Coal Dust

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1.0 Coal

1.0.1 CAS Number

NA

1.0.2 Synonyms

Coal, anthracite, bituminous coal, lignite, brown coal, hard coal, soft coal.

1.0.3 Trade Names

NA

1.0.4 Molecular Weight

The molecular weight of coal as mined is indeterminate. Alkylated coal products have molecular weights from 500 to 2000 (low- to high-rank coal) (1).

1.1 Chemical and Physical Properties

1.1.1 General

Coal is an organic, combustible, rock-like natural substance that occurs in various forms from hard and brittle anthracite to soft and friable lignite. Coal is sometimes classified into two types: hard coal and soft coal. These terms do not, however, have a standardized meaning. One definition calls anthracite "hard coal" and places all other coal types in the "soft coal" category. A more common convention is that of Speight (1), in which anthracite and bituminous coals are termed hard coal and lignite and brown coal are classified as soft coal. Research on the health effects of coal has historically been heavily concentrated on the more common anthracite and bituminous types, though in recent years epidemiological studies have included miners of subbituminous coal. Results of these studies have shown more of a toxicological gradient across coal types rather than any clear-cut divisions between types. Little research has been undertaken on lignite and other brown coals. This has prohibited drawing firm conclusions on its toxicity relative to the harder coal types. As a result of the lack of any obvious demarcation, overall patterns of effect are reported rather than separate presentations by coal type. Where data are available, attention is drawn to any findings especially relevant to a particular coal type.

Coal varies considerably in composition and consists largely of carbon, hydrogen, and oxygen with smaller amounts of sulfur, nitrogen, trace elements, and metals. Coal originated from mostly organic material that was long ago buried by sediments. Heat and pressure converted the plant remains over geologic time to coal, the process thought to have led to peat, lignite, bituminous coal, and anthracite in turn. Coal, as extracted, contains many minerals in various proportions, including quartz, clays, carbonates, and sulfides. These minerals can be intrinsic to the coal, as in silica grains within the coal matrix, or may lie in pockets or layers. Although there are different methods for classifying coal—each developed for a specific geological or economic purpose—all tend to relate to the age of the coal. Older coals are purer, harder, more brittle, have less volatile matter, and have higher calorific value. They also contain higher amounts of fixed carbon. Fixed carbon is the basis of a commonly

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Table 1. Minerals in Coal Ash[a]

Constituents	Representative Percentage
SiO_2	40–90
AI_2O_3	20–60
Fe_2O_3	5–25
CaO	1–15
MgO	0.5–4
Na_2O	0.5–3
K_2O	0.5–3
SO_3	0.5–10
P_2O_5	0–1

[a]From Speight (1)/Taylor & Francis.

used classification index of coal–coal rank. High-rank coals, such as anthracite, have the greatest amount of fixed carbon, whereas bituminous and subbituminous coals are defined as low rank. A rank-based system divides coal into four classes: anthracite, bituminous, subbituminous, and lignite (2). Each class is further divided into groups, using fixed carbon, moist Btu, or other factors. For example, the main anthracite class is divided into meta-anthracite (98% or more fixed carbon), anthracite (92–98% fixed carbon), and semianthracite (80–92% fixed carbon), while the lignite class is split into lignite (also termed consolidated) coal and brown (or unconsolidated) coal.

Exposure to "pure" coal dust, that is, the dust generated from handling washed and cleaned coal, is rare. Most occupational exposures are to coal mine dust, an entity with varying properties, that contains coal and dust generated from the accidental or intentional cutting of the rock strata adjacent to or within the coal seam. The rock strata can comprise limestone, shale, clay, or sandstone and therefore can give rise to silica and other dusts, which mix with the coal dust to give coal mine dust exposures. Table 1 shows the major inorganic constituents of coal ash. Virtually all the findings on health outcomes in humans are derived from studies of exposures to coal mine dust.

1.1.2 Odor and Warning Properties

Although there are no specific odors and warning properties for coal dust, coal dust can be an explosion hazard at high concentrations. This is not likely when airborne concentrations are below the health-based recommended exposure limits (REL) (Tables 5 and 6). However, the potential for this hazard is real (3), particularly in areas where large amounts of settled dust may be disturbed (e.g., by a blast of air) and become airborne. Factors that influence the explosion hazard of coal dust include the particle size, volatility, and copresence of flammable gas, such as methane. The minimum explosible concentrations of pulverized bituminous coal dusts are of the order of 50–100 mg/L (equivalent to 50–100 g/m^3) (4). These explosible concentrations of coal dust have been estimated to be at least 10,000 times that of typical airborne respirable concentrations.

1.2 Production and Use

Coal occurs in all continents and is mined in all but Antarctica. Currently, China accounts for more than half of the world's coal production. India is the second highest producer at 10% and the United States produces 9% of worldwide coal (5). Other countries that have significant production are Indonesia, Australia, Russia, South Africa, Germany, Kazakhstan, Poland, and Turkey (5). In the United States, coal mining, already of major importance by the year 1800, increased rapidly. Coal mining employment peaked in 1923, when 700,000 miners were employed. In 2019, the U.S. Energy Information Administration reported a continued decline in employment to 52,804 employees (6). Production and employment are projected to continue to decline as more U.S. coal-fired power plants are decommissioned and projected to retire, or are converted to natural gas as a fuel source (7). The major coalfields in the United States are shown in Figure 1. In 2019, 48% of total production comprised bituminous coal, 44% subbituminous, 8% lignite, and <0.1% anthracite (6).

Coal exists as distinct seams of mineral laid down as sedimentary deposits within the earth. The seams are extracted by surface or underground mining. Surface mining generally involves the removal of rock and other strata to uncover the top or side of a coal seam (Figure 2). Using blasting and/or mechanical means, the coal is fractured and removed from the mine by train, truck, or conveyer directly to a nearby power plant. In recent years, about two-thirds of U.S. coal production is from surface mines. There are two main types of underground mining: room and pillar and longwall (accounting for 60% and 39% of U.S. production in 2015, respectively) (8). Room and pillar mining is the traditional method, whereby pillars of coal are left to support the roof (Figure 3). These pillars may later be removed, a process that provides better reclamation of the coal but speeds up subsidence of the overlying strata. Room and pillar operations are commonly undertaken using continuous mining, where a machine with a rotating cutting head removes the coal from the coal face. Longwall mining, of more recent origin, involves removing "slices" of coal from the edge of the seam by large coal cutting machines (Figure 4). All of the coal is extracted, and the roof is left to collapse as the coal face advances. After extraction, coal is typically crushed and cleaned. Cleaning can involve screening for size, removal of impurities by wet or dry methods, and drying.

Historically, coal was the principal energy source in factories and in transportation, but nowadays the main use for coal is in electricity generation (92% in the United States in 2019) (9). Other uses include industrial use (e.g., chemical plants), production of coke, and domestic heat and steam generation.

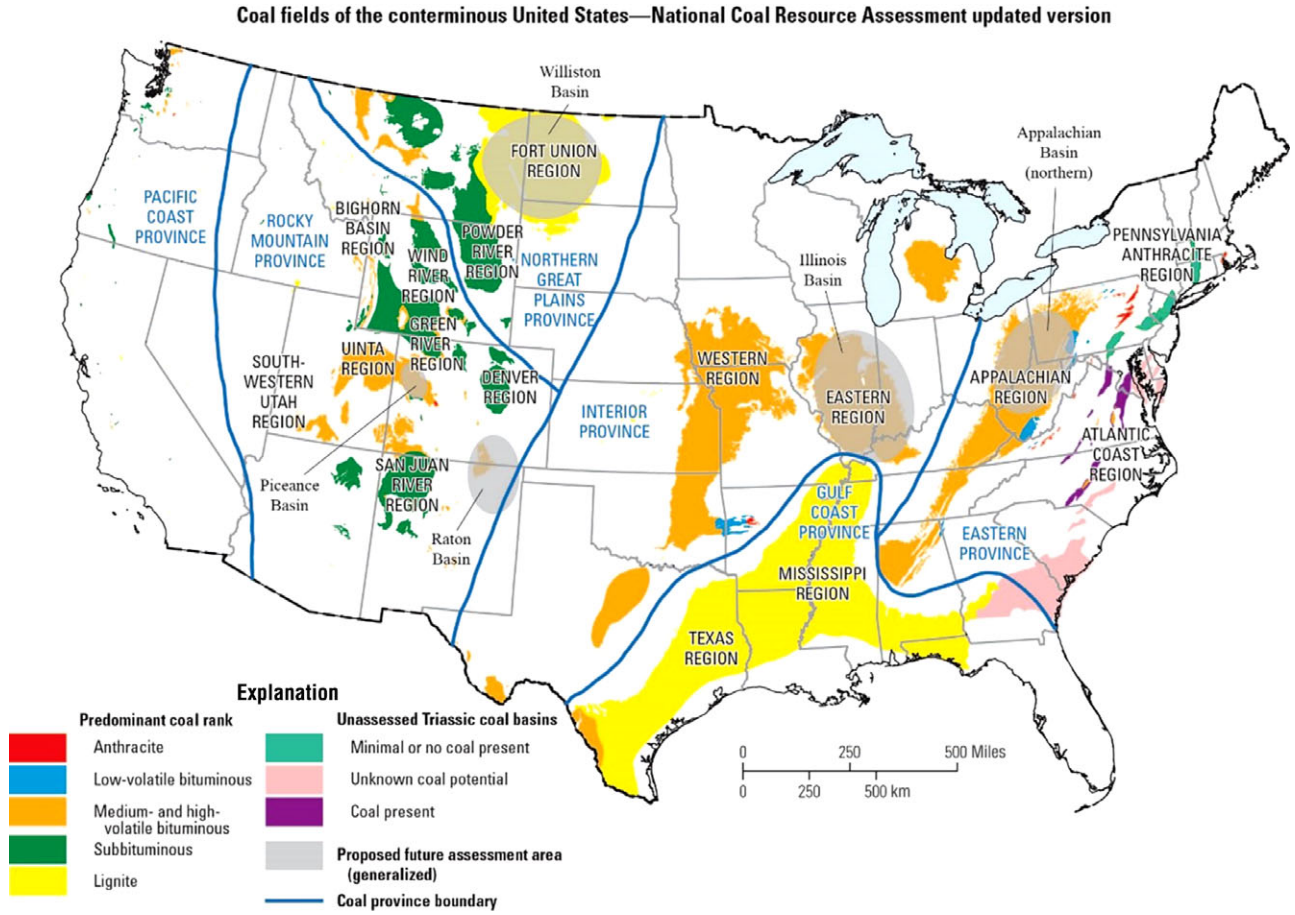

Figure 1. Map of U.S. coal fields. From USGS Coalfields of the Conterminous United States.

