathologic CWP (Somers’ D = 0.64 for small opacities; agreement for large opacities = 83%) (59). In 2011, the ILO classification was expanded to enable the classification of digitally acquired images on a high-resolution medical viewing system in
addition to traditional analog chest films (60). When appropriate techniques are used for image capture and display, proficient readers find the digital and film formats functionally equivalent, with minor differences, such as a slight increase in findings of both irregular and coalescent opacities and fewer pleural abnormalities with digital techniques (61, 62).

Chest CT and high-resolution CT scans are more sensitive than routine radiographs in detecting CMDLD, although they are not recommended for routine surveillance due to the increased radiation exposure and expense, as well as the lack of an agreed-on scoring scheme (63). CT scans may demonstrate small pneumoconiotic abnormalities in a centrilobular or perilobular pattern, even when standard chest images are not clearly abnormal (64, 65). In addition, massive fibrotic lesions may be detected on CT images when they are not reported using standard two-dimensional images (66). A classification system for dust-related changes seen on CT scans has been proposed and has shown moderate to good reliability, although it has not yet been accepted internationally (67). CT may be most useful in the evaluation and management of symptomatic coal miners and in those with normal or borderline routine radiographs or with chest lesions that are atypical (68). For miners with advanced CWP, bilateral symmetrical elongated mass lesions on a background of diffuse small opacities are unlikely to represent neoplasia. Comparison of recent images with surveillance X-rays may also help to document a long period doubling time for pneumoconiotic masses. Unless standardized uptake values are low, PET scanning may not be helpful, because PMF lesions are often metabolically active and can demonstrate standardized uptake values overlapping those of malignant lesions (69). If cancer treatment is being considered, biopsy of a suspect lesion may be necessary, despite the elevated risk of hemorrhage from a vascular PMF lesion.

**Physiologic Evaluation**

CMDLD results from inflammatory responses to dust in lung parenchyma and in large and small airways. Thus, CMDLD may present with a restrictive, obstructive, or mixed pattern of impairment on resting pulmonary function tests, depending on the relative degree of airways disease, emphysema, and fibrotic responses (38, 70, 71). In addition to FEV<sub>1</sub> losses, miners may show declines in single breath diffusing capacity for carbon monoxide due to the abnormal gas exchange associated with increasing coal mine dust exposure (72).

Cardiopulmonary exercise testing provides information on the pattern and degree of impairment in those miners who can be safely exercised. Typical exertional gas exchange abnormalities from CMDLD include a widening of the alveolar–arterial oxygen gradient, accompanied by an excessive ventilatory response, resulting in a reduction in arterial partial pressure of carbon dioxide (73–75). Breathlessness in miners correlates with an increase in the ratio of expired minute ventilation to oxygen consumption on cardiopulmonary exercise testing, likely reflecting mismatching of ventilation and perfusion (75). Resting pulmonary function tests and arterial blood gases may be insensitive to this pattern of gas exchange abnormalities; in some cases of CMDLD, the extent of the impairment may only be demonstrated by blood gas studies obtained during exercise. Pulmonary vascular involvement from CMDLD can result in pulmonary hypertension with associated increase in dead space and decrease in stroke volume, which may suggest a pulmonary circulatory limitation (76, 77).

As with other occupational lung diseases, CMDLD often occurs in patients with comorbidities, such as cardiovascular disease, smoking-related lung disease, and obesity. Heart failure or morbid abdominal obesity may cause or contribute to restrictive ventilatory defects and gas exchange abnormalities that must, based on a reasoned medical opinion, be considered in the clinical evaluation of CMDLD. For miners with a history of substantial coal mine dust exposure and obstructive physiologic findings, sometimes with partial bronchodilator reversibility, the clinician often can conclude that CMDLD contributes substantially to the observed impairments.

**MANAGEMENT OF CMDLD**

No specific medical therapy has proven effective in reversing CMDLD. Whole-lung lavage can reduce lung dust burden (78) and has recently been applied in China, but indications, risks, and benefits all require clarification. Management of patients with CMDLD focuses on three objectives: (1) prevention of disease progression, (2) periodic medical monitoring, and (3) recognition and treatment of complications and comorbid diseases. Additionally, awareness of relevant compensation and benefits programs is helpful in managing affected patients.

**Prevention of Disease Progression**

For working miners, minimizing dust exposures is key. Environmental controls are mandated by federal law: ventilation systems, water sprays, and other dust capture devices require continuous monitoring to assure they operate as intended. An underground coal miner with a diagnosis of pneumoconiosis may have a legal right to frequent exposure monitoring and transfer to a reduced dust job if one is available at the mine.

Personal dust monitors have been approved for use in coal mines to continuously monitor an individual miner’s dust exposure. These electronic devices can provide real-time measurements of dust levels, allowing for immediate interventions to reduce harmful exposures (79). Unfortunately, personal dust monitors are expensive, and their role in managing individual miners remains to be clarified.

Respiratory protection using masks can offer a degree of health protection in certain industrial settings, when part of a comprehensive respiratory protection program (80). Federal regulations specify that use of dust masks in coal mining can only be a short-term measure and is not a substitute for effective environmental controls (81). Thus, reliance on protective masks to prevent chronic lung disease from coal mining is neither practical nor legal.

