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Coal mine dust lung disease in the modern era

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Abstract

Coal workers pneumoconiosis (CWP), as part of the spectrum of coal mine dust lung disease (CMDLD), is a preventable and incurable lung disease that can be complicated by respiratory failure and death. Recent increases in coal production from the financial incentive of economic growth correspond to higher respirable coal and quartz dust levels, often associated with mechanization of long-wall coal mining. In Australia, the observed increase in the number of new CWP cases since the year 2000 has necessitated a review of recommended respirable dust exposure limits, where exposure limits and monitoring protocols should ideally be standardized. Evidence that considers the regulation of engineering dust controls in the mines is lacking even in high-income countries, despite this being the primary preventative measure. Also, it is a global public health priority for at-risk miners to be systemically screened to detect early changes of CWP and to include confirmed cases within a central registry; a task limited by financial constraints in less developed countries. Characteristic x-ray changes are usually categorized using the International Labour Office classification, although future evaluation by low dose high-resolution computerized tomography chest scanning may allow for CWP detection, and thus avoidance of further exposure, at an earlier stage. Preclinical animal and human organoid-based models are required to explore potential re-purposing of anti-fibrotic and related agents with potential efficacy. Epidemiological patterns and the assessment of molecular and genetic biomarkers may further our capacity to identify susceptible individuals to the inhalation of coal dust in the modern era.

Header: Coal mine dust lung disease (n=33)

Keywords: coal mine dust lung disease; coal mining; coal workers' pneumoconiosis; health surveillance; respirable dust

Introduction

Inhalation of dust generated by coal mining can lead to the development of coal mine dust lung disease (CMDLD). In addition to the classical coal worker's pneumoconiosis [CWP] and its severe and potentially fatal form, complicated or progressive massive fibrosis (PMF), CMDLD also includes mixed dust pneumoconiosis with co-existent silica exposure, chronic bronchitis, emphysema, and dust related diffuse fibrosis (Table 1).[1-4] Furthermore, dust exposure can adversely affect the lung function of miners in a similar pattern to chronic obstructive pulmonary disease (COPD), particularly for those who have ever-smoked. Thus miners are a high-risk group for respiratory morbidity and premature death. Given the extent of coal mining globally and its potential for a large burden of disease, the respiratory health of coal miners remains an important consideration worldwide.

Trends and distribution of CWP

❖ High income countries

In response to an unacceptable burden of lung disease in coal miners, in 1969, the Federal Coal Mine Health and Safety Act (FCMHSA) of the U.S. established statutory recommendations for respirable dust exposure limits for underground and surface mines.[5] Under the order of the FCMHSA, the National Institute for Occupational Safety and Health (NIOSH) implemented the Coal Workers' Health Surveillance Program (CWHSP) to monitor the reduction in disease that most likely resulted from new dust control regulations. In the U.S., this public health intervention effectively reduced the CWP prevalence to one fifth for underground miners from 11.2% during 1970-1974 to 2.0% during 1995-1999,[6] and reduced mortality from more than 15 to under 5 deaths per million.[7] However since 2000, there has been an observed increase in the number of cases for underground miners even for those aged less than fifty years who would have spent their entire employment under the modern dust control regulations of the of the FCMHSA.[8] This recent increase in prevalence has been attributed in part to

increased coal mine dust levels, longer working hours especially at the face of the mine, increased exposure to crystalline silica and employment in a smaller mine.[8, 9] Progressive mechanization with advancing mining equipment technologies has resulted in more respirable dust being produced by fewer miners.[10]

Unlike the surveillance for underground miners in the U.S., the FCMHSA did not specify periodic health surveillance for surface miners. Comparable prevalence data was lacking until a series of chest radiographs of surface miners was reviewed between 2010 and 2011.[11] Of 2,257 miners with at least 1 year of surface mining experience, 2.0% (n=46) were found to have CWP with 0.5% (n=12) having PMF, of whom most had never worked underground and most were from the central Appalachian region of southern West Virginia, eastern Kentucky, and western Virginia.[6, 8, 10] Thus surveillance is also important to this subgroup of miners.