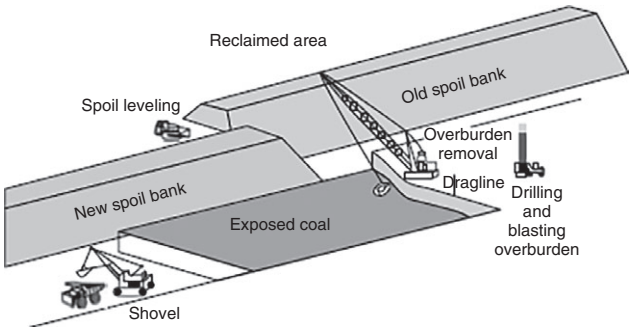

Figure 2. Surface coal mining.

1.3 Exposure Assessment

1.3.1 Air

See Section 1.3.3.

1.3.2 Background Levels

Environmental exposure to coal dust is probably negligible for most of the general population. However, individuals who live near surface coal mines and coal storage piles may experience significant exposure, particularly on windy days (10).

1.3.3 Workplace Methods

Historical measures of occupational coal mine dust exposure were based on particle counts derived from impingers or thermal precipitators. Later, gravimetric samplers were developed, using a horizontal elutriator or cyclone, concomitant with the view that dust mass rather than particle count would be a more appropriate leading indicator of dust toxicity (11). Currently, most compliance measures of coal mine dust exposure are based on personal or area gravimetric determinations of respirable dust concentration, sometimes with adjustments for the silica component of the dust. An important advance in the dust assessment arena has been the development of a continuously measuring personal dust monitor (CPDM) (12). The full shift time-weighted average (TWA) concentration measured by the CPDM is now used by the U.S. Mine Safety and Health Administration (MSHA) to verify the compliance status at the end of the shift. In

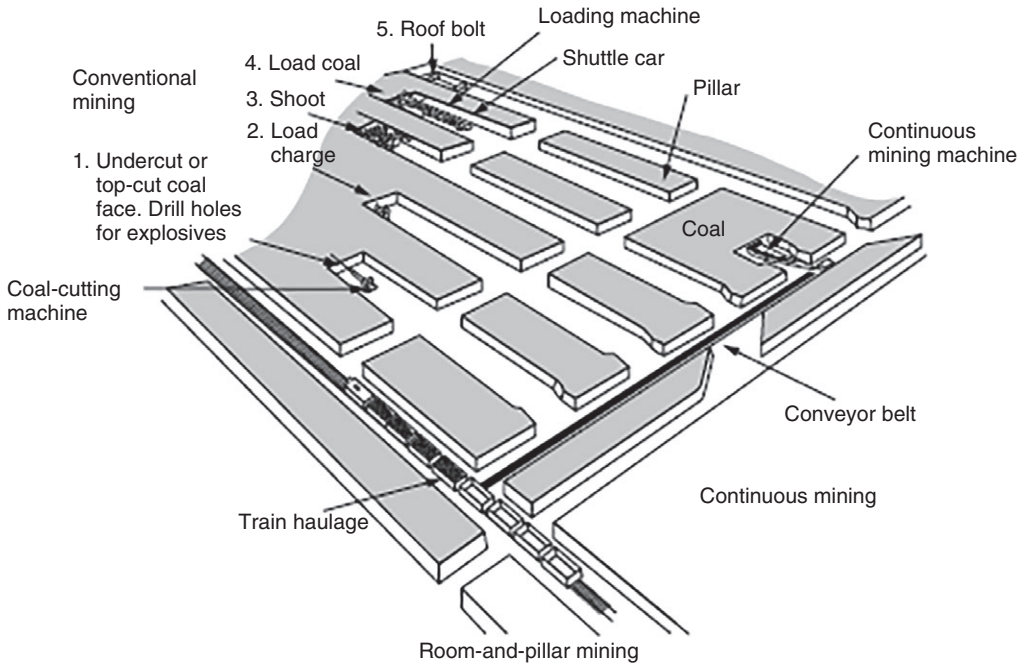

Figure 3. Room and pillar mining.

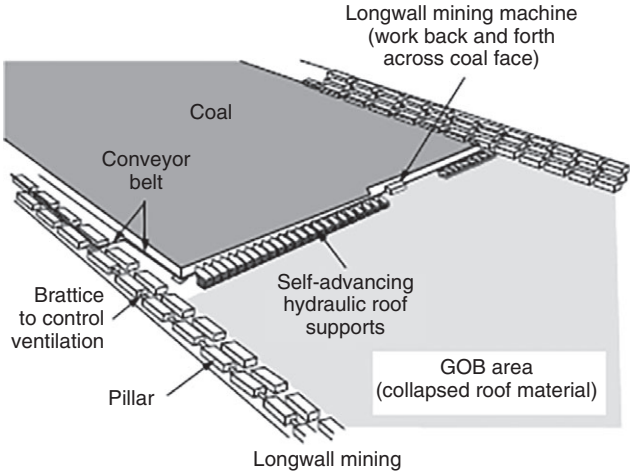

Figure 4. Long-wall mining.

addition, the CPDM enables within-shift assessment of dust exposures, facilitating prompt action to intervene and reduce excessive levels as they occur.

For research purposes, other aspects of coal dust exposure have also been frequently evaluated as strongly associated with toxicity. These include dust composition, particularly with regard to the crystalline silica component. These characterizations have employed X-ray diffraction or infrared methods for analyzing dust collected on filters or bulk samples. Indexes that reflect particle size have also been studied. For example, Cowie et al. examined inhalable dust in connection with upper airway disease (13). The National Institute for Occupational Safety and Health's (NIOSH) Analytical Methods #0600, #7500, and #7603 NIOSH Manual of Analytical Methods (NMAM IV) are recommended for the evaluation of workers' exposure. Recently NIOSH has developed a field-based respirable crystalline silica monitoring approach that can allow coal mine operators to assess the exposure of workers specifically to crystalline silica at the mine site. Compared to monitoring approaches that rely on laboratory analysis of the samples, the field-based approach can provide results at the end of the work shift by using portable infrared analyzers (14, 15).

1.3.4 Community Methods

Community exposures to coal mine dust are not routinely assessed because (*1*) few individuals are exposed to coal dust alone and (*2*) coal dust cannot be differentiated at low levels from other general ambient contaminants. If community exposures specifically to coal dust must be assessed, methods for occupational measurement might be applicable.

1.3.5 Biomonitoring/Biomarkers

1.3.5.1 Blood. Gulumian et al. (16) reviewed the available literature concerning biomarkers reported to be associated with silicosis or coal workers' pneumoconiosis (CWP). A subset of biomarkers was considered to warrant further investigation and validation. However, none were considered sufficiently well verified for clinical use in monitoring the initiation and progression of CWP.

1.3.5.2 Urine. None.

1.3.5.3 Other. The principal biomarker of exposure to coal dust is the chest radiograph (or X-ray). The chest radiograph is a somewhat insensitive indicator of dust deposition and the biological changes associated with deposition and fibrosis. It also has limited usefulness in preventing CWP. By the time the disease is apparent, it is likely to be irreversible and may progress in the absence of further dust exposure (17, 18). Nevertheless, the chest radiograph offers a convenient, inexpensive, noninvasive, and low-risk procedure for assessing dust-related disease. Using the International Labour Office (ILO) Guidelines, radiographs can be categorized for the presence and severity of abnormalities associated with pneumoconiosis (19). Workers who have early signs of disease can be informed and steps can be taken to reduce their dust exposure. Some countries have established procedures that give workers the opportunity to have periodic radiographs and permit them to work in lower-dust exposure jobs if pneumoconiosis is present, for example, in the United States the NIOSH's Coal Workers' Health Surveillance Program (CWHSP) provides the opportunity for miners to obtain respiratory health screening to demonstrate eligibility for the Part 90 job transfer program operated by MSHA (20).

For clinical diagnosis and management, high-resolution computed tomography (HRCT) has proven to provide higher sensitivity and specificity than radiographs, particularly in the early phases of disease (21). An international classification system of HRCT for occupational and environmental respiratory diseases analogous to the ILO classification system of chest radiographs has been proposed and used in some studies of pneumoconiosis in coal miners (21). Large-scale HRCT screening studies in coal miners analogous to radiographic surveillance conducted in the United States and elsewhere have yet to be conducted. The primary limitations thus far have been access, radiation dose, and cost. However, access has become more available in recent years and both dose and cost continue to decline over time; consideration of conducting such studies in the future is warranted.

Ventilatory function is another potential biomarker of the effect of inhaling coal dust. Following MSHA's 2014 final rule on respirable coal mine dust exposure (discussed in depth in Section 1.5), NIOSH expanded its CWHSP to include spirometry testing and respiratory health questionnaires. These services are in addition to chest radiographs and occupational history questionnaires. Spirometric screening has been recommended for coal miners to detect unusual decrements in pulmonary function and provide timely intervention (22).

Few other biomarkers pertinent to coal mine dust exposure and disease have been evaluated to any extent. Tumor necrosis factor (TNF) and serum type III procollagen peptide (PIIIP) have been proposed as possible biological markers for early diagnosis of CWP (16, 23), although clinical validation remains to be done.

1.4 Toxic Effects

Inhalation of coal mine dust has been associated with the development of CWP, mixed dust pneumoconiosis, emphysema, chronic bronchitis, and obstructive lung disease (18, 24, 25). CWP is categorized by degree of severity. In the early stages of CWP, coal dust macules appear predominantly in the upper regions of the lung. With continuing exposure, pulmonary lesions enlarge to form coal micronodules and later coal nodules and can progress to the most severe form, progressive massive fibrosis (PMF). At this stage, lesions are larger (2 or 1 cm in diameter as defined by pathology or radiology), more numerous, and are found predominantly in the upper and posterior regions of the lung. The Federal Coal Mine Health and Safety Act of 1969 legislatively defined "black lung disease" more broadly to include CWP, as well as chronic obstructive pulmonary disease (COPD) in coal miners.

The pulmonary response to inhalation of coal mine dust varies with exposure duration and dose (26, 27). After initial exposures, inhaled coal dust is engulfed predominantly by alveolar macrophages. As exposure duration increases, dust can be found around the walls of bronchi and respiratory bronchioles and within pulmonary lymphatics and blood vessels. The first histologic manifestation of CWP is the coal macule (Figure 5). Macules are localized accumulations of coal dust in the walls of respiratory bronchioles, most commonly at the bifurcations of these airways in the upper zones of the lung. Macules are not palpable, range from 1 to 5 mm in diameter, and are associated with varying degrees of collagen deposition that indicate the initiation of fibrosis. As dust-laden macrophages and collagen accumulate, micronodules (<7 mm diameter) and nodules (7 mm–2 cm) can form. At this stage, the coal dust lesions are firm upon palpation. Coal nodules appear irregular and pigmented histologically (Figure 6). As disease progresses, nodules can coalesce into PMF lesions which are zones of fibrosis generally larger than 2 cm in diameter. These fibrotic lesions are commonly concentrated in the upper and posterior regions of the lung; however, radiographic studies have demonstrated that a substantial proportion of radiographs from modern U.S. coal miners with evidence of simple CWP and PMF did not demonstrate this "classic" pattern (28, 29). Upon gross examination, many PMF lesions appear as hard, black masses with a sharp demarcation from surrounding tissue. Histologically, these lesions appear as amorphous zones of collagenization or aggregates of multiple nodules. The lesions contain large amounts of black pigment, and necrosis is associated with central cavitation (Figure 7).

