Mines and Disease: the Price to Pay for Raw Materials

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Topics

• Silicosis
• Coal worker’s pneumoconiosis
• (Asbestosis)

• Theme: Even though the risk of dust diseases is well established, we still see new cases or new manifestations of disease today, and we can expect to see these diseases in migrants.
Silicosis-Historic Aspects

• Recognition since antiquity that certain dusty occupations associated with lung disease
• Confusing nomenclature obscured silicosis as an entity for many years
  – For example, Ramazzini noted that “marble or stone cutters are often troubled with cough and some of them turn asthmatic and consumptive”
• What we now call silicosis was called in the 19th Century “phthisis” [Greek: “to waste away”] or “consumption”
Silicosis – Historic Aspects

- In the 19th and early 20th Century, silicosis consistently confused with tuberculosis (both called “consumption” or “phthisis”)
  - “Dusts…although they are a serious danger…ought on this account to be kept away from workpeople, as a preventive measure against consumption, are yet only remotely a cause of the disease” (Ransome: The Causes and Prevention of Phthisis, 1890)
  - The spread of consumption is caused by “defective ventilation, overcrowding of dwellings, factories and workshops, insufficient and badly selected food, dampness of soil, intemperance, undue physical or mental strain, overwork, worry and anxiety.”

[Massachusetts Board of Health 1898]
Silicosis – Historic Aspects

• Additional confusion caused by the fact that silicosis predisposes to mycobacterial infections

• Taylor 1906 reported “an enormous death rate from tuberculosis among stone cutters, grinders, lead and copper miners”

• In the early 20th Century, slow recognition that all “phthisis” was not the same:
  – “The tendency of modern pathology is to look upon all pulmonary phthisis or consumption as tuberculosis, but the fact remains that phthisis can be caused by dust”

  [Oliver: Dangerous Trades, 1902]
Silicosis – Historic Aspects

• Early 20th Century: recognition, drawn in major part from studies of South African gold miners, that as opposed to tuberculosis, patients with “fibroid phthisis” often did not have fever, sweats, hemoptysis, nor loss of muscle mass/strength

• British Committee on Compensation for Industrial Diseases (1906) suggested that there were 3 conditions: “tuberculous phthisis”, “fibroid phthisis”, and a mixed form with tuberculosis on top of fibroid phthisis
Silicosis – Historic Aspects

• Betts, JAMA, 1900: Silica dust specifically suggested as a cause of the fibroid form of phthisis or “chalicosus pulmonum” [Betts, JAMA, 1900]

• 1912: (UK) Commission on Miner’s Phthisis and Pulmonary Tuberculosis concluded that “all true cases of miners’ phthisis are thus primarily cases of silicosis”

• “Silicosis” as a diagnosis began to appear in the US in 1917
Silicosis – Historic Aspects

• Lanza 1917: examined 720 hard rock miners from Joplin, Missouri
  – 120 “1st stage silicosis” average age 31, time in mines 3.5 to 4.5 years
  – 142 “2nd stage silicosis” average age 33, time in mines 11 years
  – 68 “3rd stage silicosis” average age 38, time in mines 15 years
  – Average life expectancy after the diagnosis of silicosis: 9.6 years
  – 100 of the 720 had both silicosis and tuberculosis
Silicosis – Historic Aspects

- Introduction of power tools in the late 19th/early 20th Century increased dust exposure
  - Studies of Vermont granite cutters showed that workers in the same industry whose job used power tools had a much higher incidence of silicosis than those who used hand tools or wet processes [Hoffman, 1922]

- Early 20th Century: recognition that silicosis predisposed to tuberculosis
  - Between 1896 and 1918, TB rate for Vermont granite cutters rose from 258 to 957/100,000
  - Between 1896 and 1918, TB rate for the general Vermont population dropped from 207 to 96/100,000
Silicosis – Historic Aspects

• Gawley Bridge WVA 1930: A hydroelectric tunnel (the Hawk’s Nest Tunnel) deliberately bored through rock that was nearly pure sandstone
  – Route picked purposely so that the excavated material could be sold as sand
  – Workers had no respiratory protection
  – Workers were told they were suffering from “tunnelitis”

• Estimated that from 700 to 1500 workers out of 2000 died of acute silicosis [probably silicoproteinosis—my comment]

• [See Cherniack: The Hawk’s Nest Incident: America’s Worst Industrial Disaster, 1986]
In the photo above, dust circles a worker during the construction of the Hawks Nest Tunnel in 1930. Workers on the project were exposed to toxic levels of silica dust; hundreds ultimately died.

