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Coal Workers' Lung Diseases

Gregory R. Wagner • Michael D. Attfield • James A. Merchant

Historical Perspective

Lung disease among underground coal miners has been a recognized occupational hazard since at least the mid-seventeenth century. Miners' black lung, now called coal workers' pneumoconiosis (CWP) was first documented among Scottish coal miners in 1836.[1] Although the disease was thought to be disappearing in Britain at the turn of this century, wider use of chest radiographs following World War I showed pneumoconiosis, similar to silicosis, among coal miners in South Wales. By 1934, British physicians were beginning to accept coal dust as an occupational exposure that could result in disability and death. In 1942, the Committee on Industrial Pulmonary Diseases of the Medical Research Council introduced the term "coal workers' pneumoconiosis."[2,3]

In marked contrast, appreciation of CWP as an occupational disease and public health problem occurred much later in the United States, as did legislation to prevent CWP or to compensate for CWP and associated respiratory disease. One reason for the relatively late recognition of CWP as a distinct disease entity in the United States was the early emphasis placed on the etiological role of silica in pneumoconiosis. The Hawk's Nest tragedy (1932 to 1934), in which more than 400 workers died of acute silicosis and tuberculosis after working on the tunnel at Gauley Bridge, West Virginia, reinforced the prevalent theory that silica content was the critical etiological agent in pneumoconiosis.

The first systematic study of U.S. coal miners was conducted by the Public Health Service between 1928 and 1931 in the anthracite coal fields in eastern Pennsylvania.[4] Because of the relatively high silica content and similarity to silicosis, the term "anthracosilicosis" was used to describe the pneumoconiosis found among those miners. Of 2,711 men studied, 23 percent were found to be affected. The prevalence of pneumoconiosis was related to the number of years underground, particles per cubic meter, and free silica content of the dust. "Pulmonary infection" was more frequent among miners with higher dust exposure and more than 15 years underground. Among miners over age 55, pulmonary tuberculosis was as much as 10 times more common than in the general population.[5]

Little additional progress was made in the United States until 1954, when the Public Health Service published a bibliography of American and British reports on respiratory disease among coal miners.[6] Following this, various clinical and epidemiologic studies by Levine and Hunter,[7] Lieben et al.,[8] and Stoeckle et al.[9] further documented the importance of coal workers' pneumoconiosis. At the direction of Congress, the Public Health Service began a comprehensive survey of the Appalachian coal fields in 1963. Of 2,549 working miners and 1,191 nonworking miners, 9 percent of the working and 18 percent of the nonworking miners were found to have radiographic evidence of pneumoconiosis.[10] This study, published in 1968, together with the disastrous November 20, 1968, Farmington, West Virginia, mine explosion that killed 78 miners, triggered increased pressure from miners, their union (the United Mine Workers of America), and public health advocates and led to passage of the Federal Coal Mine Health and Safety Act of 1969 (Public Law 1973).[11] This was the first American mining bill to recognize the importance of both health and safety hazards and provide a mandate for strong preventive measures.

Since that time, an awareness has grown indicating that CWP is not the only occupational pulmonary disease affecting coal miners. The results of the study by Rogan and colleagues[12] were the first to show a clear link between chronic airflow obstruction and dust exposure, independent of CWP status, while Rae et al.[13] demonstrated that respiratory symptom prevalence was related to level of dust exposure. Emphysema is increased in coal miners,[14] and related to both FEV_1, retained dust in the lung, and to cumulative dust exposure.[15,16]

Legislation

Although the Federal Coal Mine Health and Safety Act of 1969 was a landmark piece of legislation, it was by no means the first or last legislation to deal with occupational hazards of mining (Table 20-1). The 1969 act addressed several issues specifically and has served as a model for subsequent occupational safety and health legislation. The provisions included the following:[17]