In Australia over the past century, there has been a similar and dramatic decline in pneumoconiosis-related deaths from any cause, including CWP, which has plateaued to around five deaths per million since 1970.[12] In 2006-2007, 16% of pneumoconiosis-related admissions were attributed to CWP (n=34),[12] which may have been related to longstanding disease. In contrast with the U.S., CWP mortality in Australia has been negligible since the 1970s. [7] Recently however, sixteen cases of CWP have been unexpectedly diagnosed within the Queensland mining industry, where this state produces the majority of Australia's higher quality metallurgical (coking or black) coal from both underground and open-cut mines. These events have raised two issues. First, the recommended respirable dust exposure levels vary between Australian states (2.5 mg/m^3 for New South Wales[13] versus 3 mg/m^3 for Queensland,[14]) compared with 2 mg/m^3 in the U.S that has been reduced to 1.5 mg/m^3 since July 1, 2016. Second, doubts have been raised as to the effectiveness of the modern engineering dust controls in the mines to prevent overexposure of miners to respirable dust. Neither performance nor the effectiveness of these dust controls have been the focus of global regulatory requirements, in spite of a substantial increase in global

production from all forms of mining.

❖ **Low-to-middle income countries**

In China, CWP is the main occupational lung disease and accounts for over half of the total new cases of pneumoconiosis. This equated to 13,955 new cases in 2013.[15] In a systematic analysis of 11 Chinese reports published between 2001 and 2011, the pooled prevalence of CWP was estimated to be 6.0%,[16] based on 10,821 cases in 173,646 dust-exposed Chinese workers. This was relatively higher in local mines compared with state-run mines, and compares with a prevalence of 3.2% in the U.S. and 0.8% in the U.K. during a similar time period.

Although CWP is a serious health issue in other countries of the Asian Pacific region, there are few studies that describe the extent of the problem. A comprehensive report was published from Vietnam almost 20 years ago and this described very high rates of pneumoconiosis from 'special field studies' that were conducted by the Government of Vietnam. This contrasted with official statistics for silicosis from the National Institute of Labour Protection (NILP) which acknowledged these figures were an underestimate of the true frequency.[17] As coal miners with respiratory symptoms were usually funded to have a chest radiograph, many miners with asymptomatic disease were not identified despite the potential benefits from earlier intervention. At the time, traditional manual work was the predominant mining technique, yet the respirable dust concentration in one underground coal mine was reported to be over 30-times higher than the national standard. Silica concentrations in open cut mines were estimated to be between 12 and 28%,[17] so the cases might represent mixed dust pneumoconiosis which is an entity that is now formally recognized within the CMDLD spectrum.

The Global Burden of Disease (GBD) study estimated the age standardized death rate per 100,000 persons to halve from 0.8 in 1990 to 0.4 in 2013, largely for low-to-middle income countries.[18] However this mortality data was collected at two time-points, and may not have been substantially influenced by the recent upward trend in CWP prevalence.

Clinical, pathological and radiological features of CWP

❖ "Phenotypes"

Over the years, CWP has been typically diagnosed in a coal miner who has had considerable dust exposure accompanied by compatible chest radiographic findings,[2] The latency period is usually more than 10 years, although it has been reported for miners working as few as six years in Vietnam.[17] The recent trend of a longer latency in China may relate in part to more effective dust control protocols.[19] A large number of CWP cases are diagnosed after a miner ceases employment.[2] Even in the context of dust exposure at low levels, some miners can develop CWP 15 to 20 years after exposure.[19, 20]

From the series of 495 cases diagnosed between 1963 and 2014 in Eastern China, compared with miners with early or stage 1 CWP, the mean age of diagnosis was earlier for miners with the most severe disease (43.3 versus 52.3 years).[19] This was despite a similar duration of dust exposure. In this same case series, the mean age of death for CWP cases was less than the mean age of CWP survivors which was similar to the baseline population (56.8 versus 71.6 years). This observation of differing lung function trajectories resemblances the traditional classification of simple and complicated CWP (Table 2), with a subgroup of younger and more susceptible miners having a worse prognosis. More recent data has shown consistent decrements in FEV_1% of predicted across a range of radiographic profusion subcategories, and this reflects the progressive nature of the disease.[21]

Of concern is the overrepresentation of younger miners in the subgroup with rapidly progressive CWP, defined by the development of PMF and/or an increase in small opacity profusion greater than one subcategory over five years.[9] Genetic predisposition as discussed below may play a role. Consistent with mixed dust pneumoconiosis, recent evidence from histopathological specimens has found silica to be present in high concentrations in the lungs of workers with rapidly progressive pneumoconiosis. [22]