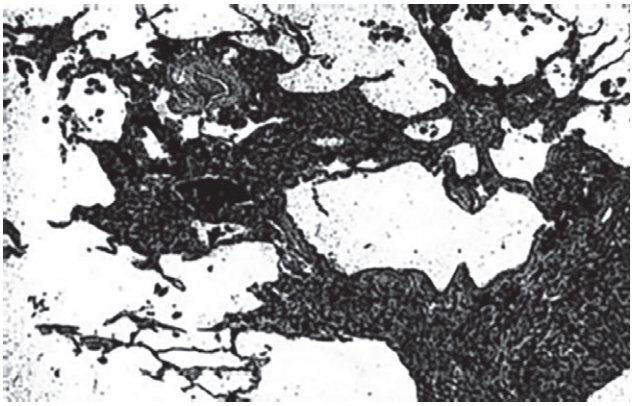

Figure 5. Coal macules in the walls of respiratory bronchioles. Note the accumulation of darkly pigmented coal dust.

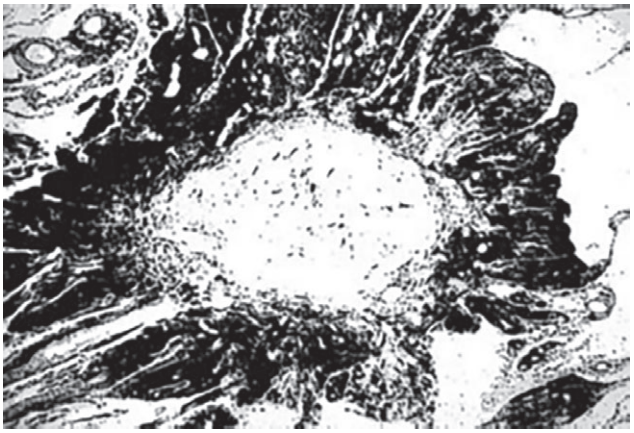

Figure 6. A coal nodule. Note the dark pigmentation and irregular border.

CWP is often associated with emphysema (27), that is, destruction of alveolar septae resulting in enlarged airspaces and a decreased surface-to-volume ratio (Figure 8). As emphysema advances, decreased gas exchange and obstructive lung disease may become apparent. Silicosis is also commonly seen in conjunction with CWP. Studies found that 13–23% of American coal miners autopsied primarily in the 1970s exhibited silicotic lesions (30, 31). Silicosis is most common in coal miners whose jobs are associated with relatively high silica exposures, such as drillers, continuous miner operators, roof bolters, and railway operators (30, 32). Histologically, silicotic nodules can be distinguished from coal nodules by the appearance of concentric spirals of collagen and birefringent silica particles (Figure 9).

Exposure to coal mine dust is known to cause bronchitis and obstructive lung disease in addition to CWP. These diseases may exist in conjunction with CWP or may occur in miners who have no radiographic evidence of CWP (33).

Figure 7. A Gough section of a coal miner's lung with PMF. Note the large, dark, fibrotic area set against a background of coal nodules.

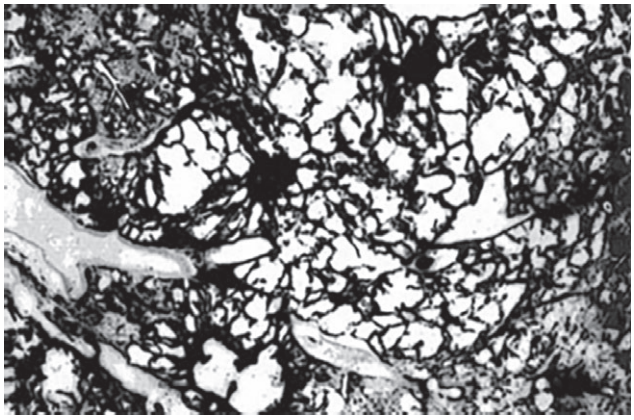

Figure 8. Histological appearance of emphysema in a coal miner's lung. Note the destruction of alveolar septae, resulting in enlarged airspaces.

Unlike CWP, which has been intensively studied using laboratory methods, research into bronchitis and obstructive lung disease has been restricted almost exclusively to epidemiological studies. For this reason, Section 1.4.1 and its subsections on the experimental evidence concentrate on CWP; the main discussion of lung diseases other than pneumoconiosis is found in Section 1.4.2.

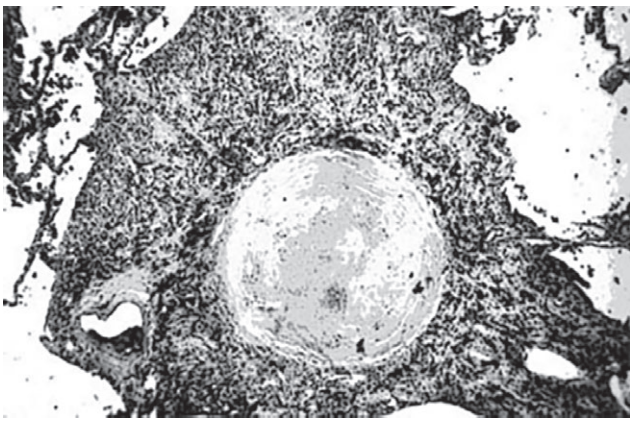

Figure 9. A silicotic nodule in a lung section of a coal miner. Note the spiral arrangement of collagen in the lesion.

1.4.1 Experimental Studies

Investigations concerning the initiation and progression of CWP have concentrated on elucidating two major mechanisms of toxicity (34). The first is the role of direct toxicity of coal dust and/or silica or trace metal contaminants of coal mine dust to lung cells. The hypothesis is that cell damage at dust deposition sites would lead to localized lesions and eventually compromised function. The second mechanism is the role of oxidant species, inflammatory cytokines, and proliferative mediators in CWP. The hypothesis is that coal dust stimulates pulmonary phagocytes to generate reactive species and inflammatory or fibrogenic cytokines that lead to disease initiation and progression. Experimental studies involve *in vitro* investigations, as well as *in vivo* studies with animal models or coal miners.

1.4.1.1 Acute Toxicity. *In vitro* exposure of red blood cells or macrophages to coal dust results in direct membrane damage, measured as the leakage of hemoglobin or lactate dehydrogenase (LDH), respectively (35–37). In contrast, coal dust does not stimulate lysosomal enzyme release from alveolar macrophages (37, 38). In in *vitro* studies, coal dust exhibits far less direct cytotoxicity than silica and ranges from 7% to 33% as toxic as crystalline silica (37). The toxicity of coal dust is significantly enhanced by adding 10% quartz to the dust (36). Grinding or fracturing coal particles increases their lytic activity (39). This direct cytotoxicity is proportional to the generation of coal-based free radicals on the fracture planes. Ground anthracite coal generates more surface radicals and is more cytotoxic than fractured bituminous coal.

Trace metals may affect the cytotoxicity of coal dust. Water leachates of coal mine dust inhibit the growth of mammalian cells in culture. Coal dust from Pennsylvania mines is more toxic than coal dust from Utah mines (40). The investigators relate this difference in potency to the higher nickel content of the Pennsylvania coal mine dust. In addition, coal dust from mines in Western Pennsylvania generates more reactive oxygen radicals and causes greater lipid peroxidation than coal dust from Utah mines (41). This difference was attributed to the greater amount of available surface iron in the Pennsylvania coal dust.

The association between iron content and bioactivity of coal dust is specifically related to bioavailable iron. Coal dust from Western Pennsylvania has been reported to release threefold more iron than coal dust from Utah mines when suspended in 10 mM phosphate solution (pH 4.5) (42). This enhanced bioavailability of iron was directly correlated with the ability of coal dust to induce lipid peroxidation in lung epithelial cells *in vitro* (42). Evaluation of the historic prevalence of CWP in seven coal mine regions in the United States indicated a strong correlation ($r = 0.94$) between prevalence of CWP and bioavailable iron in these coal samples (43). A more recent Australian study examined associations between pyrite content of coal, release of bioavailable iron, and cellular responses involved in CWP pathogenesis (44). Contrary to previous suggestions that pyrite content in coal could be a predictor of CWP risk, there was no association between pyrite content and the magnitude of pathogenic cellular response.

The effect of coal dust exposure on the production of inflammatory products has been investigated *in vitro* (Table 2). *In vitro* exposure of alveolar macrophages to coal dust increases the release of the platelet-activating factor (PAF) (45). PAF acts as a direct stimulant of oxidant generation in polymorphonuclear leukocytes and as a priming factor for particle-stimulated oxidant production in alveolar macrophages. Therefore, PAF may play a role in the inflammatory response of the lung to the inhalation of coal dust. In contrast, coal dust decreases the production of the proinflammatory factor, interferon, by virally stimulated mammalian cells in culture (46). Anthracite coal exhibits a greater effect than bituminous coal. Exposure of alveolar macrophages to freshly fractured coal dust enhances the production of cyclooxygenase metabolites of arachidonic acid such as prostaglandin E_2 (PGE_2) and thromboxane A_2 (TXA_2), whereas aged coal dust has no effect (47). PGE_2, it is thought, inhibits fibroblast proliferation (51) and inhibits production of the proinflammatory cytokine interleukin-1 (IL-1) (52). Indeed, coal dust fails to stimulate IL-1 production in alveolar macrophages (53). Data indicate that TXA_2 augments the production of tumor necrosis factor-α (TNF-α) (54), which exhibits strong inflammatory and fibrogenic properties (55, 56). Indeed, coal dust enhances the production of TNF-α by alveolar macrophages (48, 49). Likewise, coal dust stimulates the production of interleukin-6 (IL-6) by alveolar macrophages (48, 49). However, IL-6, it is thought, inhibits fibroblast proliferation (57). The net result of the differential production of pro- and antifibrotic factors in response to *in vitro* exposure is stimulation of fibroblast

Table 2. Effect of *In Vitro* Exposure to Coal Dust on the Production of Inflammatory Mediators

Mediator	Cell Type	Response to Coal Dust	Mediator Action	References
Platelet-activating factor	Alveolar macrophages	Increases production	Directly stimulates neutrophils; primes alveolar macrophages	(45)
Interferon	Mammalian kidney cells	Decreases viral-stimulated production	Activates inflammatory cells	(46)
Interleukin-1	Alveolar macrophages	No stimulation	Proinflammatory	(45)
Prostaglandin E_2	Alveolar macrophages	Increases, but only with fresh dust	Inhibits fibroblast proliferation	(47)
Thromboxane A_2	Alveolar macrophages	Increases, but only with fresh dust	Proinflammatory	(47)
Tumor necrosis factor	Alveolar macrophages	Increases production	Proinflammatory; profibrotic	(48, 49)
Interleukin-6	Alveolar macrophages	Increases production	Inhibits fibroblast proliferation	(48, 49)
Fibronectin	Alveolar type II epithelial cells	Increases production	Profibrotic	(50)

proliferation (58). Exposure of alveolar type II epithelial cells to coal dust enhances the production of fibronectin and extracellular matrix (50). TNF-α and TGF-β 1 augment this stimulatory effect of coal dust. The net result is stimulation of the fibrogenic response of the lung to coal dust.