COURTESY OF ELKEM METALS COLLECTION, WEST VIRGINIA STATE ARCHIVES
A drilling crew poses for the above photo in 1931. To create the Hawks Nest Tunnel, workers had to drill through nearly pure sandstone, which kicks up toxic silica dust. Workers in the photo are not wearing respirators — a requirement later mandated by Congress after hundreds of men died from exposure to silica in the tunnel.

COURTESY OF ELMER KNIGHT COLLECTION, WEST VIRGINIA STATE ARCHIVES
Occupations with Exposure to Crystalline Silica

- Mining
- Quarrying
- Stone Cutting
- Manufacture/use of Silica Abrasives and Fillers
- Foundry Work
- Ceramic and Refractory Manufacture
- Sandblasting and Grinding
- Hydraulic Fracking (fine sand used to fill cracks)
- Cutting artificial stone countertops
- Denim sandblasting
Pathologic Reactions of the Lung to Silica

• Simple silicosis
• Complicated pneumoconiosis (PMF)
• Silicoproteinosis (acute silicosis)
• Small airway fibrosis
• Emphysema
• Interstitial fibrosis (IPF – like: 12% of patients with radiologic simple silicosis (Arakawa: Chest 2007))
• ? Carcinoma of lung
• ? Autoimmune disease
• ? Vasculitis
Simple Silicosis

Courtesy Dr. H Weill
Simple silicosis
Simple Silicosis - Differential Diagnosis

- Tuberculosis (old, healed)
- Mixed dust (silica plus another dust) pneumoconiosis
Pathologic Reactions of the Lung to Silica

- Simple silicosis
- Complicated pneumoconiosis (PMF)
- Silicoproteinosis (acute silicosis)
- Small airway fibrosis
- Emphysema
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PMF + Simple Silicosis
Pathologic Reactions of the Lung to Silica

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- ? Carcinoma of lung
- ? Autoimmune disease
- ? Vasculitis
Acute Silicosis (Silicoproteinosis)

Courtesy Dr. H Weill
Silica Induced Interstitial Fibrosis
Complications of Silicosis

- Mycobacterial infections
  - TB
  - Atypical mycobacteria
- ?Carcinoma of the lung
- Airflow obstruction (rare in nonsmokers)
- Collagen vascular disease
- ?Glomerulonephritis/ANCA + vasculitis
  - (Makol: Am J Indus Med 2011)
Prevalence of TB in Silicosis: South African Gold Miners
(Cowie et al 1994)

• Annual Incidence of TB by X-ray Status
  – Without silicosis 981/100,000
  – With silicosis 2707/100,000
  – Relative risk 2.8

• Annual Incidence of TB by Category of Pneumoconiosis
  – Category 0 1%
  – Category 1 2.2%
  – Category 2 2.9%
  – Category 3 6.3%
Meta-Analysis of Silica, Silicosis, and Pooled Relative Risk of Lung Cancer
Kurihara and Wada: Indust Health 2004; 42: 303-314

• 54 studies between 1966 and 2001 with control for confounding by smoking and other carcinogens
• For all silica exposures: RR lung cancer = 1.32 (1.23-1.41)
• For patients with silicosis: RR lung cancer = 2.37 (1.98-2.84)
• For patients without silicosis: RR lung cancer = 0.96 (0.81-1.15)

• Reports increased risk of lung cancer with increasing silica dose in both silicotics and non-silicotics

• Reports a multiplicative effect between silica and cigarette smoke in causing lung cancer (hazard ratio 5.07 at highest silica dose)

• NB: A recent comprehensive metaanalysis (Gamble 2011) denies any association of silica exposure and lung ca, but does not separate out silicotics.
In a review of 32 series in the literature, 38-86% of scleroderma cases in men were associated with silica exposure, compared to only 2.5% of cases in women.

Interstitial lung disease was found in 81% of silica-associated cases (but in some cases the distinction between silicosis and scleroderma-associated ILD was not clear).

