

Best Practices for Dust Control in Coal Mining



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Best Practices for Dust Control in Coal Mining

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ACRONYMS AND ABBREVIATIONS USED IN THIS REPORT

CWHSP	Coal Workers' Health Surveillance Program
CWP	Coal workers' pneumoconiosis
DO	designated occupation
HVAC	heating, ventilation, and air conditioning
IARC	International Agency for Research on Cancer
ILO	International Labour Office
MSHA	Mine Safety and Health Administration
NIOSH	National Institute for Occupational Safety and Health
PDM	personal dust monitor
pDR	personal DataRAM
PEL	permissible exposure limit
PMF	progressive massive fibrosis
TEOM	tapered-element oscillating microbalance

UNIT OF MEASURE ABBREVIATIONS USED IN THIS REPORT

cfm	cubic foot per minute
cm	centimeter
fpm	foot per minute
ft	foot
ft/min	foot per minute
gpm	gallon per minute
hr	hour
in	inch
in w.g.	inch water gauge
kPa	kilopascal
lpm	liter per minute
m/sec	meter per second
mg/m ³	milligram per cubic meter
mm	millimeter
mph	miles per hour
µg/m ³	microgram per cubic meter
psi	pound-force per square inch
sec	second

BEST PRACTICES FOR DUST CONTROL IN COAL MINING

By Jay F. Colinet,¹ James P. Rider,² Jeffrey M. Listak,³ John A. Organiscak,³ and Anita L. Wolfe⁴

INTRODUCTION

Respirable dust exposure has long been known to be a serious health threat to workers in many industries. In coal mining, overexposure to respirable coal mine dust can lead to coal workers' pneumoconiosis (CWP). CWP is a lung disease that can be disabling and fatal in its most severe form. In addition, miners can be exposed to high levels of respirable silica dust, which can cause silicosis, another disabling and/or fatal lung disease. Once contracted, there is no cure for CWP or silicosis. The goal, therefore, is to limit worker exposure to respirable dust to prevent development of these diseases.

The passage of the Federal Coal Mine Health and Safety Act of 1969 established respirable dust exposure limits, dust sampling requirements for inspectors and mine operators, a voluntary x-ray surveillance program to identify CWP in underground coal miners, and a benefits program to provide compensation to affected workers and their families. The tremendous human and financial costs resulting from CWP and silicosis in the U.S. underground coal mine workforce are shown by the following statistics:

- During 1970–2004, CWP was a direct or contributing cause of 69,377 deaths of U.S. underground coal mine workers.
- During 1980–2005, over \$39 billion in CWP benefits were paid to underground coal miners and their families.
- Recent x-ray surveillance data for 2000–2006 show an increase in CWP cases. Nearly 8% of examined underground coal miners with 25 or more years of experience were diagnosed with CWP.
- “Continuous miner operator” is the most frequently listed occupation on death certificates that record silicosis as the cause of death.

In light of the ongoing severity of these lung diseases in coal mining, this handbook was developed to identify available engineering controls that can help the industry reduce worker exposure to respirable coal and silica dust. The controls discussed in this handbook range from long-utilized controls that have developed into industry standards to newer controls that are still being optimized. The intent was to identify the best practices that are available to control respirable dust levels in underground and surface coal mining operations. This handbook

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provides general information on the control technologies along with extensive references. In some cases, the full reference(s) will need to be consulted to gain in-depth information on the testing or implementation of the control of interest.

The handbook is divided into five chapters. Chapter 1 discusses the health effects of exposure to respirable coal and silica dust. Chapter 2 discusses dust sampling instruments and sampling methods. Chapters 3, 4, and 5 focus on dust control technologies for longwall mining, continuous mining, and surface mining, respectively.

Finally, it must be stressed that after control technologies are implemented, the ultimate success of ongoing protection for workers depends on continued maintenance of these controls. NIOSH researchers have often seen appropriate controls installed, but worker overexposures occurred because of the lack of proper maintenance of these controls.

CHAPTER 1.—HEALTH EFFECTS OF OVEREXPOSURE TO RESPIRABLE COAL AND SILICA DUST

By Anita L. Wolfe¹ and Jay F. Colinet²

Pneumoconioses are lung diseases caused by the inhalation and deposition of mineral dusts in the lungs. Pneumoconioses associated with working in a high-risk, mineral-related industry such as mining are coal workers' pneumoconiosis (CWP) and silicosis. Once contracted, these diseases cannot be cured. Therefore, it is critical to limit worker exposure to airborne respirable dust to prevent these diseases.

COAL WORKERS' PNEUMOCONIOSIS (CWP)

CWP, commonly called black lung disease, is a chronic lung disease that results from the inhalation and deposition of coal dust in the lung and the lung tissue's reaction to its presence. It most often affects those who mine, process, or ship coal. In addition to CWP, coal mine dust exposure increases a miner's risk of developing chronic bronchitis, chronic obstructive pulmonary disease, and pathologic emphysema.