1.4.1.2 Chronic and Subchronic Toxicity. *In vivo* animal models do not demonstrate high direct toxicity following coal dust exposure. Indeed, inhalation of coal mine dust (2 mg/m^3, 7 h/day, 5 days/week for 2 years) fails to increase signs of lung injury in bronchoalveolar lavage (BAL) samples, that is, acellular LDH or proteins (50). However, lipoproteinosis does occur following inhalation of coal mine dust (59). This lipoidotic response is greater for anthracite than bituminous coal. In general, these responses are much smaller than those measured after inhalation of silica (60–63). In a comparative pathology study, both rats and humans showed a graded fibrotic response, that is, higher exposures to coal dust caused more fibrosis (64).

Animals demonstrated little evidence of a fibrotic response after coal dust exposure in some studies (64–66). However, the addition of quartz to the coal dust results in a significant level of pulmonary fibrosis (67–69). Earlier epidemiological studies with coal miners have shown that it is coal dust rather than silica dust that most strongly correlates with CWP and PMF development (70, 71). It has been proposed that this may be due to a clay surface coating on the quartz particles that depresses their toxicity (72, 73). Clay-coated silica particles are more prominent in bituminous mines (higher silica content) than in anthracite mines (lower silica content). Other studies have shown that quartz dust can contribute to lung disease in coal miners. Radiographic category of CWP (small opacity profusion) was more strongly related to the quartz fraction of dust than to the nonquartz fraction in U.K. coal miners (74). In U.S. coal miners, cumulative exposure to quartz dust was a significant predictor of pulmonary inflammation and radiographic category of simple CWP (75).

Since the year 2000, severe cases of CWP have been increasingly reported, including PMF (76–79) and rapidly progressive pneumoconiosis (RPP) (76). In the central Appalachian states of Kentucky, Virginia, and West Virginia, the incidence of PMF is now greater than that observed in 1970 (80). While reported mass concentrations of respirable coal mine dust have been declining for decades in the United States (81, 82), it has been suggested that changes in dust including mineralogic composition and particle size could be associated with increasing disease rates. In a recent study (83), samples from mines in central Appalachia had higher percentages of silica and alumino-silicate particles compared to mines in northern Appalachia.

A follow-up analysis by Sarver et al. expanded data collection to include samples from 25 coal mines spanning the Appalachian and non-Appalachian mining regions (84). They identified meaningful differences in particle size and mineral composition within and between mines and depending on proximity to mining activities, mining method(s), and geographic region. Near production areas, the mass of dust from adjacent rock strata was inordinately high relative to the heights of coal and rock strata being mined. Particle size analysis found that a high proportion of respirable coal mine dust particles, including silica and silicates, were in the submicron (100–1000 nm) size range, but that these particles accounted for <10% of total mass; regulatory sampling in the United States is limited to mass-based measurements. Schatzel et al. studied sources of respirable dust in Appalachian coal mines and found that in continuous miner operations, the strata above the mined coal seam (roof rock) was the primary source or respirable mineral dust generated during mining, and that local dust controls in these operations could be preferentially configured to address overhead sources of dust (85, 86).

A NIOSH study reported increasing proportion of *r*-type opacities in surveillance chest radiographs of central Appalachian coal miners, suggestive of greater exposure to

Table 3. Responses of Animal Models to *In Vivo* Exposure to Coal Dust

Parameter	Consequence	References
Increased number of alveolar macrophages	Inflammation	(50, 89, 90)
Recruitment of neutrophils into the airspaces	Inflammation	(91)
Elevated blood and interstitial monocytes	Inflammation	(92)
Increased production of LTB_4 and TXA_2	Inflammation	(93)
Enhanced surface activity of alveolar macrophages	Macrophage activation	(50)
Increased chemiluminescence by alveolar macrophages	Oxidant generation	(53, 94)
Increased nitric oxide production by alveolar macrophages	Oxidant generation	(94)

silica and silicates (87). A follow-up study in 2019 reported that the prevalence of *r*-type opacities in Appalachian coal miners continues to increase (88). Finally, analysis of lung specimens from 13 deceased coal miners with RPP revealed large amounts of birefringent mineral dust particles consistent with silica and silicates while carbonaceous coal dust was less prominent (32).

In contrast to the relatively low fibrotic response in animals exposed to coal dust, more pronounced inflammatory reactions to coal dust have been demonstrated (Table 3). This inflammation includes increases in the number of alveolar macrophages (50, 89, 90) and neutrophils (91) obtained by BAL, as well as elevation of blood and interstitial monocytes (92). Exposure of rats to coal dust increases the production of leukotriene B_4 (LTB_4) and TXA_2 by alveolar macrophages (93). These arachidonic acid metabolites may be responsible, in part, for the cell recruitment noted before. *In vivo* exposure of rats to coal dust results in activation of alveolar macrophages. This activation is evidenced by increased cell spreading and ruffling and increased generation of reactive oxygen species and nitric oxide (50, 94). This production of oxidants by alveolar phagocytes may lead to lung damage associated with coal dust exposure.

In a chronic inhalation study in Sprague–Dawley rats exposed to 200 mg/m^3 of coal dust with or without 10% quartz for up to 24 months (5 h/day, 5 day/week, every 2 week), elevated lung tumor rates were reported (66). The tumor response was 11% or 44%, respectively, in rats exposed to coal dust or the coal–quartz mixture, compared to 0% in the unexposed controls. The tumor types were reported as adenocarcinomas or epidermoid tumors.

Increasing fraction of quartz (from 6.8% to 24.5%) in respirable coal mine dust was associated with increasing lung damage in rats exposed by inhalation to 20 mg/m^3 dust for 12–18 months (95). Granulomatous lung responses were observed including profusion of pigmented lung nodules and hypertrophy of the lymph nodes, which were considered consistent with the pneumoconiotic responses observed in coal miners including those exposed to unusually high levels of quartz.

Numerous studies have investigated the production of inflammatory and fibrotic mediators in coal miners. BAL parameters have been evaluated in asymptomatic underground coal miners with normal pulmonary function and chest radiographs, as well as in coal miners diagnosed with simple CWP or PMF. Evidence indicates that the profile of mediator production changes with the progression of disease (Table 4). In asymptomatic miners, no change in the number of alveolar macrophages harvested by BAL is reported (96). In contrast, alveolar macrophage numbers increase in earlier stages of CWP and are elevated further as the disease progresses to PMF (97, 98). This macrophage recruitment is associated with an increase in the production of monocyte chemoattractant peptide-1 (MIP-1) and ICAM-1 in miners who have CWP (111, 113). Coincident with the recruitment of alveolar macrophages is the activation of alveolar phagocytes to produce reactive oxygen species and inflammatory mediators. Activation is noted as an increase in surface ruffling of alveolar phagocytes harvested from coal miners compared with those from unexposed subjects (96, 97).

The release of reactive oxidants is related to disease severity. There is no change in asymptomatic coal miners but a progressive increase of reactive oxygen species production in earlier stages of CWP and PMF (96, 99–101). Additionally, an increase in nitric oxide production by alveolar macrophages harvested from coal miners has been reported, which was directly related to disease severity (102). The inflammatory arachidonic acid metabolite TXA_2 is elevated in asymptomatic miners, whereas the level of this proinflammatory mediator has not yet been reported in miners who have CWP (106). Oxidant generation by coal dust-exposed alveolar phagocytes results in shifts in lung antioxidant levels.

Elevated BAL fluid levels of superoxide dismutase, catalase, and glutathione peroxidase were reported in coal miners with category 2/2 CWP (103). However, a fall in red blood cell glutathione was noted as the disease progresses (104, 105), and oxidative deoxyribonucleic acid (DNA) damage in peripheral lymphocytes is reported (116). Proinflammatory mediators such as TNF-α and interleukin-1 β change with disease progression. In asymptomatic coal miners, acellular lavage levels of TNF-α and, to a lesser extent, IL-1 decrease, indicating initial regulation of inflammation (117). However, production of TNF-α and IL-1 by alveolar macrophages and blood monocytes and levels in BAL fluid increase in CWP (106–110). TNF-α production by alveolar macrophages is highest in miners with PMF, whereas blood monocyte levels

Table 4. Bronchoalveolar Lavage (BAL) Changes in Underground Coal Miners Disease State

Parameter	Asymptomatic	Simple CMP	PMF	References
Number of phagocytes in BAL	○[a]	↑[b]	↑↑[c]	(96–98)
Oxidant (ROS and NO) production by alveolar phagocytes	○	↑	↑↑	(96, 99–102)
Phagocyte surface ruffling	↑	↑	↑	(96, 97)
Antioxidant levels		↓Glutathione in red blood cells[d] ↑ Catalase, glutathione peroxidase, and superoxide anion in BAL (category 2/2)	↓ Glutathione in red blood cells	(103–105)
Arachidonic acid metabolites from alveolar phagocytes	↑ TXA_2	–	–	(106)
TNF	↓ Acellular BAL fluid levels	↑ Production by alveolar macrophages ↑ Levels in BAL (category 2/2) ↑↑ Production by blood monocytes	↑↑ Production by alveolar macrophages ↑ Production by blood monocyte!	(103, 106–110)
IL-1	Slight ↓ in acellular lavage levels	↑ Production by alveolar macrophages ↑ Levels in BAL (category 2/2)	↑ Production by alveolar macrophages	(103, 106–110)
Fibronectin	○ In acellular BAL fluid	↑ Production by alveolar macrophages ↑ Levels in BAL (category 2/2)	↑ Production by alveolar macrophages	(96, 103, 111)
Macrophage-derived growth factor	–[e]	↑	↑	(103)
Type I insulin-like growth factor	–	↑ Production by alveolar macrophages ↑ Acellular BAL level	↑↑ Production by alveolar macrophages ↑↑ Acellular BAL level	(112)
PDGF	–	↑ Production by alveolar macrophages ↑ Acellular BAL level	↑↑ Production by alveolar macrophages ↑↑ Acellular BAL level	(112)
TGF-βl		↑ Acellular BAL level ↑ Levels in BAL (category 2/2)	↓Acellular lavage level	(103, 112)
IL-6	↑ Acellular BAL level	↑ Production by alveolar macrophages	↑↑ Production by alveolar macrophages	(103, 109)
MIP-1 production by alveolar macrophage	–	↑ Levels in BAL (category 2/2) ↑	↑↑	(113)
ICAM-1 production by alveolar macrophages	–	↑	↑↑	(111)
Surfactant protein A	↑↑ In BAL	↑ In BAL	–	(114)
Metalloproteinase-3	↑ In serum	↑↑ In serum	–	(115)

[a] ○ Indicates no change from control.
[b] ↑ Indicates an increase from control.
[c] ↑↑ Indicates a greater increase.
[d] ↓ Indicates a decrease from control.
[e] Indicates no data are reported.

are highest in simple CWP. In coal miners, airflow limitation is correlated with levels of LPS-stimulated TNF-α release from blood monocytes (117). Taken together, these data support the hypothesis that initially disease is prevented by the upregulation of protective antioxidants and the downregulation of inflammatory cytokines. Induction and progression of the disease process are associated with an increase in oxidant generation, loss of antioxidant protection, and progressive pulmonary inflammation and damage.