Men diagnosed with scleroderma should be investigated for silica exposure.
Silicosis and Silica-Induced Autoimmunity in the Diversity Outbred Mouse

Jessica M. Mayeux¹, Gabriela M. Escalante¹, Joseph M. Christy¹, Rahul D. Pawar¹, Dwight H. Kono² and Kenneth M. Pollard¹*

after a transoral dose of 0, 5, or 10 mg crystalline silica in large cohorts of DO mice. Data were further analyzed for correlations, hierarchical clustering, and sex effects. **DO mice exhibited a wide range of responses to silica, including mild to severe silicosis and importantly silica-induced systemic autoimmunity. Strikingly, about half of PBS controls were anti-nuclear antibodies (ANA) positive, however, few had disease-associated specificities, whereas most ANAs in silica-exposed mice showed anti-ENA5 reactivity. Correlation and hierarchical clustering showed close association of silicosis, lung biomarkers, and anti-ENA5, while other autoimmune characteristics, such as ANA and glomerulonephritis, clustered separately. Silica-exposed males had more lung inflammation, bronchoalveolar lavage fluid cells, IL-6, and autoantibodies. DO mice are susceptible to both silicosis and**
Silicosis: Has the Problem Disappeared?

- **Year/Group**                  **Simple Silicosis**
  - Aberdeen Monument Makers 1950 10%
  - 1970                        2%
  - British Pottery Industry 1975 1.6%
  - US Metal Miners 1960         3.4%
  - Brazil -Stone carvers 2004   54%

- UK: silicosis deaths: 28 in 1993; 10 in 2008 (Health & Safety Executive 2011)

- In theory, controlling dust levels should make the problem go away!
Silicosis

Chi Chiu Leung, Ignatius Tak Sun Yu, Weihong Chen

but is especially prevalent in countries of low and middle income, where the burden is often under-reported because of poor surveillance. China has the most patients with silicosis, with more than 500,000 cases recorded between 1991 and 1995, and 6000 new cases and more than 24,000 deaths reported annually.4 The problem is particularly acute for workers in small-scale mines, who often have an accelerated form of disease.5 In the Brazilian gold-mining area in Minas Gerais alone, more than 4500 workers were reported to have had silicosis between 1978 and 1998.6 Of gold miners in South Africa dying from external causes (eg, injuries, burns, poisoning, and drowning), proportions with silicosis identified at autopsy increased from 3% to 32% for black miners and from 18% to 22% for white miners between 1975 and 2007.7
Silicosis

Chi Chiu Leung, Ignatius Tak Sun Yu, Weihong Chen

121,000 workers were exposed to concentrations of respirable crystalline silica of 0.05 mg/m³ or more in 1993, and 3,600–7,300 silicosis cases occurred annually from 1987 to 1996. Overall age-adjusted mortality rates in the USA declined from 8.9 per million in 1968, to 0.7 in 2004. However, silicosis deaths in young adults (aged 15–44 years), which are probably a result of intense and recent exposures, have not fallen since 1995.
Artificial Stone Associated Silicosis: A Systematic Review