- Mandatory health standards to be prescribed by the Secretary of Health and Human Services (HHS)
- Right of entry for inspection (Department of Interior) and investigation (HHS)
- Power to close mining operations, issue abatement orders, and penalize operators for noncompliance
- A respirable dust standard of 3 mg/m^3 to be reduced to 2 mg/m^3 3 years after passage of the act
- Medical surveillance of underground coal miners through entry and periodic medical examinations
- Rights of miners (transfer rights) with evidence of pneumoconiosis to work in a low-dust area (now <1 mg/m^3) with increased dust monitoring. If job transfer is necessary, there is no loss of pay (rate retention)
- Autopsies on deceased miners, administered by the National Institute for Occupational Safety and Health (NIOSH) through the National Coal Workers' Autopsy Study
- Compensation for miners with total disability and for dependents of miners who die of lung disease from coal mine employment
- Research and training

The medical surveillance provisions of the act were implemented through specifications developed by the NIOSH Appalachian Laboratory for Occupational Safety and Health in August 1970. Since that date, more than 350,000 examinations have been performed.

TABLE 20-1. COAL MINING HEALTH AND SAFETY LEGISLATION IN THE UNITED STATES

1865:	Bill is introduced to create Federal Mining Bureau. It is not passed.
1910:	Bureau of Mines is established but specifically denied right of inspection.
1941:	Bureau of Mines is granted authority to inspect, but it is not given authority to establish or enforce safety codes (Title I, Federal Coal Mine Safety Act).
1946:	Federal Mine Safety Code for Bituminous Coal and Lignite Mines is issued by the Director, Bureau of Mines (agreement between Secretary of the Interior and the United Mine Workers of America) and included in the 1946 (Krug-Lewis) UMWA Wage Agreement.
1947:	Congress requests coal mine operators and state agencies to report compliance with the Federal Mine Safety Code; 33 percent compliance is reported.
1952:	Title II of the Federal Coal Mine Safety Act is passed. All mines employing 15 or more persons underground must comply with the act. Enforcement is limited to issuing orders of withdrawal for imminent danger or for failure to abate violations within a reasonable time.
1966:	Amendments to 1952 law are passed. Mines employing under 15 employees are included under 1952 act; stronger regulatory powers are given to Bureau of Mines, such as the provision permitting the closing of a mine or section of a mine because of an unwarrantable failure to correct a dangerous condition.
1969:	Federal Coal Mine Health and Safety Act is passed. The hazards of pneumoconiosis are, for the first time, given prominence, in addition to those of accidents.
1972:	Black Lung Benefits Act of 1972 is passed. Several sections of the Title IV are amended, liberalizing the awarding of compensation benefits.
1977:	Federal Mine Safety and Health Act of 1977 is passed. It amends Coal Mine Health and Safety Act of 1969 largely by adding health and safety standard setting, inspections, and research provisions for metal and nonmetal miners, while leaving the 1969 act largely intact. This act also consolidates health and safety compliance activities for general industry (OSHA) and mining (MSHA) in the Department of Labor.
1977:	Black Lung Benefits Revenue Act of 1977 is passed. This provides for an excise tax on the sale of coal by the producer to establish trust funds to pay black lung benefits.
1977:	Black Lung Benefits Reform Act of 1977 is passed, to improve and further define provisions for awarding black lung benefits. Additionally, it establishes (a mandate) that a detailed study of occupational lung disease would be undertaken by the Department of Labor and NIOSH.

From Key MM, Kerr LE, Bundy M (eds): Pulmonary Reactions to Coal Dust. New York: Academic Press, 1971, with permission.

Subsequently, Title IV of the 1969 act has been amended twice by Congress, each time modifying requirements that qualify miners for benefits and making coal operators responsible for providing trust funds to pay these benefits. In 1977, the 1969 act was revised and largely incorporated into a new, comprehensive mining law—the Federal Mine Safety and Health Act of 1977, Public Law 91-173, amended by Public Law 95-164, 101[18]—which extended many of the provisions of the 1969 act to metal and nonmetal miners. Significant new responsibilities were given to the Department of Labor (Mine Safety and Health Administration) for establishing health standards and mine inspections and to HHS (NIOSH) for research and surveillance in noncoal mines.