The pulmonary fibrotic process is regulated by a complex mix of mediators that either augment fibroblast proliferation or control it. Evidence indicates that production of these mediators changes with the progression of CWP (Table 4). The level of the profibrotic mediator fibronectin in acellular lavage fluid is unchanged in asymptomatic coal miners (107), but macrophage production of fibronectin and BAL levels are elevated as CWP progresses (99, 103). Similarly, the macrophage-derived growth factor enhances fibroblast proliferation, and its production is elevated in alveolar macrophages harvested from CWP patients (99). Platelet-derived growth factor (PDGF) and type I insulin-like growth factor are profibrotic cytokines (56, 113). The production of each of these mediators by alveolar macrophage is increased in simple CWP and further elevated as the disease progresses to PMF (112). Transforming growth factor (TGF-β) tends to depress fibroblast proliferation (112). BAL fluid levels of TGF-β are elevated in coal miners with simple CWP but are depressed in PMF (112). In contrast, interleukin-6, that is, an antifibrotic cytokine (57), is elevated in the BAL fluid of asymptomatic coal miners, is increased in BAL from miners with category 2/2 CWP, and secretion of IL-6 is most marked in alveolar macrophages from PMF patients (103, 109). Surfactant protein A levels in BAL are elevated in coal miners with the degree of elevation decreasing as disease progresses (114). Metalloproteinase-3 is associated with lung remodeling. Serum levels are elevated in coal miners with levels rising as disease progresses (115). Therefore, as CWP is initiated and progresses, the balance between pro- and antifibrotic cytokines shifts to a state that favors fibroblast proliferation.

1.4.1.3 Pharmacokinetics, Metabolism, and Mechanisms. A few experimental studies of animals exposed to coal dust have been performed that provide information on the disposition of particles in the lungs. In rats exposed by chronic inhalation to 200 mg/m^3 of coal dust (with or without added quartz), the average amount of dust retained in the rat lungs at 24 months was 96 mg (66), or approximately 53 mg dust per g of control (unexposed) rat lung tissue. In rats exposed by inhalation to 20 mg/m^3 coal dust (containing 5–20% quartz) for 12 or 18 months, the average retained lung dust burdens were 16 mg at 12 months and 24 mg at 18 months, or approximately 11 or 17 mg/g lung tissue in control rats (95). Following rats for up to 14 months after the end of exposure indicated little or no clearance of dust from the lungs (95).

Rodent studies have shown that the chronic inhalation of various types of insoluble, respirable particles can lead to the impairment or overloading of alveolar macrophage-mediated clearance (118–122), which can continue after exposure has ended (123).

Two hypotheses have been proposed to explain this reduced clearance. The sequestration hypothesis (124, 125) predicts that some fraction of dust is retained in the lungs even at low exposures. This sequestered dust (e.g., within the interstitium) is not available for clearance by alveolar macrophages. Studies by Vincent et al. (126) provide evidence for the sequestration hypothesis. The overload hypothesis is based on evidence of the progressive decline or impairment of pulmonary clearance of particles (119). Morrow (127) hypothesized that alveolar macrophages have a volumetric limit for particle engulfment, above which macrophage mobility begins to decline progressively until both mobility and clearance essentially cease. In several studies of rats exposed to various poorly soluble particles, retained lung dust burdens from approximately 1–10 mg dust per g of lung tissue were associated with reduced clearance, and nearly complete cessation of clearance occurred at the higher lung burdens (121). In rats with overloading doses, there is increased penetration of particles through the epithelium into the interstitium, as well as increased transfer of particles to the lymph nodes (128). Pathological responses associated with overloading lung burdens in rats include persistent inflammation, fibrosis, and lung tumors (122, 129).

The exposure–dose–response relationships for inhaled particles have been investigated through the development of biologically based mathematical models to describe the kinetics of particle retention and clearance in the lungs of rats (130–132), including a model showing a relationship between the particle surface area dose and the overloading of lung clearance (133). Coal miners have historically received lung dust burdens equivalent to those that cause overloading in animal studies (134–143). Studies involving lung dosimetry model development and evaluation based on data from U.S. and U.K. coal miners have shown differences in the particle clearance kinetics in rodents and humans (144–146) (Section 1.4.2.3.3).

Species differences in the particle retention patterns in the lungs were observed (147) in a study of rats and monkeys exposed for 2 years to coal dust (2 mg/m^3), diesel exhaust particulate (2 mg/m^3), or coal dust and diesel exhaust particulate (1 mg/m^3 each) (148). In rats, a greater proportion of either coal or diesel exhaust particulate was retained in the alveolar spaces within macrophages than in the alveolar septal interstitium. In contrast, a greater proportion of dust was retained in macrophages within the interstitium in monkeys. In humans, a greater proportion

of the retained particulate matter was also found within the lung interstitium, especially among miners with higher lung burdens (149). This finding is consistent with the finding of minimal or no clearance of particles from the lungs of retired miners (150, 151). Nikula et al. (147, 149) suggest that the differences in particle retention patterns could relate to differences in rodent and primate lung responses to inhaled particles.

1.4.1.4 Reproductive and Developmental. To date, no reports are available to suggest that coal dust exposure produces adverse effects on reproductive function or prenatal or postnatal development of offspring of those exposed to coal dust.

1.4.1.5 Carcinogenesis. The epidemiologic evidence for a relationship between coal mine dust exposure and lung cancer is presented in Section 1.4.2.3.5. Histological evaluation of lung tumors in coal miners reveals that these tumors vary little in cell type or pathological features from those associated with cigarette smoking (152).

Elevated stomach cancer rates have been reported in studies of U.S. underground coal miners compared to general population rates (see Section 1.4.2.3.5). A mechanistic explanation for coal dust-induced stomach cancer suggests that swallowed coal dust mixes with nitrates in food and under the acidic conditions in the stomach, nitrosation of organic material associated with this coal dust occurs, resulting in the production of carcinogenic products (153). Evidence indicates that nitrosation of coal dust extracts becomes mutagenic and can cause neoplastic transformations in mammalian cells (154).

1.4.1.6 Genetic and Related Cellular Effects Studies. As mentioned in the preceding section, the reaction of nitrates with coal dust under acidic conditions resulted in nitrosated coal compounds that can cause genetic alterations. In vitro studies have shown evidence of cytotoxicity and DNA damage following exposure to coal dust and coal fly ash, while epidemiological studies have identified evidence of increased genotoxicity among coal miners when compared to a reference population (155, 156). Several studies have shown associations between worker susceptibility to CWP and polymorphisms in genes coding for cellular mediators involved in inflammatory, fibrotic, DNA-repair, and telomeric processes (16, 157–162).

1.4.1.7 Other: Neurological, Pulmonary, and Skin Sensitization. The pulmonary effects of exposure to coal dust in experimental studies were discussed earlier (Sections 1.4.1.1 and 1.4.1.2). Associations between exposure to coal dust and adverse neurological or dermal effects are lacking at present.

1.4.2 Human Experience

1.4.2.1 General Information. Meiklejohn provides extensive documentation of the long human experience of working in coal mining (163–165). Various reasons for these chronic outcomes were postulated initially, including impure air, soot from candles and lamps, emissions from gunpowder explosions, coal dust, and stone dust. Later, attention shifted to silica exposure as the putative causative factor (resulting in anthracosilicosis). However, it was not until it was shown that CWP develops in workers exposed to cleaned coal (with little silica component) that exposure to coal dust was considered other than benign (166). Subsequently, results from epidemiological investigations confirmed that exposure to both anthracite and bituminous coal mine dust was the predominant instigator of respiratory disease, rather than silica (167–169). However, miners exposed to higher concentrations of respirable crystalline silica (quartz) have an increased risk of developing silicosis (32, 74, 87, 88, 170), even if the duration of exposure is relatively short (166). More recently, Chris Hamby reported on the human experience of coal miners in central Appalachia including the day-to-day experience of living with CWP and the challenges associated with diagnosis and navigating the disability compensation system (171).

1.4.2.2 Clinical Cases

1.4.2.2.1 Acute Toxicity. Exposure to coal mine dust does not cause acute toxicity, except where miners are exposed to high levels of silicaceous rock dust (32). In these cases, it is the intense exposure to silica dust, rather than coal dust that causes acute silicosis.

1.4.2.2.2 Chronic and Subchronic Toxicity. The first detailed description of "black lungs" attributable to coal mining employment was provided by Gregory (172), who noted autopsy findings consistent with PMF in a coal miner with about 10 years of work experience. This report was followed closely by that of Thomson (173), who in 1836 described numerous cases among Scottish miners. These early reports noted the expectoration of black sputum and the deposition of black matter in the lungs, as well as disease and debility among the miners.

Coal mine dust deposition in the lungs brings about changes that may be symptomless in some miners. On chest radiograph, the dust deposits are seen as opacities that increase in profusion with increased exposure. In some miners, early stages of CWP can develop into PMF. PMF is often associated with significant impairment and can lead to premature death. Signs and symptoms associated with PMF include shortness of breath, cough, increased sputum production, altered blood gas levels, and emphysema (174–176). Miners with CWP, particularly PMF, are more susceptible to tuberculosis infections. There is currently no effective treatment for CWP (25). Various therapies have been tried (177),

and recently lung transplantation has become increasingly common (178).