Veruscka Leso, Luca Fontana, Rosaria Romano, Paola Gervetti and Ivo Iavicoli

“Artificial Stone” = crushed rocks with resin binder
Silica content ~ 90%
Used for countertops
Reported series date from late 1990s to current
<table>
<thead>
<tr>
<th>Activity Description</th>
<th>Age of Workers (Years)</th>
<th>Exposure Time (Years)</th>
<th>Quality Rating by JBI</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry cutting and polishing of artificial stone for fabrication of small kitchen and</td>
<td>44 (median)</td>
<td>7.3 (median)</td>
<td>Fair</td>
<td>Hoy et al. [17]</td>
</tr>
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<td>bathroom benchtop (7)</td>
<td></td>
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<tr>
<td>Dry cutting of synthetic stone material (Caesar Stone containing &gt;85% crystalline</td>
<td>52 (median)</td>
<td>17 ± 9–22 ± 7 (mean ±</td>
<td>Good</td>
<td>Kramer et al. [7]</td>
</tr>
<tr>
<td>silica) for kitchens and other countertop applications (25)</td>
<td></td>
<td>SD)</td>
<td></td>
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<tr>
<td>Dry cutting and polishing synthetic stone material (with high content of crystalline</td>
<td>44.1 (n. 9 -mean); 50.4</td>
<td>6–26 (9 with</td>
<td>Good</td>
<td>Shtraichman et al.</td>
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<tr>
<td>silica) for kitchens and other countertop applications (40 whom 9 with autoimmune</td>
<td>(n. 31-mean)</td>
<td>autoimmune disease)</td>
<td></td>
<td>[41]</td>
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<td>disease)</td>
<td></td>
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<tr>
<td>Dry cutting and polishing artificial decorative stone products (&gt;93–94% crystalline</td>
<td>47.26 (mean)</td>
<td>19.8 ± 9.4 (mean ± SD)</td>
<td>Fair</td>
<td>Grubstein et al.</td>
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<td>silica) for kitchens and other countertop applications (82)</td>
<td></td>
<td></td>
<td></td>
<td>[42]</td>
</tr>
<tr>
<td>Occupations carrying out job tasks consistent with over-exposure to silica</td>
<td>50 (median)</td>
<td>Not reported</td>
<td>Good</td>
<td>Rosengarten et al.</td>
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<td>through handling artificial stone (17)</td>
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<td>[16]</td>
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<tr>
<td>Cutting, polishing and assembling quartz conglomerates composed of at least 90%</td>
<td>39.81 (mean)</td>
<td>12.54 (mean)</td>
<td>Poor</td>
<td>Pascual et al.</td>
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<td>natural quartz (crystallized silicon dioxide [SiO₂] and silica) (6)</td>
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<td>[44]</td>
</tr>
<tr>
<td>Working activities (cutting, shaping and finishing) in which agglomerated quartz</td>
<td>33 (median)</td>
<td>12.8 (mean)</td>
<td>Good</td>
<td>Perez-Alonso et al.</td>
</tr>
<tr>
<td>was used in the manufacturing of countertops for kitchens (46)</td>
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<td>[43]</td>
</tr>
<tr>
<td>Cutting, sanding and assembling artificial quartz aggregates (with a high content</td>
<td>46.62 ± 13.33 (mean ±</td>
<td>11.00 ± 3.58 (mean ±</td>
<td>Poor</td>
<td>Pascual et al.</td>
</tr>
<tr>
<td>of crystalline silica: 70–90%) for kitchen and bath countertops (13)</td>
<td>SD)</td>
<td>SD)</td>
<td></td>
<td>[45]</td>
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<tr>
<td>Cases (n.)</td>
<td>Respiratory Function Tests</td>
<td>Radiological Assessment</td>
<td>Diagnosis</td>
<td></td>
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<td>7</td>
<td>- 3 restrictive defects;</td>
<td>High-resolution computerized tomographic: semiconfluent nodules in the mid and upper zones, ground glass nodules, bilateral upper lobe fibrosis and volume loss with reticulonodular and large confluent mass-like densities</td>
<td>6 with progressive massive fibrosis; 1 chronic silicosis.</td>
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<tr>
<td></td>
<td>- 3 mixed obstructive/restrictive defects;</td>
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<td></td>
<td>- 1 normal respiratory function test.</td>
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<tr>
<td>25</td>
<td>Moderate to severe restrictive lung disease</td>
<td>Diffuse micronodular pattern and progressive massive fibrosis</td>
<td>2 with progressive massive fibrosis (consistent with accelerated silicosis); 23 chronic silicosis.</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>- Restrictive lung disease (8);</td>
<td>Chest X-ray: reticulonodular interstitial pattern (89%); High-resolution computerized tomographic: lymphadenopathy (with or without calcification), alveolar infiltrates, ground glass opacities</td>
<td>Silicosis</td>
<td></td>
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<td></td>
<td>- Normal (1).</td>
<td></td>
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<tr>
<td>82</td>
<td>Reduced FEV₁: 68.4±26 (mean±SD)</td>
<td>High-resolution computerized tomographic: centrilobular and perilymphatic nodules, nodal enlargement with or without nodal calcification, emphysema, and conglomerate masses—progressive massive fibrosis</td>
<td>31 with progressive massive fibrosis (consistent with accelerated silicosis); 51 chronic silicosis.</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Reduced FEV₁ (median: 31; 25th-75th percentile range: 27-38) TLC (median: 47; 25th-75th percentile range: 41-54)</td>
<td>High-resolution computerized tomographic: picture of interstitial lung disease that was consistent with silicosis in all cases</td>
<td>Silicosis</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>- Mild and moderate restrictive ventilatory disorder (2); - Moderate obstructive ventilatory disorder (1)</td>
<td>Chest X-ray: radiographic patterns of simple chronic silicosis (83.3%) and progressive massive fibrosis (16.66%)</td>
<td>1 with progressive massive fibrosis; 5 chronic silicosis.</td>
<td></td>
</tr>
<tr>
<td>46</td>
<td>- Very moderately restrictive pattern (42): FEV₁=85.9±13, FEV₁/FVC=79.9±5; In 4 cases was observed a more restrictive spirometric profile: FEV₁= 74.5±14, FEV₁/FVC= 76.5±9</td>
<td>Chest X-ray: bilateral diffuse micronodular pattern in 80.4% (37) of the cases; High-resolution computerized tomographic: Micronodules in upper lung zones, diffuse ground glass pattern (3)</td>
<td>4 with complicated chronic silicosis; 42 simple chronic silicosis.</td>
<td></td>
</tr>
</tbody>
</table>
Summary of Leso et al Data

