Pneumoconiosis in Coal Miners: Anthracosilicosis after All?

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In this issue of *AnnalsATS*, Cohen and colleagues (pp. 1469–1478) address a question that has kept medical and nonmedical experts and stakeholders busy for more than a century: what is the role of silica in coal workers' pneumoconiosis (CWP) (1)? The question may seem futile, but it has (had) serious implications for the prevention, diagnosis, and compensation of lung disease in coal workers (2).

From the 1830s, it became clear that the black material in dead miners' lungs was inhaled coal dust. Publications of cases of coal miners' lung disease—with symptoms of breathlessness and black sputa or black pigmented lungs at autopsy—produced various names for the condition, such as miners' asthma, black spit, spurious melanosis, anthracosis, phthisis melanotika, and pneumoconiosis anthracosica (3–5). However, by the 1860s the issue lapsed into obscurity. By the beginning of the 20th century, authoritative physicians in the United Kingdom, such as J. S. Haldane and T. Oliver, even claimed that improved working conditions had virtually eliminated coal workers' lung disease (5, 6). Moreover, by the 1930s, the (rightful) attention given to silica dust and silicosis overshadowed all other causes of pneumoconiosis. The coal dust itself was not considered hazardous, but the silica that was present in the dust was (or tuberculosis "caught not in the pit but in the home or elsewhere") (6). Anthracosis (or black lung) was discounted as a real disease, and because lung disease in coal miners was to be considered merely a form of silicosis, the term "anthracosilicosis" was proposed.

This concept has had profound implications. First, because employers drew reassurance from this ill-founded narrow etiology of coal workers' lung disease, barely any attempts for dust control were made, while mechanized mining methods caused increased concentrations of airborne coal dust (2). Second, the concept impeded many ill coal miners from obtaining compensation. In Europe and the United States, the first compensation schemes applied only to miners who had worked in siliceous rock and, hence, had developed classical silicosis—but these constituted only a small fraction of all miners with lung disease. In the United Kingdom, the idea that only silica was deleterious changed after a study conducted by the Medical Research Council in 1936–1942 convincingly showed that coal trimmers in the South Wales docks—who did not inhale rock dust—had similar radiographic and histologic lung changes as coal-face workers (7). The Medical Research Council recommended dropping the word silicosis and labeling the condition coal workers' pneumoconiosis. From 1943, the new British compensation scheme would apply to coal miners at the face, rock workers, and coal trimmers at the docks (2). Despite similar evidence in the United States (8), an analogous scheme was only introduced in the United States in 1969 with the enactment of the Federal Coal Mine Health and Safety Act.

Since 1970, regulatory measures to limit respirable dust exposure in U.S. coal mines, together with active surveillance of miners' lung health, have been accompanied by a steady decline in the occurrence of CWP until its "near eradication" by the mid-1990s (9). However, as demonstrated in numerous publications, a "troubling resurgence of severe pneumoconiosis" began in the late 1990s, especially in the Central Appalachian coalfields (9). This reversal of the downward trend in the occurrence of serious CWP, often affecting miners with relatively short tenures, was soon believed to be due to higher respirable silica exposures, brought about by changes ("advances") in mining technology, which allowed obtaining coal from thin coal seams (while also cutting more silica-containing rocks). The possible role of silica, however, remained based on ecologic associations or plausible but largely anecdotal evidence.

Cohen and coworkers addressed the issue of the role of silica by means of careful pathological studies of lung tissue of coal workers with pneumoconiosis. In a first study (10), a review of the lung pathology of 13 selected cases of coal miners with rapidly progressive pneumoconiosis indicated that silica and silicates were indeed implicated in the disease. Cohen and coworkers then smartly designed another study (1) to test formally the hypothesis that silica exposure may be implicated in the resurgence of severe pneumoconiosis in U.S. coal miners. Seven pathologists with expertise and experience in dust-related lung disease (three of whom had participated in the first study [10]) independently reviewed lung tissue slides from 85 coal miners with histologically defined progressive massive fibrosis, using a standardized scoring system (while being blinded to demographic or other individual information). They compared the findings of two groups defined according to their presumed intensity of silica exposure: 23 "contemporary" miners born after 1930—who had worked mainly after the introduction of

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technologies generating high concentrations of silica dust—versus 62 "historical" miners born before 1930. *In situ* mineralogic analysis to quantify silica particles was available for lung tissue specimens from 17 contemporary and 33 historical miners. As expected, silicotic lesions and silica particles were present in addition to coal macules in lung tissue from both groups. However, lung tissue from contemporary miners showed higher proportions of features associated with silicosis, as well as substantially higher silica particle concentrations, than lung tissue from historical miners. Of interest—and importance—the lesions suggestive of high silica exposure included not only typical textbook silicotic nodules but also "immature" silicotic nodules as well as foci of "mineral dust–related alveolar proteinosis," a hitherto neglected feature indicative of acute and accelerated silicosis, according to the authors. Although these features were already known before the landmark 1930 Johannesburg International Labor Office (ILO) Conference on silicosis (11), they have indeed not often been taken into consideration.

In spite of relatively low numbers of contemporary miners (and, hence, potentially some selection bias) and the absence of radiology, the merit of Cohen and colleagues' study (1) is that it provides strong support for the hypothesis that the resurgence of severe pneumoconiosis is due to more intense exposure to respirable silica. The scientific conclusion and practical implication are, therefore, that even though silica is not necessary for causing CWP, high exposure to respirable silica increases the likelihood of early severe pneumoconiosis. In other words, anthracosilicosis was not such a bad concept after all.

Currently, it is well recognized that coal mine dust can induce a spectrum of conditions, now collectively termed "coal mine dust lung disease" (12). In addition to the "classical" CWP, coal miners are at risk of dust-related diffuse fibrosis (with features of "idiopathic" lung fibrosis) and chronic airway diseases, including emphysema and chronic bronchitis (12). Moreover, the concept of coal dust as an isolated cause of respiratory disease is artificial, because coal miners are commonly exposed to other dusts, fumes, and vapors, of which silica is arguably the one that must require most—but not exclusive—attention. Training pulmonologists, radiologists, and lung pathologists in recognizing dust-induced lung disease remains crucial not only to do justice to the victims but also to detect failures in exposure control and to discover emerging causes of diseases. We believe it is time for ILO to reconvene a conference to disseminate broadly the older and more recently acquired knowledge on dust-induced lung diseases.

In the meantime, dust exposure in coal mines must be kept as low as possible. However, the most effective prevention of coal workers' lung disease will, and should, be achieved through a worldwide cessation of the extraction of coal, because its combustion is one of the most important sources of air pollution and CO_2 production. ■

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

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