Coal miners are subject to lung diseases that can occur in conjunction with or in the absence of CWP. Pneumoconiosis is primarily associated with a restrictive pattern of lung impairment, whereas COPD is an obstructive lung disease. Inhalation of coal dust leads to enlargement of the mucus glands in the airways, and the result is increased cough and phlegm production. Airflow obstruction can occur, leading to shortness of breath, and disability in some miners (179–181). Emphysema is independently related to coal mine dust exposure after accounting for cigarette smoking and other factors (182, 183). For many years, there was much controversy over the cause of nonpneumoconiotic lung diseases in coal miners. Some authorities stated that they were the result of smoking or were of no medical consequence. However, scientific studies have since shown clear exposure–response relationships for these diseases with increasing cumulative coal mine dust exposure and demonstrated that the lung function impairment can be clinically significant, even in the absence of chest radiographic evidence of CWP (33, 179, 181, 184–187). In 1998, after a comprehensive review of the evidence, the British courts ruled in favor of miners who stated that their (nonpneumoconiotic) lung diseases were attributable to coal mine dust exposure (188). In the United States, NIOSH recommends the control of coal mine dust exposures to prevent obstructive lung disease, as well as CWP (189). For disability compensation purposes in the United States, pneumoconiosis is defined as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment (190). This definition is inclusive of COPD.

1.4.2.3 Epidemiology Studies

1.4.2.3.1 Acute Toxicity. Acute toxicity from coal dust exposure has not been reported in humans. However, coal miners who are exposed to extreme levels of silica at work, such as surface coal mine drillers, are at risk of developing acute and accelerated silicosis.

1.4.2.3.2 Chronic and Subchronic Toxicity. Historically, most research has focused on working underground coal miners in bituminous and anthracite coal mines. In recent years, there has been an increased focus on health effects in surface coal miners and miners experiencing mixed coal mine dust exposures. An increasing number of studies have also focused on coal mine dust-related lung diseases in former miners.

Health effects among underground coal miners were first noted more than 300 years ago. Serious study of the health effects of coal mine dust exposure began in the early 1800s, when the term miners' black lung was coined (172). Detailed epidemiological investigation of lung disease in coal miners began around 1950 in Britain and later in the United States. The initial emphasis was on CWP, the classic interstitial lung disease associated with coal mine dust exposure. Later, the research was extended to other lung diseases common among miners, including COPD (which includes bronchitis and emphysema), mixed dust pneumoconiosis, dust-related diffuse fibrosis, and small airway disease. The term "coal mine dust lung disease" (CMDLD) has been proposed to describe this broader suite of respiratory diseases caused by exposure to coal mine dust (25).

Most of the major exposure–response findings on occupational lung disease in coal miners come from two major studies. The first, the Pneumoconiosis Field Research (PFR), was begun under the auspices of the British National Coal Board (191). Focused on periodic medical examinations at coal mines and having the benefit of resident industrial hygienists at each mine who collected daily measurements of dust levels, the PFR provided information to support the development of occupational exposure limits. Results from the PFR were incorporated into the decision-making processes for both the United States and British compliance limit for coal mine dust. In the United States, the National Study of Coal Workers' Pneumoconiosis (NSCWP) was initiated following passage of the 1969 Coal Mine Health and Safety Act (192). The NSCWP provided results consistent with the British findings (33, 193–195), in addition to longitudinal industry-wide surveillance data (196).

Earlier epidemiological studies found that dust exposure, coal rank, and age were predictors for the development of CWP (167). Overall, the main determinant of disease occurrence was exposure to mixed mine dust. Increasing coal rank was associated with increased risk of disease. The coal from the U.S. anthracite region (where mining has mostly ceased presently) is the most fibrogenic, and that from the bituminous and subbituminous regions is less so. This finding has also been observed in Britain and Germany (167, 197).

Exposure–response relationships for CWP have been reported among coal miners in Britain and the United States (168, 169, 193, 195, 198). Earlier studies showed that the risk of developing PMF increased as the radiographic category of simple CWP increased (168, 199, 200). Subsequent studies confirmed this but also showed that miners with minimal or no simple CWP are at risk of developing PMF within a 5-year period (201, 202). Cumulative exposure to respirable coal mine dust was shown to be the most important factor in the development of simple CWP and PMF (201).

Results from the earliest British studies (168, 199, 200) provided the basis for the U.S. coal dust standard of 2 mg/m^3 established by the Federal Coal Mine Health and Safety Act of 1969 (Coal Act) (P.L. 91–173), which became effective in 1972. The Coal Act led to the creation of a national respiratory health surveillance program for coal miners. Since 1970 NIOSH has administered this program, now called the CWHSP. The CWHSP offers periodic chest radiographs to

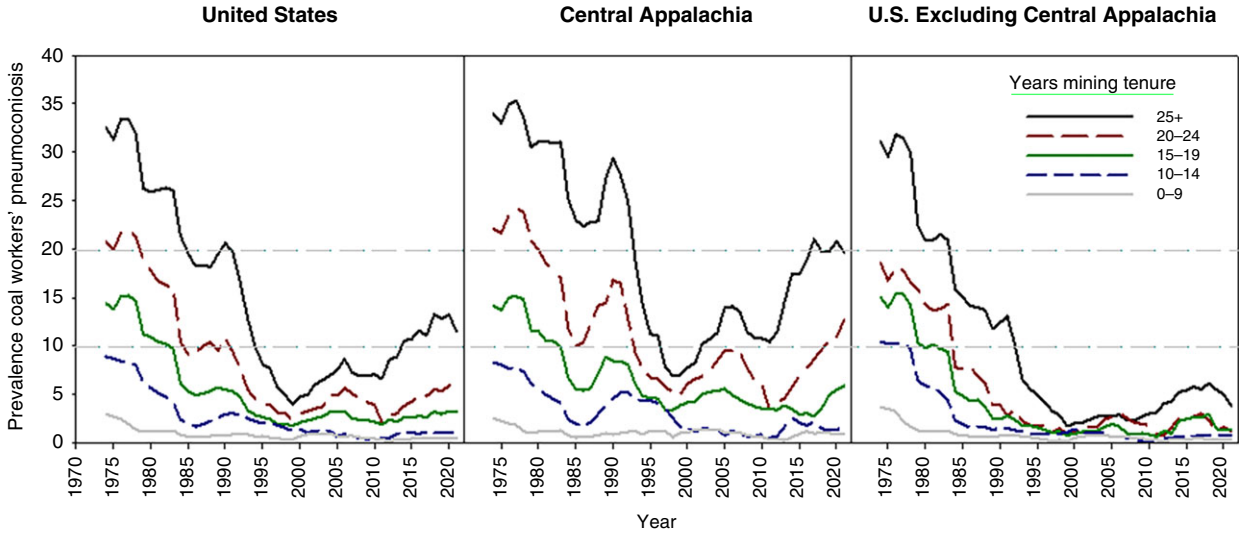

Figure 10. Prevalence of coal workers' pneumoconiosis among working underground coal miners in the United States, Central Appalachia (Kentucky, Virginia, and West Virginia), and the United States Excluding Central Appalachia: NIOSH Coal Workers' Health Surveillance Program, 1970–2021. Data are the 5-year moving average; surveillance is conducted on a 5-year national cycle.

working coal miners at regular intervals and the resulting data have enabled NIOSH to study risk factors for CWP and monitor trends for more than five decades (80). The reduction in coal mine dust levels mandated by the Coal Act resulted in a steep decline in the prevalence of CWP in underground miners beginning in the 1970s. Autopsy findings affirmed this trend: deceased miners who worked exclusively before the implementation of dust standards were found to have significantly more coal macules, nodules, silicotic nodules, and PMF when compared to those who worked only under post-Coal Act dust standards (203). However, by the late 1990s, CWP was becoming increasingly common, especially in the central Appalachian states of Kentucky, Virginia, and West Virginia (204–208). By 2020, the prevalence of CWP in underground coal miners with at least 25 years of tenure exceeded 10% nationally and 20% in central Appalachia (Figure 10) (204). The prevalence of PMF also increased sharply. After near-eradication in the 1990s, the prevalence of PMF in long-tenured coal miners in central Appalachia surpassed 5% by 2015, the highest documented level in the era of national surveillance. Data from disability compensation claims and targeted studies suggest rates of PMF among former coal miners are likely much higher (209).

Exposure to crystalline silica has been suggested as a key factor driving the resurgence of PMF. In recent decades there has been a sixfold increase in the prevalence of *r*-type small opacities among underground coal miners in central Appalachia; these opacities are associated with silicosis lung pathology (87, 88). A 2016 case series examined coal miners with RPP and identified pathologic characteristics consistent with accelerated silicosis and mixed dust pneumoconiosis (32). A number of recent studies have found that miners in occupations associated with higher silica exposures (e.g., roof bolters, continuous miner operators, surface drillers) are more likely to experience severe and RPP than miners in occupations with lower silica exposures (78, 79, 210, 211). Case reports have identified contemporary work practices associated with egregious silica exposures including situations where miners used continuous mining equipment to cut through extensive sections of rock to reach coal seams (78, 212, 213). A study characterizing dust composition in 25 coal mines across the United States found that in central Appalachian mines, rock represented 37% of total mining height, compared to 16% in mines outside this region (84).

While the largest clusters of CWP and PMF have been identified in central Appalachia, CWP also occurs outside the Appalachian region, but at lower rates. Among coal miners screened during 2005–2015, the prevalence of CWP in the eastern region (Alabama, Maryland, Ohio, Pennsylvania, and Tennessee) was 3.4%; the prevalence was 1.7% in the western region (Arizona, Colorado, Montana, New Mexico, North Dakota, Utah, and Wyoming) and 0.8% in the interior region (Illinois, Indiana, Louisiana, Oklahoma, and Texas) (204, 206).

In recent years, serial radiographs collected through CWHSP surveillance have been examined to describe CWP progression patterns in coal miners. A study of working miners found mean duration from a normal radiograph to one with evidence of PMF was approximately 20 years; nearly one-in-five of these miners progressed from a normal radiograph to PMF in <10 years (214). A study of miners participating in the Part 90 job transfer option, a program administered by MSHA intended to limit coal mine dust exposure for miners with evidence of CWP, found that nearly

one-third experienced continued radiographic progression (215). Surveillance data have also enabled researchers to examine determinants of CWP more closely to identify groups that could benefit from enhanced surveillance and exposure prevention. In addition to age and mining tenure, higher rates of CWP are associated with smaller mine size (50 or fewer employees) (205, 216), lower seam height (217), and region, which is likely a proxy for coal rank, mine size, and seam height (76).