Definition of CWP

CWP is a specific occupational lung disease arising from the prolonged inhalation of coal mine dust. Black lung is a generic term that has been used legislatively and popularly to mean any lung disease that may arise from coal mine employment. This includes both pathologically defined CWP and also obstructive airway disease among coal miners. CWP occurs in two forms: (*a*) simple (chronic) CWP and (*b*) complicated CWP, or progressive massive fibrosis (PMF). The characteristic lesion of simple CWP is the coal macule, which is a focal collection of dust-laden macrophages at the division of the respiratory bronchioles together with associated focal emphysema.[19] Micronodules and macronodules of simple CWP usually are smaller than 1 cm in diameter. Complicated CWP, or PMF, consists of solid, heavily pigmented masses generally greater than 2 cm in diameter, commonly located in the apical region of the lung and occurring on a background of simple CWP.

Environmental Exposures

Significant exposure to coal mine dust may occur not only underground but also in surface strip and auger mines, in coal preparation plants, and in coal-handling operations. U.S. coal reserves are extensive, covering some 400,000 square miles across the country (Fig. 20-1). Coal in the United States may be classified by four ranks: lignite, subbituminous, bituminous, and anthracite, reflecting the degree of metamorphosis of the coal. Anthracite deposits, which are mined on a limited basis only in northeastern Pennsylvania, are associated with the highest rates of pneumoconiosis. Bituminous coals, which are mined from central Pennsylvania westward to Utah are less fibrogenic than anthracite, there being a gradient in toxicity from low volatile bituminous (more fibrogenic) to subbituminous coal (less fibrogenic). Lignite, which also is mined on a limited basis, has not been adequately studied epidemiologically. Workers engaged in face work (coal cutting) and coal preparation often have the highest exposures to respirable coal dust and thus the highest rates of CWP. Drillers and other workers involved in tasks that generate free silica dust are also at risk of contracting silicosis.

Prior to 1970, dust concentrations in face jobs in underground mining were ranging from 6 to 10 mg/m^3. Subsequent to the 1969 act,[11] dust levels were limited first to 3 mg/m^3, and then to 2 mg/m^3. Overall, there is evidence that the regulations have brought about a marked reduction in dust exposures in coal mines,[20,21] although there has been continued concern that overexposure is still occurring.[22,23] The relatively new high-production technology of longwall mining poses a challenge to control engineers for maintenance of exposure levels within the compliance limit.[24]

Surface coal miners generally experience lower levels of dust exposure than do their counterparts underground.[25] Some surface mine jobs, however, can involve very high exposures to silica, especially if dust control measures are missing or ineffective. Drillers, in particular, are at risk of both acute and chronic silicosis, and severe cases have been reported.[26]

Pathophysiology

Pathologically defined simple CWP consists, at a minimum, of the characteristic coal macule lesion(s).[17,19] These may occur as microscopic manifestations of CWP associated with little or no functional impairment. With greater dust deposition in the lung, micronodules

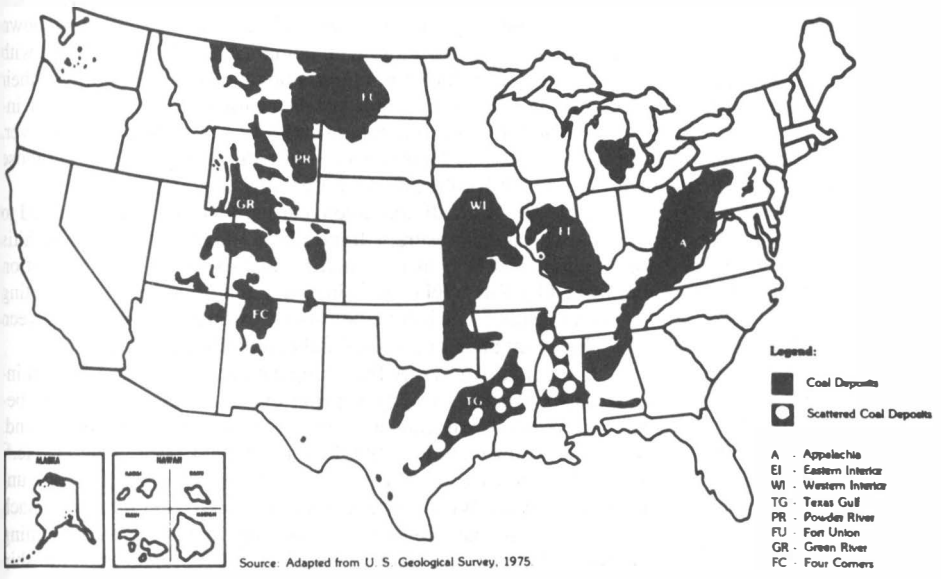

Figure 20-1. Coal deposits in the United States.