❖ Pathological features of CWP

Coal dust accumulation in lung tissue leads to a variety of pathological findings from innocuous airway anthracosis to irreversible lung fibrosis and emphysema. Coal dust accumulates in the terminal bronchovascular bundle and is engulfed by alveolar and interstitial macrophages which result in the formation of pulmonary macules and nodules with deposition of dense collagen fibers.[23] Focal emphysema changes around respiratory bronchioles wall can also be observed. The stimulated release of pro-inflammatory cytokines including tumour necrosis factor-α is a potential biomarker of lung pathology,[24] and iron within coal dust may reflect the extent of exposure and be a marker of oxidative lung damage.[1, 25]

❖ Radiological features of CWP

Classical chest x-ray reticulonodular features are typically categorized according to the ILO (International Labour Office) International Classification of Radiographs of Pneumoconiosis, and this was updated in 2011 to extend its applicability to digital images.[26] As distinct from the ILO classification system, the Chinese National Diagnosis Criteria of Pneumoconiosis (GBZ 70-2009) is used in China. The diagnosis can be made by consensus readings, expert panel readings, independent B reading and final determinations derived from multiple independent readings.

Both CWP and silicosis are typically characterized by small (<1 cm) nodular interstitial opacities in the upper zones. However, radiological features of CWP may not be typical. An evaluation of the U.S. Coal Workers Health Surveillance Program (CWHSP) over 30 years found 38% of coal miners with radiographic interstitial changes had predominantly irregular opacities, of whom 41% were largely confined to the lower lung zones.[27] These irregular opacities on plain films are also a feature of mixed-dust pneumoconiosis, as opposed to the predominance of rounded opacities in silicosis.[28] Dust diffuse fibrosis (DDF) is a specific form of CMDLD with radiological findings similar to those seen in idiopathic pulmonary fibrosis, including lower lobe interstitial opacities, honeycombing and traction bronchiectasis.[29, 30]

Complicated CWP, or progressive massive fibrosis [PMF], has been observed to occur in around one-third of cases that fulfilled criteria for rapidly progressive CWP,[9] and features the coalescence of smaller opacities to large nodules of at least 1cm in size. [3] In cases of suspected lung malignancy, positron emission tomography (PET) scanning is of limited value because the majority of nodules more than 1cm in diameter can be metabolically active. [31] This can lead to a false positive result as standardized uptake values can overlap with malignant nodules.[1]

❖ Clinical features of CWP

The respiratory symptoms of CWP are non-specific and most overlap with other coal dust related conditions such as chronic bronchitis, COPD and emphysema.[32] Black-pigmented sputum production (melanoptysis) can occur when large nodules necrotized and liquefied into the airways. Lung function patterns include chronic airflow obstruction, true lung restriction, mixed ventilatory defects, and reductions in carbon monoxide diffusing capacity, [1, 33] and these can vary according to the inhaled silica content, stage of disease and extent of tobacco smoke exposure. Even in the presence of radiologically confirmed pneumoconiosis, airflow obstruction seems to play a predominant role in coal workers breathlessness.[34] Coal miners are frequently exposed to cigarette smoke, diesel exhaust and bio-aerosols and this can make it difficult to determine the role of coal mine dust in miners who develop COPD. In a series of 722 CWP autopsies, Kuempel and colleagues recently demonstrated that coal mine dust exposure was associated with a greater risk of developing emphysema compared with cigarette smoking in their population.[35] Furthermore, coal mine dust exposure is a predictor of chronic bronchitis and emphysema mortality even in non-smokers.[36, 37]

High resolution computed tomography of the chest (HRCT) should be performed in coal workers with either borderline interstitial findings on plain radiographic films or in whom other diagnosis like neoplasm, vasculitis, hypersensitivity pneumonitis or mycobacterial infection need to be considered. Open lung biopsy

or bronchoscopy sampling is usually not necessary in patient with significant exposure and typical imaging.