Historically, surface coal miners were thought to be at lower risk of CWP compared to underground miners (218–220). Surface miners make up a growing proportion of the contemporary workforce and currently comprise half of the U.S. coal mining population. Research from the 1980s identified surface coal mine drillers as being at higher risk of silicosis (221, 222). Because surface miners were not previously included in the CWHSP, NIOSH conducted focused outreach to surface coal miners using a mobile examination unit during 2010 and 2011. More than 2000 surface miners were screened and 2% had radiographic evidence of CWP and a threefold higher prevalence was observed in central Appalachia compared to the rest of the country (210). Follow-up with nine surface miners, who showed evidence of PMF and reported no underground mining tenure, revealed most spend a majority of their mining careers as drillers or driller/blasters (211). In 2014, MSHA promulgated a respirable dust rule which entitled surface coal miners to participate in the CWHSP for the first time (223). A subsequent study found that among 6790 working surface coal miners examined by the CWHSP during 2014–2019, 1.6% had radiographic evidence of CWP, and 12 had PMF (224).

The CWHSP's legislative mandate is to provide respiratory health screenings to working coal miners. In recent years NIOSH has conducted targeted outreach to former coal miners using a mobile health unit. Compared to working coal miners, former miners had higher prevalence of CWP and lung function impairment (225). To date, the largest U.S. clusters of PMF have been identified in clinics serving mostly former miners in central Appalachia. A total of 60 cases of PMF were identified over a 20-month period during 2015–2016 at a community radiology practice in Pike County, Kentucky (226). Within a network of clinics in rural southwestern Virginia, 416 cases of PMF were identified over a recent 4-year period; 90% of these were in former miners, and many had radiographic findings suggestive of exceptionally severe and RPP (79). Participation in the CWHSP is voluntary and several potential barriers to participation have been suggested. Many cases of CWP are not detected during miners' working careers and instead are identified postemployment when they apply for state or federal black lung compensation benefits. Researchers examined data from the Federal Black Lung Benefits Program and identified a total of 4679 cases of PMF among miners applying for benefits during 1970–2016 with more than half identified since 1996. Most of these miners last worked in Kentucky, Pennsylvania, Virginia, or West Virginia (209). Former miners can also develop CWP after their occupational dust exposures have ceased. A study of >3300 former coal miners applying for federal benefits found that over 14 years, >3% progressed from no disease or simple CWP to PMF (227). Factors associated with likelihood of progression in this population included a higher profusion of small opacities at baseline and younger age.

An increasing number of coal miners with severe CWP have undergone lung transplantation. At least 79 lung transplants for CWP were performed in the United States through 2018, and additional cases were likely undetected because of inconsistencies in the way cases are recorded in the transplant registry. Most lung transplant recipients were from central Appalachia and median posttransplant survival was 6.6 years (228).

Officially reported mortality for CWP in the United States has declined since the 1980s, even in states that have experienced sharp increases in the prevalence of CWP. Several potential factors have been suggested to explain the difference in these statistics. Miners often live for decades following a CWP diagnosis, so the relatively recent resurgence of cases might not yet be reflected in mortality statistics. As a result of mechanization and reductions in demand for thermal coal used for electricity generation, the coal mining workforce has contracted since the 1980s. The smaller workforce could partially explain lower mortality rates over time. Declining mortality rates could also be due to a low proportion of CWP deaths being recorded as such on death certificates potentially due to lack of familiarity with CWP among those charged with completing death certificates. It is possible to assess premature mortality by estimating years of potential life lost among persons with a specific cause of death listed on their death certificate. A study found that during 1996–2016, mean CWP-associated years of potential life lost per decedent increased from 8.1 to 12.6 years, which could be attributable to increases in the prevalence of severe CWP, especially among relatively young coal miners (229).

Although early attention was focused on radiographically apparent CWP, researchers soon turned to investigating other lung diseases caused by coal mine dust. Coal mine dust exposure is a risk factor for COPD (chronic bronchitis and emphysema), which is not usually identified by chest radiography. Chronic bronchitis was initially studied by analyzing responses to questions on respiratory symptoms. The findings showed that the prevalence and incidence of persistent cough and phlegm were related to dust exposure, after controlling for age and smoking (230, 231). COPD has been investigated extensively using measures of ventilatory function, primarily the forced expiratory volume in 1 s (FEV_1) (33, 184, 195, 232–234). Progressively lower FEV_1 has also

been identified as associated with increasing profusion of simple CWP among working miners while controlling for smoking status, body mass index, and tenure (235). The evidence has overwhelmingly shown an inverse relationship between cumulative dust exposure and lung function. The effect has been observed in both smokers and never smokers, and, among older miners, has tended to be somewhat smaller in never smokers. Importantly, the exposure–response relationship was evident in miners without radiographic evidence of CWP (33, 184), and the degree of functional impairment can be clinically important (184, 236, 237), and lead to premature mortality (238–240). Research has also identified a higher prevalence of airflow obstruction among never-smoking coal miners with CWP compared with those without CWP (181). Research focused on miners in the western coalfields of the U.S. found that female coal miners were less likely than male counterparts to have COPD after adjustment for relevant risk factors (241).

Emphysema develops in coal miners in association with CWP (182, 242). Centriacinar (focal) emphysema is a component of the coal dust macule according to the pathological definition of simple CWP (27). Emphysema severity has been shown to be greater in coal miners than in nonminers, after adjustment for smoking, and has been associated with cumulative dust exposure or lung dust burden, after adjustment for smoking and other factors (182, 183). Coal mine dust exposure has been identified as an independent risk factor for emphysema (183).

Other diseases known to be associated with coal mine dust exposure, but less frequently, are Caplan's syndrome, rheumatoid pneumoconiosis, and tuberculosis (243). The latter disease, which historically was a major problem among miners, is seen much less frequently today. It is typically manifested in conjunction with PMF, and it is still not known whether it is an etiologic factor in the occurrence of that disease or whether it develops more frequently among individuals with compromised lungs. Small airway disease can cause significant functional impairment and is increasingly acknowledged as an important respiratory consequence of coal mine dust exposure (244).

1.4.2.3.3 Pharmacokinetics, Metabolism, and Mechanisms

1.4.2.3.3.1 Deposition. The major route of exposure of coal dust is through inhalation. The human respiratory tract includes the nasopharyngeal (head airways), tracheobronchial, and alveolar (pulmonary) anatomical regions (245, 246). Particles in the inhaled air that remain behind after exhalation are referred to as deposited. The main factors that determine the depth of particle penetration and site of deposition within the human respiratory tract depend primarily on the particle characteristics (especially the aerodynamic particle size, as well as shape and charge), the breathing pattern, the level of exertion, and the lung morphology (245). The particle deposition fraction can be greater among individuals with lung diseases such as COPD (247). The size distribution of coal mine dust includes particles that can deposit in each of the regions of the respiratory tract (248). Respirable particles are those particles that are capable of depositing in the alveolar (gas-exchange) region of the lungs (245, 246), where pneumoconiosis can develop in lung tissues around particle deposits (Section 1.4.2.2.2).

1.4.2.3.3.2 Clearance. Clearance is the removal of material from the respiratory tract by particle transport or absorption into the blood (245). For poorly soluble particles such as coal mine dust, clearance occurs mainly by mechanical removal of the particles. Particles that deposit in the tracheobronchial region are cleared by mucociliary transport. Ciliated epithelial cells of the airways move mucus and particles upward toward the oropharynx, where particles may be cleared by coughing, or swallowed and transported to the gastrointestinal tract. A small fraction of particles that deposit in this region are retained in the airway walls (245). Particles that deposit in the alveolar region are engulfed by macrophages and then transported to the tracheobronchial region for mucociliary clearance. Particles that escape alveolar macrophage clearance may enter the alveolar interstitium, where they may be slowly cleared to the lung-associated lymph nodes.

Based on studies of humans who inhaled monodisperse radiolabeled, insoluble particles, both fast- and slow-phase particle clearance from the human respiratory tract have been observed. A fast phase with a clearance half-time of less than 24 h is thought to represent mucociliary clearance, whereas the slower phases with clearance half-times ranging from 30 to several hundred days are thought to represent particle clearance from the alveolar and interstitial regions (245), including both alveolar macrophage-mediated clearance from the lungs and the transport of interstitialized particles to the lung-associated lymph nodes.

Clearance is reduced in the lungs of coal miners who had relatively high cumulative dust exposures compared to that expected based on studies of individuals without occupational dust exposure. In a study of retired coal miners, Freedman and Robinson (150) found no detectable clearance using magnetopneumography among 7 of the 26 retired miners examined and reduced clearance among the other 19 miners (clearance half-time >5 years). In a separate group of U.S. coal miners, Kuempel et al. (151) observed no evidence of particle clearance from the lungs of miners during the retirement period. These findings indicate that particles are not being cleared from miners' lungs as a simple first-order clearance process. A simple first-order clearance model predicts that a steady-state lung dust burden would eventually be reached and that clearance would not be impaired either during or after exposure. However, reduced clearance results in higher retained lung dust burdens, which

is associated with the development of pneumoconiosis and other lung diseases, as discussed in the following section.

1.4.2.3.3.3 Retention. Retention is the temporal distribution of uncleared particles in the respiratory tract (249). Historically, coal miners have retained lung dust burdens greater than those associated with overloading of alveolar clearance in rodents (see Section 1.4.1.3). Studies from the United States, United Kingdom, and Germany have reported average lung dust burdens from approximately 10–20 mg dust per g tissue (134–143), while lower lung burdens (<5 mg/g) were reported among Bulgarian coal miners exposed to brown (lignite) coal (250). The reduced particle clearance from the lungs of retired U.S. coal miners (150) is consistent with a process, such as overloading, that would lead to greater lung dust retention than expected from first-order clearance kinetics. Other processes such as interstitialization and/or sequestration of dust could also cause increased dust retention and decreased lung clearance in these miners. Although the retained lung dust burdens in coal miners have been substantially higher than expected based on first-order clearance kinetics (i.e., no impairment), they were lower than predicted from rat-based overload models (144–146). The ICRP model (245) with three separate first-order clearance rate coefficients representing the different particle clearance phases more closely predicts the long-term particle retention pattern observed in coal miners (251). However, the model structure that best describes the retained particle lung burdens in coal miners is a higher-order kinetic model containing an interstitial-sequestration compartment with very slow clearance to the lung-associated lymph nodes (145, 146). This model was developed and calibrated using data in autopsied U.S. coal miners (144) and independently verified using data of autopsied U.K. coal miners (146). The findings from the dosimetry modeling studies in coal miners are consistent with the observations of little or no clearance of particles from the lungs of retired coal miners (150, 151). Although these coal miner datasets included mostly individuals with relatively high cumulative dust exposures, a study in workers with low exposures to inhaled radioactive cobalt particles also showed that the interstitial-sequestration model provided a better fit to the long-term particle retention data (252).