(less than 7 mm in diameter) and nodules (larger than 8 mm but less than about 1 cm) are found, predominantly in the upper lung zones (Fig. 20-2). These nodules consist of collagen in addition to a preponderance of reticulin. With increased profusion of nodular lesions in the lung comes greater functional abnormalities, but until marked, CWP often is not associated with significant respiratory symptoms or limiting impairment.

The presence of simple CWP is a significant risk factor for development of PMF; and its probability increases with the severity of simple CWP (Fig. 20-3).[27,28] PMF lesions usually occur in the posterior portion of the upper lobes and in the superior segment of the lower lobes. Unlike silicotic lesions, they cut easily and may have cavities containing inky fluid. The margins may be rounded or irregular, with fibrous strands extending into adjacent lung tissue.

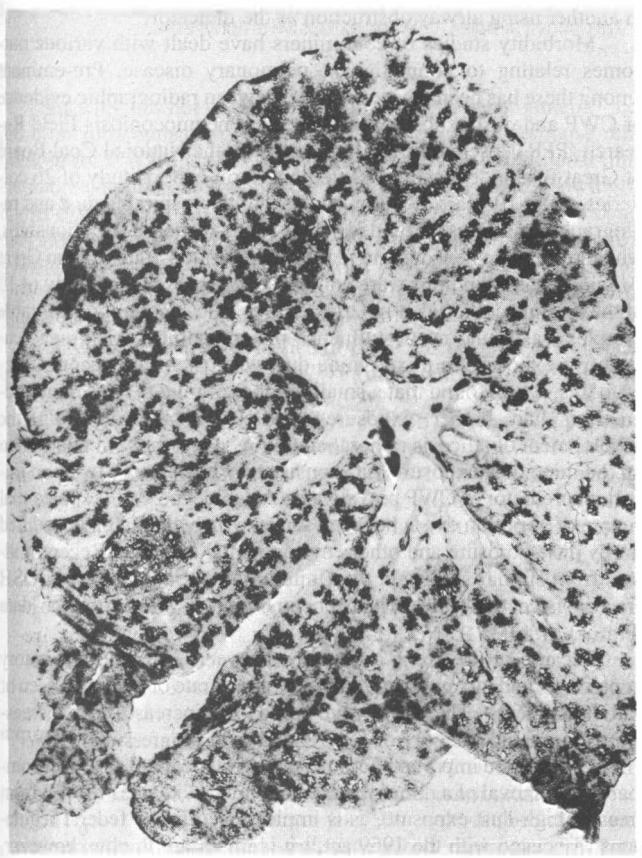

Figure 20-2. Whole lung section showing simple CWP with associated focal emphysema but otherwise preserved lung architecture.