As with other chronic lung diseases, patients with CMDLD should adhere to recommended schedules for vaccinations against viral and bacterial pathogens, and for those who smoke, cessation efforts should be encouraged and supported.

**Medical Monitoring**

Historically, lung health of working coal miners has been monitored using periodic (every 3 yr) radiographs. The recent reports of rapid progression have challenged that approach. In addition to radiographs, spirometry every 1 to 3 years is now recommended, to detect rapid functional declines and permit timely interventions and preservation of lung health (28). Several approaches to longitudinal spirometry assessment have been published (including a free public-domain software package) that facilitate identification of individuals with accelerated declines, even when lung function remains within population normal limits.

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2Transfer rights are granted by the Mine Safety and Health Administration (MSHA), under Title 30 United States Code of Federal Regulations, Part 90–Mandatory Health Standards–Coal Miners Who Have Evidence Of The Development Of Pneumoconiosis, requiring a letter showing medical evidence of pneumoconiosis from the National Institute for Occupational Safety and Health.
Complications

The clinician should be alert to the major complications of dust disease, including airflow obstruction, respiratory infection, hypoxemia, respiratory failure, cor pulmonary, arrhythmias, and pneumothorax. Although an elevated risk of mycobacterial infection among patients with silicosis is recognized (84), in CMDLD, the association is not as well documented. However, numerous case reports suggest that vigilance is appropriate for coal miners with lung infiltrates or PMF lesions, particularly those who have been exposed to respirable silica (85). In miners with silicosis, recognition and isolation of tuberculosis and other mycobacterial organisms may be more challenging. Case fatality rates are higher, but aggressive treatment and careful follow-up is generally successful for infections with sensitive organisms.

For miners with impairment from CMDLD, a program of rehabilitation including medications, diet, exercise, and supplemental oxygen, targeted to the specific needs and capacities of the individual, is appropriate in addressing health-related quality of life (86). Lung transplantation has been performed for coal miners with very advanced lung disease. Opinions vary regarding the risks and benefits of this procedure (87, 88).

Compensation and Comorbidities

Once a diagnosis of CMDLD is made, clinicians may be asked the degree to which it contributes to impairment. This judgment often is confounded by other factors, such as smoking, obesity, and/or cardiac disease. Administrators of state and federal compensation systems or insurance agencies often provide specific criteria to guide opinions offered within their jurisdiction. For example, eligibility for U.S. Black Lung Benefits under the Federal Coal Mine Safety and Health Act are guided by published Department of Labor regulations, which include an extensive preamble reviewing the established science on which medical opinions should be based (89). Mining exposures must be a substantially contributing cause of a miner’s totally disabling respiratory or pulmonary impairment. The Department of Labor provides a listing of the clinics in each district that are certified as meeting testing standards to evaluate miners for benefits eligibility under the Federal Black Lung Act (http://www.dol.gov/owcp/dcmwc/blcontac.htm).

In addition to compensation, miners with occupational lung disease that interferes with work or causes disability may be eligible for rehabilitation and/or retraining. Clinicians should consult the specific rules, criteria, and timelines of state and federal programs (89). A network of clinics offers specific experience and resources in evaluating, treating, counseling, and rehabilitating current and retired coal miners (http://www.cdc.gov/niosh/topics/surveillance/ords/cwsp-resources.html).

CONCLUSIONS

The spectrum of lung disease associated with coal mine dust exposure is broader than generally recognized and includes classic CWP, silicosis, and mixed dust pneumoconiosis as well as dust-related diffuse fibrosis (which may be clinically indistinguishable from idiopathic pulmonary fibrosis absent the exposure history or pathologic evidence). Radiographic opacities in CWP occur frequently in the lower zones and often appear predominantly irregular in shape, not just rounded (25). Progression from a normal chest radiograph to advanced pneumoconiosis may be considerably more rapid than the 15 to 25 years previously considered typical. Among coal miners, dust inhalation is also an important cause of emphysema and COPD, and the rate of the associated functional decline is similar for dust exposure and tobacco smoking. Individual miners often present with multiple effects of coal mine dust, including bronchitis, interstitial lung disease, and emphysema, a spectrum of overlapping diseases appropriately categorized as coal mine dust lung disease.

Coal miners’ lung diseases are not only of historic interest; they are a concern in the 21st century. The increasing CWP prevalence (Figure 1) is reminiscent of the “U-shaped Curve of Concern” described in the 1990s by Reichman (90) in reference to resurgent tuberculosis. The vigorous societal efforts required to control a disease are too often cut prematurely when disease rates decline, with the result of disease resurgence.

The cause of the recent resurgence and severe forms of CMDLD is likely multifactorial. Flaws have been recognized in existing regulations, dust control practices, and enforcement (28, 91, 92). Depletion of thicker coal seams has expanded the mining of thinner seams, increasing miners’ exposure to silica from adjacent rock (93). Smaller mining companies may have fewer health-directed resources (40, 94). Given our continued reliance on coal as an important source of energy, physicians must remain vigilant in recognizing the continuing and evolving patterns of illness among those individuals who labor to furnish this valuable resource.

Author disclosures are available with the text of this article at www.atbjournals.org.