The mass of dust retained in the lungs is associated with the severity of fibrosis (142, 143). Among miners who had developed PMF, the mass of dust retained in the lungs was greater than that in other miners who had similar cumulative exposure. Bergman and Casswell (253) also reported greater dust accumulation in the lungs of miners who had developed PMF, as well as among miners who had worked in high-rank coal (which is associated with higher disease prevalence). Both studies showed that miners with severe fibrotic disease had higher retained lung burdens per unit of exposure, although it could not be determined whether the increased dust retention occurred before or after the disease development. Interindividual differences in the particle deposition and clearance kinetics (245) as well as susceptibility factors could contribute to disease development. However, other evidence suggests that the presence of pneumoconiosis may not influence the deposition rate of inhaled particles. In a study of U.K. coal miners, Love et al. (254) observed no difference in the average deposition rate of a 1 mm diameter aerosol (amount deposited per volume air breathed) among coal miners with or without radiographic evidence of pneumoconiosis (groups were matched in age and occupational history).

The composition of airborne dust that workers breathed was associated with the composition of dust found in miners' lungs and lymph nodes (143). For example, miners of the lower-ranked coal had a greater proportion of noncoal dust, that is, ash (including kaolin, mica, and quartz) in their lungs and lymph nodes than miners of higher-rank coal.

Smoking appears to influence the amount of dust retained in the lungs of coal miners. Among U.S. coal miners, lower lung dust burdens have been observed among smokers than nonsmokers (150). This finding was also observed in a separate group of U.S. coal miners, in which those who smoked had lower end-of-life lung dust burdens than miners who had never smoked, even after accounting for cumulative exposure (128, 151). Because it has been shown in other populations that alveolar clearance is slower among smokers than nonsmokers (255, 256), it is not likely that miners who smoked had faster alveolar clearance of dust. Alternatively, miners who smoked may have had reduced deposition of dust in the alveolar region. This reduced deposition could be due to mucus hypersecretion and dust trapping in the proximal airways combined with enhanced cough clearance (151). This possibility is consistent with the finding of reduced deposition of particles in the alveolar region of individuals with bronchitis (257) or in the terminal bronchioles of smokers (258).

The retention of particles in the lungs may also depend on particle toxicity. Quartz in the lungs may be transferred to the lung-associated lymph nodes to a greater extent than other less toxic components of inhaled coal miner dust. Chapman and Ruckley (259) found mean quartz of 20.3% in the lymph nodes compared to 6.1% in the dust in the lung tissue, although no association was observed with the degree of fibrosis. The proportions of kaolin and mica were comparable in the lungs and lymph nodes. A higher rate of particle translocation from the lungs to the lymph nodes was also observed for quartz compared to coal dust in the lung dosimetry modeling of the U.K. coal miners (146).

1.4.2.3.3.4 Extrathoracic Transport. Particles may also be transported to other organs of the body apart from the lungs and lung-associated lymph nodes. Particles, as black pigment, have been found in the liver and spleen of coal

Table 5. Coal Mining and Underground Miner Exposure Monitoring in Selected Countries

Country	Total Production in 2015[a]		Underground Mining		Dust Exposure Limits and Required Monitoring Device
	Rank	Thousand Short Tons	Est. Percent of Total Production[a]	Typical Method	
People's Republic of China	1	4,376,984	90%	Longwall	Depending on a silica content ranging from 5% to 50%, exposure limits are between 6 and 1 mg/m^3 for RCMD and between 20 and 2 mg/m^3 for total coal mine dust, using a personal gravimetric sampler
United States	2	896,941	34%	Room-and-pillar, longwall	RCMD exposure limit is 1.5 mg/m^3, using a continuous personal dust monitor. Quartz exposure limit is 0.10 mg/m^3, using a personal gravimetric sampler
India	3	643,720	10%	Room-and-pillar, longwall	RCMD exposure limit is 2 mg/m^3, when the silica content is less than 5%, using a monitoring device approved by the Indian government. When the silica concentration is 5% or more, the exposure limit is calculated as 10 divided by the percent silica content in the RCMD
Australia	4	560,714	20%	Longwall	New South Wales: Exposure limits: 2.5 mg/m^3 for RCMD with a quartz content less than 5%, 10 mg/m^3 for the inhalable fraction (particles less than 100 μm in diameter), and 0.12 mg/m^3 for quartz. Personal gravimetric sampling is used Queensland: Exposure limits: 3 mg/m^3 for RCMD with a quartz content less than 5%, 10 mg/m^3 for the inhalable fraction, and 0.10 mg/m^3 for quartz. Personal gravimetric sampling is used
Republic of South Africa	7	256,876	50%	Room-and-pillar, some longwall	Exposure limit is 2 mg/m^3 for RCMD with a quartz content less than 5%. If the quartz content is greater than 5%, an exposure limit for quartz is 0.1 mg/m. Personal gravimetric sampling is used
Germany	8	203,613	3%	Longwall	Using a dose-based approach, limiting cumulative exposure for a 2-year, 220-shift exposure would be an estimated dose accumulated from an average exposure of 4.0 mg/m^3. Area gravimetric sampling is used
Poland	9	149,147	53%	Longwall	Exposure limits: 1 mg/m^3 for RCMD containing free crystalline silica from 2% to 50%. 4 mg/m^3 for total dust. Personal gravimetric sampling is used

[a]EIA (271).

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miners (260). The amount of black pigment was significantly and positively correlated with the number of years worked underground, the duration of retirement, the age at death, and the severity of pneumoconiosis or emphysema. These findings indicate that extrathoracic particle transfer increases with increasing exposure and continues after exposure ends. There was a significant negative association between the amount of black pigment in the liver and spleen and smoking status in these miners. The basis for this is unclear, although it may reflect the lower lung dust burdens among smokers in that dataset, after accounting for cumulative exposure (151).

1.4.2.3.4 Reproductive and Developmental. No information.

1.4.2.3.5 Carcinogenesis. While earlier mortality studies of lung cancer among coal miners demonstrated either no differences or lower rates when compared with reference populations, more recent studies have identified higher rates among coal miners (155, 261, 262). A large multi-study database analysis, including miners from Canada, New Zealand, and several European countries, did identify an elevated smoking-adjusted lung cancer risk among coal miners, though a specific toxic substance was not implicated (263). A study focused on U.S. coal miners identified increased mortality from malignant respiratory disease, controlling for smoking history (264), and a study of Chinese coal miners also identified an increased risk of lung cancer when compared to nonminers (265). An elevated

Table 6. Exposure Limits, Recommendations, and Guidelines for the United States

Agency	Limit	Type	Comments
MSHA (PEL)	1.5 mg/m^3	TWA	Coal dust with ≤ 5% silica, respirable fraction
	10 mg/m^3/%SiO_2	TWA	Coal dust with > 5% silica, respirable fraction
OSHA (PEL)	2.4 mg/m^3	TWA	Coal dust with < 5% silica, respirable fraction
	10 mg/m^3/%SiO_2 + 2	TWA	Coal dust with > 5% silica, respirable fraction
NIOSH (REL)	1 mg/m^3	TWA	Respirable fraction

PEL, permissible exposure limit; REL, recommended exposure limit; TWA, time-weighted average.
Adapted from MSHA (223), OSHA (272), and NIOSH (273).

lung cancer rate in German coal miners was associated with pneumoconiosis (CWP radiographic category >1/1), suggesting increased susceptibility related to fibrosis (266). A study of U.K. coal miners reported an association between lung cancer and quartz exposure (261).

Elevated stomach cancer rates have been reported in underground coal miners (267, 268), and a study of U.K. coal miners reported a relationship between digestive system cancer mortality and dust exposure (240). In U.S. coal miners, the stomach cancer mortality rate was somewhat increased at higher cumulative exposures but not significantly elevated (239).

1.4.2.3.6 Genetic and Related Cellular Effects Studies. See Section 1.4.1.6.

1.4.2.3.7 Other: Neurological, Skin Sensitization, and So On. No information.

1.5 Standards, Regulations, or Guidelines of Exposure

The health risks associated with exposure to coal mine dust in the United States and other high-producing countries continue to be emphasized (269, 270). Because of this ongoing recognition, most coal-producing nations have established or implemented dust exposure limits for protecting workers from overexposure to injurious dust levels. The U.S. National Academy of Sciences undertook a review of the practices of the top coal-producing nations' approaches to dust monitoring in 2018 (8). Table 5 presents dust exposure limits and required monitoring approaches for selected countries.

Exposure limits for coal mine dust in the United States are provided in Table 6. A recent NIOSH review of pneumoconiosis among U.S. coal miners described these exposure limits:

In 1995, NIOSH published a criteria document providing a scientific basis for an updated REL for respirable coal mine dust of 1.0 mg/m^3 as a TWA concentration for up to 10 h/day during a 40-h workweek and recommended adherence in coal mines to the NIOSH REL for respirable crystalline silica of 0.05 mg/m^3 as a TWA concentration for up to 10 h/day during a 40-h workweek (189). In 2016, as specified by the 2014 MSHA final rule, the long-standing permissible exposure limit (PEL) for respirable coal mine dust in underground coal mines was reduced from a maximum 8-h shift average of 2.0 mg/m^3 to a full-shift average of 1.5 mg/m^3. The exposure limit is reduced if the respirable quartz content of respirable coal mine dust exceeds a full-shift average of 100 μg/m^3, as specified in the 2014 MSHA final rule (223, 274). The rule also made changes to how coal mine dust exposures are measured, including increased sampling frequency in higher dust areas and added requirements for using continuous personal dust monitors (CPDMs) in high-risk mining occupations. CPDMs generate real-time dust measurements for the wearer, and results can be transmitted to MSHA more rapidly than traditional sampling methods. According to federal data, operator compliance with dust standards has been high for decades, with >99% compliance since the PEL was reduced (8, 81, 83).

The current American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV)-TWA for coal dust is 0.9 mg/m^3 for respirable bituminous or lignite coal dust and 0.4 mg/m^3 for respirable anthracite (275).

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DISCLAIMER

The findings and conclusions in this chapter are those of the author(s) and do not necessarily represent the official position of the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention.

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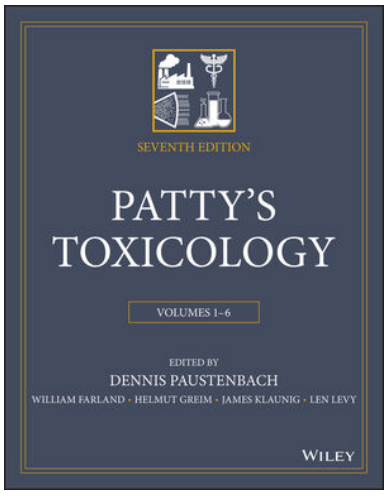

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