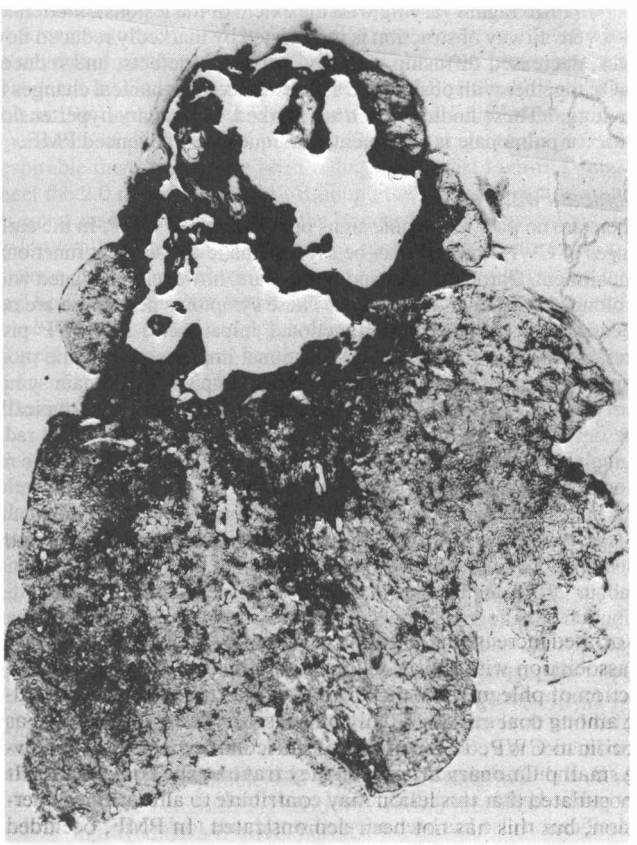

Figure 20-3. Whole lung section showing progressive massive fibrosis with cavitation involving the superior segments of the lung on a background of simple CWP and extensive emphysema.

Caplan's syndrome, consisting of pulmonary nodules associated with rheumatoid arthritis, occurs rarely in coal miners. The nodules, Caplan lesions, are similar to large (up to 5-cm) silicotic nodules on gross examination, usually have smooth borders and concentric internal laminations, and in contrast to PMF lesions, often have little dust contained with the lesion.[19]

Although other forms of emphysema occur in coal miners as they do in the general population, focal emphysema is integral to the coal macule (Fig. 20-2). Focal emphysema is associated with local loss of elastic fibers and alterations in capillary density. The panlobular, irregular, centrilobular, and bullous emphysema associated with these massive lesions is often extensive and destructive; it frequently results in marked pulmonary impairment.[19] Increasing pathological and physiological evidence has strengthened the view that coal mine dust exposure causes centrilobular emphysema.[29–31] Chronic bronchitis, characterized pathologically by hypertrophy and hyperplasia of the bronchial mucous glands with an associated increase in the goblet cells of the small airways, occurs as a result of dust exposure.[32] Clinically defined as the chronic production of phlegm, chronic bronchitis is a frequent clinical finding among coal miners,[33] and its prevalence and incidence are related to dust exposure.[13,34]

Only one vascular lesion is accepted as specific to CWP. This consists of muscular hypertrophy involving small pulmonary arteries as they traverse the coal macule. It is postulated that this lesion may contribute to alterations in perfusion, but this has not been demonstrated. In PMF, occluded and destroyed blood vessels are common and contribute to right ventricular hypertrophy or cor pulmonale, which is frequent among miners with severe CWP.[19] Physiologically, miners with simple CWP have been found to have increased residual volumes, decreased maximal expiratory flow rates, reduction in PaO_2, increased alveolar arterial oxygen differences, and slight hyperventilation, especially with exercise.[35,36] These findings may be nonexistent or slight in those in the earliest stages of CWP, but become progressively more significant with increasing extent of disease.

In PMF (again varying with the extent of the lesions), moderate-to-severe airway obstruction is manifested by markedly reduced flow rates, decreased diffusing capacity, perfusion defects, and reduced PaO_2, together with obstructive and restrictive mechanical changes in the lung.[35] These findings often are marked. Pulmonary hypertension with cor pulmonale is a frequent consequence of advanced PMF.

Clinical Features

There are no pathognomonic signs or symptoms of CWP. In the early stages of CWP, workers may be asymptomatic and without functional impairment. Chronic cough and phlegm are, however, associated with prolonged inhalation of coal dust. These symptoms per se also are not necessarily associated with functional impairment. As CWP progresses, shortness of breath and functional impairment become more common, yet some miners with advanced simple CWP remain symptom free. Those with PMF, especially those with large lesions, typically present with cough, phlegm, and shortness of breath. The chest radiograph is the standard method for detection of CWP. Although the radiographic examination is somewhat limited in sensitivity, the correlation between the profusion of CWP pathologically and radiographically is reasonably good.[37] An internationally developed and accepted method of radiograph classification distributed by the International Labour Office can be used to describe the extent, size, shape, and distribution of radiographic opacities and also to describe pulmonary, cardiac, pleural, and other thoracic abnormalities that may appear on a chest radiograph.[38] This classification divides simple pneumoconiosis into four major subcategories (0, 1, 2, and 3), each of which is subdivided into three categories (i.e., 1/0, 1/1, and 1/2), resulting in an approximation to a continuous scale. PMF is divided into three categories (A, B, and C), depending on lesion size. Although designed as a tool for public health surveillance and epidemiological investigation, this classification also has been adopted worldwide to describe CWP clinically and for compensation purposes.