• Very high incidence of simple silicosis
  – In some series 100% of patients
• Patients are young
• Relatively high proportion with complicated pneumoconiosis (PMF)
  – In one series 6/7 patients
• Some series have patients with silicoproteinosis
Silicosis Appears Inevitable Among Former Denim Sandblasters
A 4-Year Follow-up Study

Metin Akgun, MD, FCCP; Omer Araz, MD; Elif Yilmazel Ucar, MD; Adem Karaman, MD; Fatih Alper, MD, PhD; Metin Gorguner, MD; and Kathleen Kreiss, MD

BACKGROUND: The course of denim sandblasting silicosis is unknown. We aimed to reevaluate former sandblasters studied in 2007 for incident silicosis, radiographic progression, pulmonary function loss, and mortality and to examine any associations between these outcomes and previously demonstrated risk factors for silicosis.

METHODS: We defined silicosis on chest radiograph as category 1/0 small opacity profusion using the International Labor Organization classification. We defined radiographic progression as a profusion increase of two or more subcategories, development of a new large opacity, or an increase in large opacity category. We defined pulmonary function loss as a $\geq 12\%$ decrease in FVC.

RESULTS: Among the 145 former sandblasters studied in 2007, 83 were reassessed in 2011. In the 4-year follow-up period, nine (6.2\%) had died at a mean age of 24 years. Of the 74 living sandblasters available for reexamination, the prevalence of silicosis increased from 55.4\% to 95.9\%. Radiographic progression, observed in 82\%, was associated with younger age, never smoking, foreman work, and sleeping at the workplace. Pulmonary function loss, seen in 66\%, was positively associated with never smoking and higher initial FVC \% predicted. Death was associated with never smoking, foreman work, number of different denim-sandblasting places of work, sleeping at the workplace, and lower pulmonary function, of which only the number of different places worked remained in multivariate analyses.

CONCLUSIONS: This 4-year follow-up suggests that almost all former denim sandblasters may develop silicosis, despite short exposures and latency. CHEST 2015; 148(3):647-654
### TABLE 2  ] Chest Radiograph ILO Categorization by Nonparticipants, Decedents, and Participants by Year of Examination

<table>
<thead>
<tr>
<th>ILO Category</th>
<th>Nonparticipants(^a) (n = 62)</th>
<th>Decedents(^a) (n = 9)</th>
<th>Participants (n = 74)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category 0</td>
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<tr>
<td>0/-</td>
<td>27</td>
<td>...</td>
<td>25</td>
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<td>0/0</td>
<td>1</td>
<td>...</td>
<td>2</td>
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<tr>
<td>0/1</td>
<td>7</td>
<td>...</td>
<td>6</td>
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<tr>
<td>Category 1</td>
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<tr>
<td>1/0</td>
<td>5</td>
<td>...</td>
<td>14</td>
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<td>1/1</td>
<td>5</td>
<td>...</td>
<td>4</td>
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<td>1/2</td>
<td>4</td>
<td>...</td>
<td>2</td>
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<td>Category 2</td>
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<td>2/1</td>
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<td>...</td>
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<td>2/2</td>
<td>0</td>
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<td>2/3</td>
<td>7</td>
<td>...</td>
<td>3</td>
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<td>Category 3</td>
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<td>3/2</td>
<td>3</td>
<td>1</td>
<td>4</td>
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<tr>
<td>3/3</td>
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<td>1</td>
<td>3</td>
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<td>3/+</td>
<td>...</td>
<td>6</td>
<td>20</td>
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<tr>
<td>Large opacities</td>
<td>2 (3.2)</td>
<td>4 (44.4)</td>
<td>8 (10.8)</td>
</tr>
<tr>
<td>Type A</td>
<td>...</td>
<td>2</td>
<td>4</td>
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<tr>
<td>Type B</td>
<td>...</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Type C</td>
<td>2</td>
<td>2</td>
<td>1</td>
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</tbody>
</table>