Cardio-pulmonary exercise testing can be used to assess a patient's physical limitations and the impact of lung disease. In CWP patients, a high ventilatory equivalent ratio for oxygen (suggesting mismatch between ventilation and perfusion) has been found to be the best predictor of dyspnea severity when compared with other functional indices.[34] An early and accelerated FEV_1 (forced expiratory volume in one second) decline has been suggested to relate more closely to small airways disease than to either emphysema or fibrotic lung disease.[38] Interestingly, recently published results in a broader mild to moderate COPD population with functional assessment of small airways with expiratory and inspiratory CT technique were consistent.[39] Early recognition of small airways disease might be beneficial in detecting workers at risk of COPD development; these observations need to be reproduced by a larger cohort with detailed functional and radiological assessment of the small airways.

Severe pulmonary fibrosis associated with significant impairment in gas exchange may lead to chronic hypoxemia, pulmonary hypertension and right heart failure. Particularly for low-to-middle income countries, CWP can be complicated by *Mycobacterium tuberculosis* superinfection, especially when associated with silica exposure and more advanced stages.[19, 20]

Environmental risk factors

The early development of simple CWP is regarded to be the most important risk factor for the development of complicated pneumoconiosis which is closely related to the intensity and duration of respirable dust exposure.[40] Modern mining technology used globally that has the capability to generate high volumes of coal per shift has been identified as a major determinant for the recent increase in CMDLD. In Australia, for example, the development of medium and thick seam mines has allowed the installation of bigger and more productive longwall equipment. Such longwall mining practices has been compared with

other underground mining techniques in Table 3.

In terms of respirable coal (and silica) dust exposure, besides the type of mine, important factors to consider include the profile of the coal; tenure and hours of employment; job type ranging from rock driller to dozer operator; job duties such as tunneling, set explosives or shoveling coal; and the pattern of respirator or mask use (Figure 1).[2] Diesel-powered machinery can generate diesel exhaust particulates and transporting coal out of the mine can disperse particles further. Specific to underground mines, the type and effectiveness of the engineering dust controls, mining technique, coal seam height, time spent at the coalface, the use of powered air-purifying respiratory helmets, and adequacy of dust control with water sprays and ventilation systems are highly relevant. For surface mines, factors to take into account include the amount of dust in open or enclosed cabs while operating heavy machinery, and the time spent outside cutting, drilling or blasting rock.[2]

With the progressive mining of thinner coal seams by the industry, particularly in the U.S., an increased co-existence of silica-related fibrosis can contribute to CWP.[7] This in part explains the higher prevalence of CWP in the U.S. in spite of lower recommended respirable dust limits, where the 2 mg/m^3 limit was based on a 1.4% risk of PMF for miners working with medium to low rank coal.[41] The practice of roof bolting that is particularly frequent in the U.S. is closely linked to silica exposure as these miners work outside the coal seams in quartz-containing rock.[2, 10]

Prevention

There is no cure for CWP, therefore prevention is crucial. CWP can be associated with reduced lung function, even prior to the development of chest radiograph changes.[42] This provides an opportunity to monitor lung function by means of serial spirometric measurements ranging from annually to 3-yearly, in addition to radiographs.[43] This is currently recommended for U.S. miners,[1] particularly if

symptomatic of dry cough and/or progressive breathlessness on exertion. The early identification of CWP is essential to implement appropriate management and make potential changes to employment. However, even after dust exposure has ceased, PMF can appear or if present, progress as manifest by an accelerated decline in lung function and/or development of typical radiographic changes. A comprehensive and coordinated approach to regular screening is important to secondary and tertiary preventative strategies that can improve quality of life and life expectancy. This may be limited by financial constraints in low-to-middle income countries, but this is especially important as respirable dust concentrations can be relatively high and pulmonary tuberculosis may complicate advanced CWP and mixed dust pneumoconiosis in up to 6-7% of cases,[19]

Mining operators of all underground and surface mines have a responsibility to ensure that respirable coal dust and silica exposures remain below recommended or regulatory levels. In some countries, compliance sampling methods that average respirable dust concentrations may conceal instances of several random samples that are well above the limit,[7] which may otherwise be detectable by continuous monitoring. Between 2012 and 2014 in Queensland, Australia, the estimated mean respirable dust concentration for workers in longwall production was below the recommended limit of 3 mg/m^3, but periodically this level peaked above 6 mg/m^3. These exceedences occurred particularly during 2014,[44] and in the absence of a change in monitoring ± measuring practices, this is consistent with progressive mechanization. Whilst engineering dust control equipment and processes are reasonably standardized throughout the world, the equipment and processes implemented can vary from mine to mine,[45] Wearing respiratory protection equipment can limit dust exposure, but a more comprehensive evaluation of the type and practices is needed.