With continued exposure to the dust, the lungs undergo structural changes that are eventually seen on a chest x-ray. In the simple stages of disease (simple CWP), there may be no symptoms. However, when symptoms do develop, they include cough (with or without mucus), wheezing, and shortness of breath (especially during exercise). Figure 1-1 shows a normal lung and a lung from a miner who has been diagnosed with CWP. In the more advanced stages of disease, the structural changes in the lung are called fibrosis. Progressive massive fibrosis (PMF) is the formation of tough, fibrous tissue deposits in the areas of the lung that have become irritated and inflamed. With PMF the lungs become stiff and their ability to expand fully is reduced. This ultimately interferes with the lung's normal exchange of oxygen and carbon dioxide, and breathing becomes very difficult. The patient's lips and fingernails may have a bluish tinge, and there may be fluid retention and signs of heart failure. If a person has inhaled too much coal dust, simple CWP can progress to PMF.

Simple CWP is characterized by the presence of small opacities (opaque spots) on the chest x-ray that are less than 10 mm in diameter. The profusion (density) of small opacities is classified as major category 1, 2, or 3 as defined by the International Labour Office (ILO) guidelines [ILO 1980]. Category 0 is defined as the absence of small opacities or opacities that are less profuse than the lower limit of category 1. Within the 12-point ILO profusion scale, each major category may be followed by a subcategory, if an adjacent main category was considered during classification (e.g., classification 1/2 was judged as category 1, but category 2 was seriously considered) [NIOSH 1995].

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Figure 1-1.—Normal lung (*left*) and a lung from a miner diagnosed with CWP (*right*).

PMF is classified radiographically as category A, B, or C when large opacities with a combined area of 1 cm or larger are found on the chest x-ray. PMF usually develops in miners already affected by simple CWP, but can develop in miners with no previous radiographic evidence of simple CWP [NIOSH 1995].

There is no specific therapy for these diseases. Primary prevention of lung disease in miners must include continued efforts to reduce coal mine dust exposure. Medical management is best directed at prevention, early recognition, and treatment of complications. The major clinical challenges are the recognition and management of airflow obstruction, respiratory infection, hypoxemia (an abnormally low amount of oxygen in the blood), respiratory failure, cor pulmonale (enlargement of the right side of the heart), arrhythmias (abnormal heart rhythm), and pneumothorax (collapsed lung).

Since passage of the Federal Coal Mine Health and Safety Act of 1969, the Mine Safety and Health Administration (MSHA) enforces regulations designed to limit mine workers' exposure to respirable coal mine dust to 2 mg/m^3 or less if the silica content in the sample is less than 5%. Periodic sampling is conducted by MSHA inspectors and mine operators to demonstrate compliance with this dust limit. In underground coal mines, airborne dust concentrations are typically the highest for workers involved in the extraction of coal at the mining face. Longwall shearer operators, jack setters, and continuous miner operators are occupations with greater potential for exposure to excessive levels of respirable coal mine dust. Workers in some aboveground coal mining operations also have increased exposure to coal mine dust. These include workers at preparation plants where crushing, sizing, washing, and blending of coal are performed and at tipples where coal is loaded into trucks, railroad cars, river barges, or ships.

Also included in the 1969 Act was the establishment of the NIOSH Coal Workers' Health Surveillance Program (CWHSP). As part of this program, underground coal miners are periodically offered the opportunity to voluntarily receive a chest x-ray (free of charge to the miner) in an effort to detect the presence of CWP. The rates of black lung steadily declined through 1999. However, recent data from NIOSH [2008] show that the declines have stopped and rates are actually starting to rise (see Figure 1-2). For miners with 25 or more years of experience who were examined in the CWHSP after the year 2000, the rate of black lung being

found has nearly doubled. In addition, disease is showing up in younger miners, and miners are progressing from the beginning stages of black lung disease to the more advanced PMF at a faster rate. In 2004, the deaths of 703 miners were attributed to CWP. (For additional statistics, see: NIOSH [2008]).