Overall category data given as No. (\%); subcategory data given as No. ILO = International Labor Organization.
Prognosis in Silicosis

• Simple silicosis rarely shortens lifespan unless uncontrolled mycobacterial infection develops
• PMF may lead to loss of function/death
• Acute silicosis (silicoproteinosis) has a poor prognosis even with lavage (Souza: Eur J Radiol 2011)
• Some patients with simple silicosis may develop chronic airflow obstruction (Leung: Lancet 2012; Bruske: Occup Environ Med 2014); the higher the dust exposure, the greater the risk
• NB: OSHA lowered the permitted silica exposure level from 100 μg/m³ to 50 μg/m³ in 2018
Coal Workers’ Pneumoconiosis (CWP) – Historic Aspects

• Reports of coal mining date from 852 AD [Saxon Chronicle of Peterborough]

• Practical underground mining requires (a) ventilation (b) means of keeping the mine dry
  – Invention of the steam engine allowed both of these requirements to be met

• By 1855 Britain was producing 100 million tons of coal per year
Coal Workers’ Pneumoconiosis

• Reports of lung disease in coal miners date to the early 1800’s
  – Laennec 1819: “melanosis in which the black matter infiltrates into the lung”

• 19th Century: lung disease observed in coal miners under a variety of names:
  – Spurious melanosis
  – Miner’s asthma
  – Anthracosis
  – Miner’s phthisis
Coal Workers’ Pneumoconiosis

• Initial confusion of CWP with silicosis since chest x-ray findings similar

• Establishment of CWP as a separate disease dates from studies on coal trimmers in the UK
  – Coal trimmers loaded and distributed coal in the holds of ships
  – UK coal trimmers found to have chest x-ray abnormalities identical to silicosis
  – The coal they loaded had virtually no silica [Collis and Gilchrist 1928; Gough 1940]
  – Clear relationship found between chest x-ray profusion and coal dust content of lungs
Coal Workers’ Pneumoconiosis

• Nonetheless, widespread belief that coal dust was innocuous
  – Haldane: TB uncommon in coal miners, therefore silicosis uncommon in coal miners
  – Gardner: Unlike silica, coal dust nonfibrogenic

• Introduction of mechanization greatly increased dust levels and respiratory disease

• 1942 Hart and Aslett: reported on CWP in the UK

• 1960s: CWP formally recognized in the US
Coal Deposits in North America

Figure 4.2. North American coal deposits by rank.
Prevalence of CWP by Anatomic Lesion in the National Coal Workers’ Autopsy Study 1971-1980

- Macules 46%
- Macules with focal emphysema 36%
- CWP Micronodules 19%
- PMF 5.5%
- Silicotic nodules in lung parenchyma 13%
- Silicotic nodules in hilar nodes 53%
Simple Coal Workers’ Pneumoconiosis (CWP)
Simple Coal Worker’s Pneumoconiosis: Macules with Focal Emphyema
Coal Macule with Focal Emphysema
CWP: Complicated Pneumoconiosis (Progressive Massive Fibrosis, PMF)
Has Coal Worker’s Pneumoconiosis Disappeared?
Prevalence of CWP Lesions For Miners with <25 Years Employment First Employed Pre or Post 1969 (2mg/m³ Standard Introduced 1970)

(Vallyathan et al: Arch Path 2011)
All is Not Well!