Epidemiology

Mortality patterns among coal miners have been studied extensively and have generally shown increased standard mortality ratios (SMRs) for accidents, respiratory disease, respiratory tuberculosis, and stomach cancer.[39–43] Mortality rates by major radiographic category have shown significant excesses for those with complicated CWP over those with category 0,[44] particularly for miners who developed PMF early in their working life.[45] Little evidence has been found for a gradient of increasing mortality with increasing category of simple CWP, however, although Miller and Jacobsen showed reduced survival among those with simple CWP compared to those with category 0.[45]

Mortality from all nonviolent causes was found to be related to cumulative dust exposure.[45] Importantly, mortality from bronchitis and emphysema was also related to dust exposure, an observation confirmed by Kuempel et al. using both underlying and contributing causes of death.[46] The latter study also showed a relationship between mortality from pneumoconiosis and cumulative dust exposure.

In the main, mortality from lung cancer in coal miners is not increased, but there is widely varying evidence regarding a link between CWP and lung cancer. In studies where excesses were found, lack of control for confounding factors may have been responsible.[47] Using detailed case-control methods, Ames and colleagues were unable to detect a CWP-lung cancer relationship. By contrast, stomach cancer mortality has been almost uniformly increased in coal mining cohorts in both Britain and the United States,[39,40,43] and a relationship with dust exposure has been detected.[45] Ong and coworkers[48] have hypothesized, supported by laboratory mutagenesis data, that compounds in coal may undergo intragastric nitrosation or interaction with exogenous chemicals or both to form carcinogenic compounds that may with time cause stomach cancer. The Meyer hypothesis,[49] which posits that miners with good lung clearance are at increased risk of stomach cancer because of ingestion of cleared dust while those with impaired clearance get nonmalignant lung disease, has been invoked as one explanation of the increased mortality from stomach cancer in coal miners. This hypothesis was confirmed in one analysis using CWP as an indicator of impaired clearance,[50] but not in another using airway obstruction as the indicator.[51]

Morbidity studies of coal miners have dealt with various outcomes relating to nonmalignant pulmonary disease. Pre-eminent among these has been the association between radiographic evidence of CWP and dust exposure. In 1959, the Pneumoconiosis Field Research (PFR), a scientific study initiated by the National Coal Board of Great Britain, began a massive, long-term cohort study of 26 collieries. After 10 years of study, analysis of the respirable dust and radiographic findings provided clear dose-response relationships, which resulted in new dust standards in the United States and in Great Britain.[52] These findings were confirmed in a subsequent study of 10 of the original collieries (Fig. 20-4).[53] Free silica content in respirable samples was found not to influence pneumoconiosis risk, once cumulative exposure to mixed mine dust was taken into account. Despite this, it was found that a small number of miners with rapid progression had higher exposure to free silica, suggesting the development of silicosis rather than CWP.[54] Coal rank, in addition to mixed mine dust exposure, has consistently been found to be an important predictor of CWP prevalence and incidence.[55–57] A substantial degree of variation exists between mines which cannot be accounted for by dust exposure and other environmental factors.[58] Recent findings from similar studies in the United States conducted by NIOSH are consistent with the British pneumoconiosis field research data (Fig. 20-5).[56,59]

Because of the strong association between PMF and respiratory impairment and increased mortality, the attack rate of PMF has been of particular interest. The risk of developing PMF increases with increasing radiographic category of CWP[60] and with progression of CWP.[28] These studies are important because they provide the basis for recommending removal of a miner with radiographic evidence of CWP from areas of high-dust exposure, as is implemented in the federal regulations associated with the 1969 act.[11] It is important to note, however, that recent findings show the potential for PMF to develop in response to dust exposure directly from a background of category 0.[61] This indicates that the incidence of PMF cannot be controlled merely by the prevention of simple CWP. The attack rate of PMF does not appear to depend on presence of pulmonary tuberculosis, as once suspected.[17,19,62]