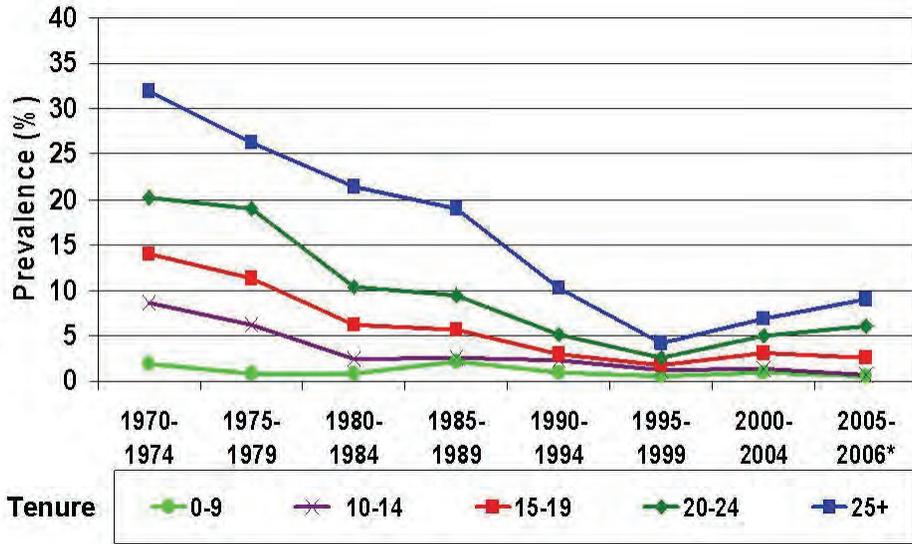


Figure 1-2.—Trends in CWP prevalence among examinees employed at underground coal mines by years of experience (tenure). (Source: NIOSH Coal Workers' X-ray Surveillance Program).

30 CFR³ 90 establishes procedures for miners who have developed evidence of pneumoconiosis to work in an area of a mine where the average concentration of respirable dust in the mine atmosphere during each shift is continuously maintained at or below 1.0 mg/m³. The rule sets forth procedures for miners to exercise this option and establishes the right of miners to retain their regular rate of pay and receive wage increases. The rule also sets forth the mine operator's obligations, including respirable dust sampling requirements for Part 90 miners. The goal is to prevent further development of the pneumoconiosis in the affected miner.

³Code of Federal Regulations. See CFR in references.

SILICOSIS

Occupational exposures to respirable crystalline silica occur in a variety of industries and occupations because of its extremely common natural occurrence. Workers with high exposure to crystalline silica include miners, sandblasters, tunnel builders, silica millers, quarry workers, foundry workers, and ceramics and glass workers. Silica refers to the chemical compound silicon dioxide (SiO_2), which occurs in a crystalline or noncrystalline (amorphous) form [NIOSH 2002]. Crystalline silica may be found in more than one form: alpha quartz, beta quartz, tridymite, and cristobalite [Ampian and Virta 1992; Heaney 1994]. In nature, the alpha form of quartz is the most common [Virta 1993]. This form is so abundant that the term “quartz” is often used instead of the general term “crystalline silica” [USBM 1992; Virta 1993].

Quartz is a common component of rocks. Mine workers are potentially exposed to quartz dust when rock within or adjacent to the coal seams is cut, crushed, and transported. Occupational exposures to respirable crystalline silica are associated with the development of silicosis, lung cancer, pulmonary tuberculosis, and airways diseases. These exposures may also be related to the development of autoimmune disorders, chronic renal disease, and other adverse health effects. In 1996, the International Agency for Research on Cancer reviewed the published experimental and epidemiologic studies of cancer in animals and workers exposed to respirable crystalline silica. The IARC concluded that there was sufficient evidence to classify silica as a human carcinogen [IARC 1997].

Silicosis is also a fibrosing disease of the lungs caused by the inhalation, retention, and pulmonary reaction to the crystalline silica. The main symptom of silicosis is usually dyspnea (difficult or labored breathing and/or shortness of breath). This is first noted with activity or exercise and later as the functional reserve of the lung is also lost at rest. However, in the absence of other respiratory disease, there may be no shortness of breath and the disease may first be detected through an abnormal chest x-ray. The x-ray may at times show quite advanced disease with only minimal symptoms. The appearance or progression of dyspnea may indicate other complications, including tuberculosis, airways obstruction, PMF, or cor pulmonale. A productive cough is often present.

A worker may develop one of three types of silicosis, depending on the airborne concentrations of respirable crystalline silica that were inhaled:

- (1) *Chronic Silicosis*: Usually occurs after 10 or more years of exposure at relatively low concentrations. Swellings caused by the silica dust form in the lungs and chest lymph nodes. This disease may cause people to have trouble breathing and may be similar to chronic obstructive pulmonary disease.
- (2) *Accelerated Silicosis*: Develops 5–10 years after the first exposure. Swelling in the lungs and symptoms occur faster than in chronic silicosis.
- (3) *Acute Silicosis*: Develops after exposure to high concentrations of respirable crystalline silica and results in symptoms within a period of a few weeks to 5 years after initial exposure [Parker and Wagner 1998; Peters 1986]. The lungs become very inflamed and can fill with fluid, causing severe shortness of breath and low blood oxygen levels.

PMF can occur in either simple or accelerated silicosis, but is more common in the latter. Figure 1-3 shows a lung that has been damaged by silicosis.