- Traditionally rock was hard to separate from coal, so only large coal seams were mined.
- Current technology allows rock to be separated from coal, so seams of 1 meter height can be exploited.
- This leads to silica exposure in some mines.
Severe Occupational Pneumoconiosis Among West Virginian Coal Miners

One Hundred Thirty-eight Cases of Progressive Massive Fibrosis Compensated Between 2000 and 2009

W. Alex Wade, MD; Edward L. Petsonk, MD, FCCP; Byron Young, RPFT; and Idrees Mogri, MD

Background: Miners inhale dust at work and are at a risk for coal workers pneumoconiosis (CWP), a preventable and potentially fatal lung disease. After regulations were implemented in the 1970s, declines were reported in both dust levels and the prevalence of simple and advanced CWP until about 2001, when despite stable reported dust levels, disease levels sharply increased.

Methods: A structured, retrospective chart review was performed to describe the demographics and disease progression for 138 coal miners with progressive massive fibrosis (PMF) whose claims were approved by the West Virginia State Occupational Pneumoconiosis Board between January 2000 and December 2009.

Results: PMF, a complication of CWP, developed in 138 West Virginian coal miners at a mean age of 52.6 years after an average of 30 years work tenure. The time of progression averaged 12.2 years from the last normal chest radiograph until PMF was detected. Lung function declined sharply in both smokers and nonsmokers, averaging 87 mL/y for FEV₁ and 74 mL/y for FVC. The board has confirmed 21 deaths in this group. The most common job activities were operating continuous-mining machines (41%) and roof bolting (19%). Virtually all of these miners’ dust exposures occurred after the implementation of current federal dust regulations.

Conclusions: Contemporary occupational dust exposures have resulted over the past decade in rapidly progressive pneumoconiosis and massive fibrosis in relatively young West Virginian coal miners, leading to important lung dysfunction and premature death.

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Abbreviations: CWP = coal workers pneumoconiosis; PMF = progressive massive fibrosis; WVSOPB = West Virginia State Occupational Pneumoconiosis Board
Lung Pathology in U.S. Coal Workers with Rapidly Progressive Pneumoconiosis Implicates Silica and Silicates

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Abstract

Rationale: Recent reports of progressive massive fibrosis and rapidly progressive pneumoconiosis in U.S. coal miners have raised concerns about excessive exposures to coal mine dust, despite reports of declining dust levels.

Objectives: To evaluate the histologic abnormalities and retained dust particles in available coal miner lung pathology specimens, and to compare these findings with those derived from corresponding chest radiographs.

Methods: Miners with severe disease and available lung tissue were identified through investigator outreach. Demographic as well as smoking and work history information was obtained. Chest radiographs were interpreted according to the International Labor Organization classification scheme to determine if criteria for rapidly progressive pneumoconiosis were confirmed. Pathology slides were scored by three expert pulmonary pathologists using a standardized nomenclature and scoring system.

Measurements and Main Results: Thirteen cases were reviewed, many of which had features of accelerated silicosis and mixed dust lesions. Twelve had progressive massive fibrosis, and 11 had silicosis. Only four had classic lesions of simple coal workers’ pneumoconiosis. Four had diffuse interstitial fibrosis with chronic inflammation, and two had focal alveolar proteinosis. Polarized light microscopy revealed large amounts of birefringent mineral dust particles consistent with silica and silicates; carbonaceous coal dust was less prominent. On the basis of chest imaging studies, specimens with features of silicosis were significantly associated ($P = 0.047$) with rounded (type p, q, or r) opacities, whereas grade 3 interstitial fibrosis was associated ($P = 0.02$) with the presence of irregular (type s, t, or u) opacities.

Conclusions: Our findings suggest that rapidly progressive pneumoconiosis in these miners was associated with exposure to coal mine dust containing high concentrations of respirable silica and silicates.

Keywords: anthracosis; coal mining; pneumoconiosis; pathology; silicosis
Simple Silicosis (contemporary West VA coal miner)
“CWP” in contemporary WVA coal miner with rapidly progressive disease: simple silicosis + PMF. Note absence of black coal pigment (from the Cohen et al series 2016)
Prognosis in CWP

- Simple CWP and Category A PMF not associated with shortening of lifespan
- Category B and C PMF may develop pulmonary hypertension and/or severe functional deficits
- In longitudinal studies, high dust exposure or increasing profusion of CWP opacities associated with accelerated loss of function (Blackley et al: Chest 2015)
- Some patients develop chronic airflow obstruction
  - Effects of long term coal dust exposure just as potent as heavy cigarette smoking in producing emphysema (Kuempel AJRCCM 2009)
- Most authors report no association of CWP with carcinoma of lung, but this has recently been challenged (Graber et al: Occup Env Med 2013)
Conclusions