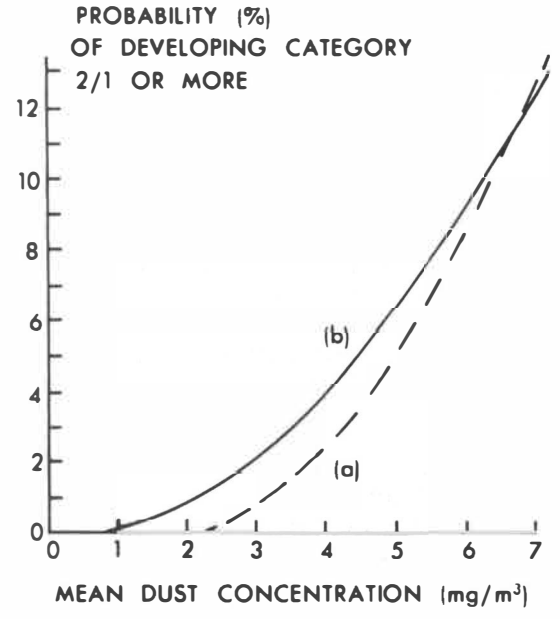

Figure 20-4. Lines (*a*) and (*b*) are estimates of probabilities of developing category 2 or 3 of simple pneumoconiosis over an approximately 35-year working life at the coalface, in relation to the mean dust concentration experienced during that period. (*a*) is based on 10 years of data, Interim Standards Study, Pneumoconiosis Field Research. (*b*) is update of (*a*), based on 20 years of data, Pneumoconiosis Field Research. (From Hurley JF, et al: Simple Pneumoconiosis and Exposure to Respirable Dust: Relationships from Twenty-five Years' Research at Ten British Coal Mines. Institute of Occupational Medicine, Report No. TM/79/13.)

Smoking has not been found to affect CWP development,[63] nor did bronchitis appear to play a role.[64] The exposure-response relationship for CWP and dust exposure is similar for current coal miners and ex-miners, although ex-miners had more disease owing to higher exposures.[65] Although rounded-type small radiographic opacities have been traditionally studied in connection with CWP, there is evidence that small irregular opacities also increase in prevalence with degree of dust exposure.[66,67] Small irregular opacities may be linked with lung function deficits.[68]

While radiographic evidence of CWP has been the major focus of epidemiological research on CWP, much attention has also been paid to coal dust exposure and other nonmalignant lung diseases (including bronchitis, obstructive airway disease, and emphysema). Unlike CWP, these diseases are known to be of multifactorial etiology, including a major influence of cigarette smoking among smokers. Hence their interpretation and significance in terms of occupational exposure has been associated with some controversy.

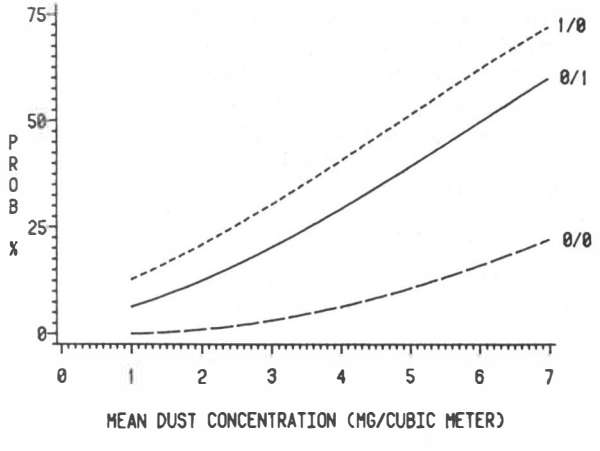

Figure 20-5. Ten-year predicted incidence and progression of CWP for various starting categories (From the Division of Respiratory Disease Studies/NIOSH.)