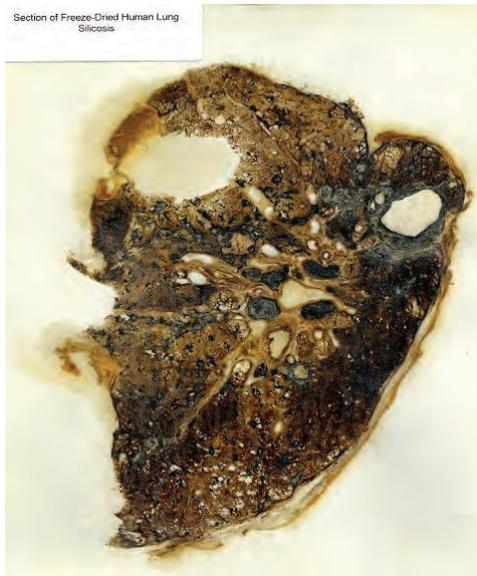


Figure 1-3.—Section of a freeze-dried human lung with silicosis.

In an effort to prevent the development of silicosis, MSHA regulates the exposure of mine workers to silica. For coal mining operations, quartz levels up to 5% in compliance dust samples do not alter the respirable dust standard of 2 mg/m^3 . However, if the percent of quartz in the sample exceeds 5%, a reduced dust standard is calculated by dividing 10 by the percent quartz. For example, if a sample contains 10% quartz, the reduced standard would be equal to 1 mg/m^3 ($10 \div 10\%$ quartz). In essence, these regulations limit the exposure to respirable quartz to $100 \text{ }\mu\text{g/m}^3$, although this limit is not specifically quantified in the regulations.

MSHA compliance sampling data identify those occupations in coal mining that are at high risk for overexposure to quartz. Figure 1-4 shows the percentage of samples collected by MSHA inspectors that exceeded reduced permissible exposure limits (PELs) for several high-risk occupations in coal mining.

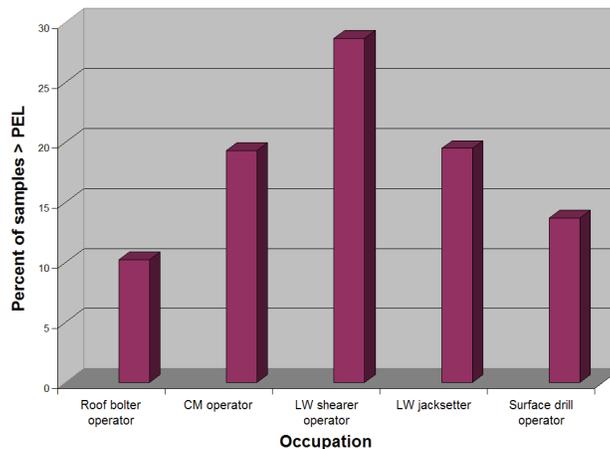


Figure 1-4.—Percentage of MSHA inspector samples during 2003–2007 that exceeded reduced PELs.

DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSES

A doctor may diagnose CWP or silicosis based on the combination of an appropriate history of exposure to coal mine dust or silica, compatible changes in chest imaging or lung pathology, and absence of plausible alternative diagnoses. A chest radiograph is often sufficient for diagnosis, but in some cases a computed tomography (CT) scan of the chest can be helpful. Lung biopsy, a procedure where a sample of lung tissue is taken for lab examination, is not usually required if a compatible exposure history and findings on chest imaging are present. Pulmonary function tests and blood tests to measure the amounts of oxygen and carbon dioxide in the blood (arterial blood gases) can help in objectively assessing the level of impairment caused by CWP or silicosis.

Epidemiologic studies of gold miners in South Africa, granite quarry workers in Hong Kong, metal miners in Colorado, and coal miners in Scotland have shown that chronic silicosis may develop or progress even after occupational exposure to silica has been discontinued [Hessel et al. 1988; Hnizdo and Sluis-Cremer 1993; Ng et al. 1987; Kreiss and Zhen 1996; Miller et al. 1998]. Therefore, removing a worker from exposure after diagnosis does not guarantee that silicosis or silica-related disease will stop progressing or that an impaired worker's condition will stabilize.

Treatment of CWP or silicosis may include use of bronchodilators (medications to open the airways) or supplemental oxygen use. Once disease is detected, it is important to protect the lungs against respiratory infections. Thus, a doctor may recommend vaccinations to prevent influenza and pneumonia. In some cases of severe disease, a lung transplant may be recommended. Prognosis depends on the specific type of pneumoconiosis and the duration and level of dust exposure.

There is no cure for these lung diseases, and they cannot be reversed. Effective control technologies must be implemented and continually maintained to prevent the development of the disease.

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