• The historic recognition of lung disease induced by dusts was often confused and convoluted

• For the major types of dusts (coal dust, silica, asbestos) the types of disease and exposures that cause disease are well established

• Nonetheless, for silica and coal exposure we continue to see new and often severe cases

• Migrants may well have these diseases
Diseases Caused by Asbestos

• Benign Pleural Disease
  – Pleural effusion
  – Pleural fibrosis
  – Pleural plaques
  – Rounded atelectasis

• Malignant Mesothelioma
  – Pleura
  – Peritoneum

• Parenchymal Fibrosis
  – Asbestosis
  – Small airways fibrosis

• Carcinoma of Lung
  – (when asbestosis present)
Asbestosis

- Asbestos used in classical times for funeral shrouds, cloth, and lamp wicks
- 1872: the Italo-English Pure Asbestos Company formed in London
- Quebec deposits first mined at large scale 1860s
- South African deposits first mined at large scale 1890s
- By 1892 the United Asbestos Company offered 100 different products
Asbestosis

• First case of (probably) pulmonary fibrosis reported by Deane in 1898
• First description of asbestosis made by Murray in 1907
  – Issues of separating ‘tuberculous phthisis’ and ‘dust phthisis’ arose
• Radiologic features described by Pancoast 1917
• Word ‘asbestosis’ coined by Cook in 1927
• First suggestion that asbestosis associated with carcinoma of the lung 1934 (Wood and Gloyne)
• Merewether 1949 showed that 13.2% of asbestotics died of lung cancer as opposed to 1.3% of silicotics
• Doll 1955 studied 113 asbestos textile workers and found that 11 died of lung cancer with an expected no of 0.8
Asbestosis: Definition

• Diffuse interstitial fibrosis of the lung caused by exposure to asbestos

• The term asbestosis should not be used for asbestos-induced pleural disease, nor for the mere presence of asbestos bodies without an appropriate pattern of interstitial fibrosis
Criteria for the Diagnosis of Asbestosis

• On Clinical Grounds (from 1986 ATS Statement)
  – A good history of *(high)* exposure
  – Appropriate latency
  – Chest x-ray showing 1/1 or greater s,t, or u type opacities
    • ATS 2004 guidelines accept 1/0
  – Restrictive pattern of pulmonary function
  – Diffusing capacity below the lower limit of normal
  – Inspiratory rales at the lung bases

• On Pathological Grounds
  – An appropriate pattern of diffuse interstitial fibrosis
  – 2 asbestos bodies/cm² of tissue using ordinary 5 micron sections
    • (CAP guidelines 2009)
Asbestosis
Pathologic Patterns in Asbestosis

• Lower zone predominant
• Always fibrotic and paucicellular
  – Early disease: interstitial fibrosis around bronchioles, then linking bronchioles
  – Advanced disease: Mimicking UIP
    • Fibroblast foci less common than in UIP
  – Advanced disease: Resembling fibrotic NSIP
  – Unclassifiable interstitial fibrosis/honeycombing
• 2 or more/cm² asbestos bodies present in ordinary 5μ sections
  – Counted on iron stains!
  – You must average over all sections counted!
Grade 1 asbestosis (interstitial fibrosis in alveolar walls around bronchioles)
Early asbestosis (grade 2) – interstitial fibrosis around bronchiole
Asbestosis with UIP-like pattern (ungradeable)
Asbestos bodies
Asbestosis is a high exposure disease!
Progression and Prognosis in Asbestosis

• Many cases do not progress
• Cases with higher ILO readings at presentation more likely to progress
• Cases with chrysotile exposure less likely to progress than those with amphibole exposure
• Those with severe disease may die of respiratory failure, pulmonary hypertension, lung cancer
# Deaths in Patients with Certified Asbestosis

<table>
<thead>
<tr>
<th>Series</th>
<th>Asbestosis</th>
<th>Ca lung</th>
<th>Mesothelioma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coutts 87</td>
<td>19%</td>
<td>44%</td>
<td>10%</td>
</tr>
<tr>
<td>Berry 81</td>
<td>20%</td>
<td>36%</td>
<td>5%</td>
</tr>
<tr>
<td>McVittie 65</td>
<td>29%</td>
<td>36%</td>
<td>9%</td>
</tr>
</tbody>
</table>
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