From an Australian perspective, surface and underground coal miners are recommended to be part of a periodic surveillance system to identify early CMDLD,[11] At-risk miners who could be part of a more targeted intervention

include those with early and more prolonged coal mine dust exposure, those with a greater exposure to silica dust and/or tobacco smoke, and especially those with respiratory symptoms. Personal continuous dust monitoring devices is an option to more accurately document and worker's true exposure.[43] As a priority, the surveillance system requires a coordinated approach to the timely reporting of tests and effective communication of potential CMDLD to individual miners.

Although the use of low-dose conventional and HRCT scanning for screening purposes has not been accepted internationally,[1, 46] it offers the advantage of being more sensitive at detecting reticulonodular patterns in early pneumoconiosis than plain films alone,[47] Further evaluation of this potential screening tool will need to encompass the management of potentially malignant pulmonary nodules, especially for miners with other known risk factors. At least in Australia, this evaluation would be extended to include the feasibility of conducting these investigations as some miners would need to travel up to 150km to such a facility, and whether it is cost-effective at a population level.

Patients with CMDLD should be offered best supportive care that includes exposure avoidance, smoking cessation, pulmonary rehabilitation, appropriate disease burden compensation, home oxygen and lung transplant evaluation when eligible.

Future scientific approaches

1) Epidemiology and Public Health

Prospective cohort studies of coal miners would ideally have serial lung function measurements and chest radiographs, which can correspond with the testing performed in periodic health surveillance programs. Specifically, adverse respiratory health outcomes such as impaired lung function growth, accelerated lung function decline and/or compatible radiological findings can be used to identify risk factors for early onset disease. To complement routine

questionnaire data including personal smoking history, other epidemiological questions could be incorporated into the periodic health visits to help address formulated research questions. Establishing any evidence of reduced lung potential by comparing coal mine workers with non-mining workers may then influence the medical workforce, industry and government to guide public policy in the interests of coal miners.

For countries that have collected insufficient information about the health of coal miners and burden of pneumoconiosis, well-designed epidemiological studies should ideally be undertaken. Differing radiographic classification is an important consideration for international studies that collaborate with China. As working environments have become more heavily polluted with increasing economic incentives to mine coal, there also should be strong focus on assessing the adequacy of dust monitoring and controls in order to minimize dust exposure wherever possible.

2) Molecular Biomarkers

The primary underlying mechanism of CWP relates to the release of pro-inflammatory cytokines by alveolar macrophages, which eventually lead to interstitial fibroblast formation and coalescing of fibrotic nodules into conglomerate masses. Compared with coal dust, dust from silica is highly fibrogenic and so silicosis as a single entity has been more extensively investigated in animal models and in human cell culture systems. Similarities with a murine-based model of bleomycin-induced fibrosis have been seen.[48]

Currently there are no reliable, validated biomarkers for CMDLD in human cohorts.[24] Many lack specificity, or require invasive testing such as bronchoscopy or lung biopsy. There is, however, extensive preclinical *in vitro* and *in vivo* animal data. Early biomarkers that might correlate with the extent of exposure include those of oxidative damage, antioxidant enzymes, reactive nitrogen species (RNS) and oxygen species (ROS) and the activation of downstream transcription factors such as NFkB and the STAT family that activate inflammatory and fibrogenic gene expression programs. While most relate to

silicosis, the oxidative damage marker of iron within coal dust appears to correlate well with rates of pneumoconiosis in different mining regions, especially the more bioavailable and complex forms.[1, 25] Tumour necrosis factor-α (TNF-α) and its receptor are potential candidate biomarkers that might reflect lung pathology at a cellular level before the development of CWP and silicosis.[24] TNF-α and interleukin-8 are pro-inflammatory cytokines which have been associated with the presence and progression of CWP, although both still require validation using well-designed and adequately powered prospective cohort studies.[24, 49, 50]