There is now overwhelming evidence of an exposure-response relationship for ventilatory function and cumulative dust exposure. This has been found in cross-sectional studies,[12,69–71] and in longitudinal studies.[72,73] Smoking was not found to potentiate the effect of dust exposure, nor was presence of CWP a prerequisite for ventilatory function loss. Although the average effect of dust exposure obtained from the exposure-response analyses may appear small, this appearance is misleading, and there is evidence that some miners suffer important deficits in ventilatory function from their work.[27,74] There is no epidemiologic evidence that the effect of smoking and dust exposure differ in nature.[75] Recent evidence suggests that new recruits to mining suffer large initial declines in ventilatory function; these then ameliorate.[34,76,77]

Respiratory symptoms associated with chronic bronchitis have been shown to be related to cumulative dust exposure and its surrogates, in both smokers and never smokers.[13,33,78] The presence of emphysema, as detected on the chest radiograph, is linked with extent of cumulative dust exposure.[79] This finding is consistent with the results of several pathologic studies, which indicate that emphysema is associated with both retained dust and cumulative exposure (or its surrogates) during life.[15,16,80]

Prevention

The key to preventing coal workers' pneumoconiosis is prevention of prolonged inhalation of significant concentrations of coal mine dust. This can be accomplished in two ways (*a*) by the control of respirable coal mine dust through proper ventilation, use of water spray dust suppression, and enclosure of mining operations, or (*b*) by removal of miners with early evidence of CWP to low-dust jobs. Of these two, dust control clearly is more effective. These two provisions were mandated by Congress in the Federal Coal Mine Health and Safety Act of 1969 and have been implemented successfully in underground operations of the U.S. coal industry. Since passage of the 1969 act, respirable dust levels have been reduced for most high-risk jobs to meet the 2.0 mg/mL standard. Although the vast majority of mining sections are in compliance with the standard, certain operations such as longwalls have proved difficult to control. Dust concentration in surface mines has averaged less than half that of underground mining; however, high exposure to coal dust and free silica may occur for those who drill, crush, and prepare coal for transport. NIOSH recently described several cases of acute or accelerated silicosis in young (<35 years old) drillers, and has recommended the use of wet drilling and exhaust ventilation as effective prevention measures.[26]

NIOSH CWP surveillance of U.S. miners has documented decreases in radiographic prevalence of CWP (category 1 or greater) over the period 1970 to 1991 from about 32 to about 20 percent in miners with 25 or more years in mining, and from 7 to about 3 percent in miners with 10 to 14 years in mining (Fig. 20-6). Although there was a sharp decline in prevalence from 1970 to 1980, rates since then have leveled off.

In response to this evidence of limitations in the effectiveness of current U.S. effort to fully control lung disease in coal miners, NIOSH produced new comprehensive recommendations for addressing this problem.[81] This criteria document makes the following recommendations:

- Control of respirable coal mine dust to 1 mg/m³
- Improved engineering control and work practices
- Improved hazard surveillance
- Extension of health screening and surveillance to include tests of pulmonary function with all coal miners being eligible

Recently the U.S. Secretary of Labor empaneled an Advisory Committee on the Elimination of Pneumoconiosis Among Coal Mine

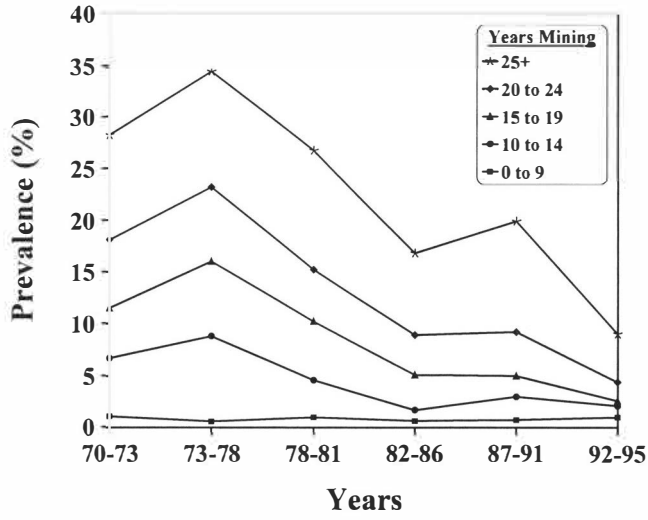

Figure 20–6. Radiographic prevalence of CWP in miners.

Workers.[82] This committee reviewed the scientific data on the causes of disease persistence and issued 20 recommendations. Those include recommendations for improved dust control, and inspection and enforcement of exposures to coal mine dust including silica dust. A strengthened program of medical screening and health surveillance was also endorsed.

Ultimately, improved prevention depends on adoption and application of these recommendations.

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