3) Genetic testing and genomic biomarkers

Genetic-based research using case-control study data has been largely been performed in China, and has focused on the potential for genetic factors to predispose to CWP particularly in relation to inflammation and silicosis. No genomic markers, however, have been validated to date.[1] This includes potential susceptibility loci from genome wide association studies (GWAS, rs73329476, rs4320486, and rs117626015),[51] and the T1559C/rs5368 polymorphism.[52] Regarding TNF-α, the TNF-α −308A allele has been linked to ever having CWP especially with nodules as opposed to PMF,[53] whereas TNF-α promoter TNF2 polymorphism has been associated with the development of large opacities.[54]. Other identified polymorphisms include IL-4 C-590T[55]; cyclo-oxygenase-2 rs689466 and rs20417[56]; microRNA-149 rs2292832 TT[57]; a potentially protective effect of MMP3 rs522616 GG[58]; and NLRP3 rs1539019, especially in early disease.[59] The latter is of particular interest given evidence implicating the NALP inflammasome in silicosis, activation of the Nalp3 inflammasome by silicates,[60, 61] in the context of mixed dust pneumoconiosis. However, genetic candidate markers may be most relevant to future research that examines gene-by-environmental interactions as contributing factors for rapidly progressive CWP, while considering racially-restricted CWP susceptibility gene polymorphisms may exist.

4) Novel mechanism and drug discovery

Although limited to date, there is great potential for filling the knowledge gap with regard to mechanisms and drug targets specific to CWP. Examples of recent work in murine models of silicosis include the demonstration that the Wnt/β-catenin pathway is required for activation of TGFβ and fibrosis.[62] Dasatinib, the FDA-approved kinase inhibitor, has been shown to ameliorate structural changes in the lungs as well as changing macrophage phenotype in an intervention that commenced once disease had been established,[63] as have annexin A1 mimetic peptides.[64] The assessment of short-term responses of single cell populations in culture systems, known as high throughput screening, is a particularly useful approach to drug discovery. However, silicosis and pneumoconiosis represent multicellular pathological processes that are likely to be more amenable to lower throughput of drug candidates using organoid-on-a-chip type technologies.[65]

Cost and cost-effectiveness strategies

CWP is a progressive, debilitating and potentially fatal disease and the financial burden it forces onto patients, families, Governments and the wider community is costly. The U.S. spends an estimated $US 1 billion annually in treatment and compensation. In China, which has a high prevalence of CWP, the future medical treatment, welfare and associated costs were estimated in 2011 to equate to 0.55% of its 2009 gross domestic product, thereby placing a substantial burden on its economy.[66] Furthermore in China, there have been attempts to economically evaluate preventative strategies including the use of advanced protective equipment.[67]

Even in high-income countries, the increasing CWP prevalence is still of great concern. In Australia, there has been a call to standardize recommended exposure parameters and monitoring procedures, to implement a comprehensive screening program and establish a centralized occupational lung disease register.[33] There is also a need to employ methods such as cost-effectiveness which have been used to evaluate other occupational health

interventions like silicosis.[68] Such an analysis could involve costing this program of prevention and early diagnosis of CWP, PMF and other lung related fibrotic diseases; benefits in terms of potential downstream cost-savings that may arise (reduced hospitalizations); as well as modelling the improvements in health outcomes. The potential benefits from the prevention of these diseases could also be quantified using Quality Adjusted Life Years that capture the likelihood for gains in survival and quality of life from preventing cases of lung related fibrotic diseases that are due to exposure to coal dust. The purpose of such an evaluation would be to measure the impact of these diseases and to look at the cost and cost-effectiveness of strategies for prevention. This would enable governments and industry to assess whether a greater commitment of resources is required given the expansion in industry and re-emergence of some mining-related lung diseases in recent years.

Conclusion

Coal worker's pneumoconiosis in its most severe form can lead to chronic respiratory failure and premature death. A high cumulative exposure to respirable coal and especially silica dust is central to the development of CMDLD, and other possible susceptibility factors are still not well defined. With improved technology of mining equipment to maximize productivity in recent decades, there has been a resurgence of CWP that has attracted media attention and political interest in higher-income countries. Globally, there is evidence to support the need for a greater emphasis on the efficiency and performance of the engineering dust controls in mines and stricter policy to facilitate early detection and timely treatment for miners in the coal industry. Recommendations include more stringent measurement and monitoring of respirable dust exposure levels, a more systematic screening program and a centralized registry of CWP cases. Improved collection of epidemiological data may identify at-risk miners, and in doing so, provide information for health education strategies and a new platform to validate candidate biomarkers and develop experimental models including novel drug discovery; although there are substantial financial barriers for some countries to adequately address the health problems of coal mine workers.[17]

The prevention of CMDLD is an important public health target, and the quality of life cost utility may enable governments and industry to ultimately develop cost-efficient strategies for its prevention.

Key points

- Coal workers pneumoconiosis (CWP) is a preventable and potentially fatal occupational lung disease without a clearly proven effective treatment, that has been incorporated into the spectrum of coal mine dust lung disease (CMDLD)
- After successful control of the disease in some high-income countries, there has been a reappearance or an increase in cases of CWP, in association with increased coal production and greater mechanization of mining techniques
- Compared with Australia, a greater prevalence of CMDLD for mines that have higher respirable silica dust concentrations may reflect a rise in mixed-dust pneumoconiosis-related cases
- In addition to excessive exposure to respirable dusts, increased susceptibility that manifests as a more severe and progressive disease has been seen in younger miners, and the predisposing factors have not yet been well defined
- Effective dust monitoring and control are a crucial step to ensure employer adherence to the recommended respirable dust limits, which is a task that is especially challenging for low-to-middle income countries
- Early disease recognition together with efficient reduction/elimination of ongoing respirable dust exposure is central to minimizing the emergence of the severe form, progressive massive fibrosis
- The rising prevalence in global burden creates an imperative for efforts to discover new and effective treatments
- Future research into the potential use of HRCT chest scanning, epidemiological identification of at-risk miners and validation of candidate biomarkers will advance scientific knowledge that aims to improve the health of miners in the coal industry.

Table 1. The spectrum of CMDLD

Nature of lung function changes	Diagnoses
Normal lung function in most cases	Anthracosis † Chronic bronchitis Caplan's syndrome (rheumatoid pneumoconiosis) ‡
Restrictive pattern	Coal workers' pneumoconiosis (CWP) - Simple CWP - Rapidly progressive pneumoconiosis (RPP) - Progressive massive fibrosis (PMF) Mixed dust pneumoconiosis (MDP) Diffuse dust-related fibrosis (DDF)
Obstructive pattern	COPD, with and without smoking history
Mixed pattern	Restrictive CMDLD with COPD

Definitions of Abbreviations: COPD, chronic obstructive pulmonary disease; CMDLD, coal mine dust lung disease

† Endoscopic and/or pathological diagnosis

‡ Also associated with mild restriction in some cases

Table 2. Epidemiological comparisons between traditional CWP categories that represent opposing ends of the spectrum of CMDLD

	Traditional CWP phenotypes	
	'Simple CWP'	**'Complicated CWP'**
Alternate nomenclature	Anthracosis	Progressive massive fibrosis (PMF)
Age	Tends to affect older miners	Younger miners are over-represented [19]
Symptoms	Few	Longstanding cough +/- exertional shortness of breath
Latency	Typically >10 years	Typically >10 years
FEV_1 decline	Minimal change	FEV_1 decline >60ml/year greater than miners without CWP [38]
Prognosis	No difference in mortality [19]	At risk of respiratory failure and premature death
Intervention	Adequate dust control and health surveillance	Adequate dust control and health surveillance
Definitions of abbreviations: CMDLD, coal mine dust lung disease; CWP, coal workers' pneumoconiosis		

Table 3. Description of mining techniques and procedures [2]

<table>
<tr><td rowspan="3">Underground mining</td><td rowspan="2">‘Room and pillar’ mining
Coal is mined and remaining ‘pillars’ provide roof support</td><td>Conventional mining
Holes are drilled then blasted using compressed air or explosives</td></tr>
<tr><td>‘Continuous’ mining
Steel bolts placed in the mine ceiling to prevent collapse</td></tr>
<tr><td colspan="2">Longwall mining
A specialized machine cuts across the length of the coal face to extract coal
A hydraulic roof support system supports the mine ceiling, which is allowed to collapse as cutting advances</td></tr>
<tr><td>Surface mining</td><td colspan="2">Overburden (topsoil and rock) is removed by drilling, blasting and excavating</td></tr>
<tr><td>Transport</td><td colspan="2">Underground diesel-fueled shuttle cars and conveyor belts transport coal to the surface → preparation plant</td></tr>
<tr><td>Preparation plant</td><td colspan="2">Coal is washed, crushed to size and separated from waste (rock and soil)</td></tr>
</table>

Figure 1. Job-specific environmental risk factors, common and specific to underground and surface mining [2]

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