Pneumoconioses

- Lung diseases caused by inhalation of dust, mostly at work (*pneumo* = *lung; conis* = dust in Greek).
- Diseases are, also called ‘dust diseases’ or ‘occupational lung diseases’.
- Type of lung disease varies according to the nature of inhaled dust
- Some dusts are inert, cause no reaction, no damage, others cause immunologic damage and predispose to tuberculosis or to neoplasia
- Factors which determine the extent of damage caused by inhaled dusts are:

- 1. Size and shape of the particles;
- 2. Solubility and physicochemical composition;
- 3. Amount of dust retained in lungs;
- 4. Additional effect of other irritants such as tobacco smoke; and
- 5. Host factors such as efficiency of clearance mechanism and immune status of the host.
- Inhaled dust particles larger than 5 μm reach the terminal airways where they are ingested by alveolar macrophages.
- Most of these too are eliminated by expectoration but the remaining accumulate in alveolar tissue.

- Tissue response to inhaled dust may be one of the following three types:
- *Fibrous nodules e.g. in coal-workers' pneumoconiosis and* silicosis.
- *Interstitial fibrosis e.g. in asbestosis.*
- *Hypersensitivity reaction e.g. in berylliosis*

TABLE 17.9: Classification of Pneumoconioses.

Agents	Diseases
A. **INORGANIC (MINERAL) DUSTS**	
1. Coal dust	*Simple coal-workers'* pneumoconiosis
	Progressive massive fibrosis
	Caplan's syndrome
2. Silica	*Silicosis*
	Caplan's syndrome
3. Asbestos	*Asbestosis*
	Pleural diseases
	Tumours
4. Beryllium	*Acute berylliosis*
	Chronic berylliosis
5. Iron oxide	*Pulmonary siderosis*
B. **ORGANIC (BIOLOGIC) DUSTS**	
1. Mouldy hay	*Farmer's lungs*
2. Bagasse	*Bagassosis*
3. Cotton, flax, hemp dust	*Byssinosis*
4. Bird droppings	*Bird-breeders' (bird* fancier's) lung
5. Mushroom compost dust	*Mushroom-workers' lung*
6. Mouldy barley, malt dust	*Malt-workers' lung*
7. Mouldy maple bark	*Maple-bark disease*
8. Silage fermentation	*Silo-fillers' disease*

Coal-Workers' Pneumoconiosis

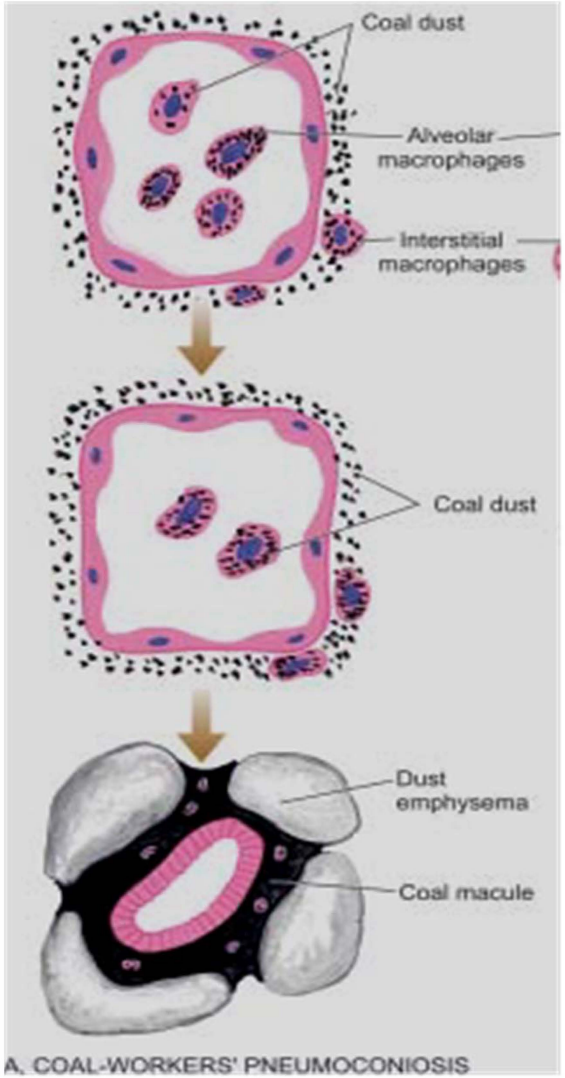
- Lung disease resulting from inhalation of coal dust particles, in coal-miners engaged in handling soft bituminous coal for a number of years, often 20 to 30 years.
- **2 types:**
- Milder form of the disease called *simple coal workers' pneumoconiosis*
- *Advanced form termed progressive massive fibrosis (complicated coal-miners'* pneumoconiosis)
- *Anthracosis, is not a lung* disease in true sense, is common, benign and asymptomatic accumulation of carbon dust in lungs of most urban dwellers due to atmospheric pollution and cigarette smoke

Pathogenesis

- Predisposing factors:
- 1. Older age of the miners.
- 2. Severity of coal dust burden engulfed by macrophages.
- 3. Prolonged exposure (20 to 30 years) to coal dust.
- 4. Concomitant tuberculosis.
- 5. Additional role of silica dust.

- Activation of alveolar macrophage plays the most significant role in the pathogenesis of progressive massive fibrosis by release of various mediators

- i) *Free radicals which are reactive oxygen species which* damage the lung parenchyma.
- ii) *Chemotactic factors for various leucocytes (leukotrienes,* TNF, IL-8 and IL-6) resulting in infiltration into pulmonary tissues by these inflammatory cells which on activation cause further damage.
- iii) *Fibrogenic cytokines such as IL-1, TNF and platelet derived* growth factor (PDGF) which stimulate healing by fibrosis due to proliferation of fibroblasts at the damaged tissue site.



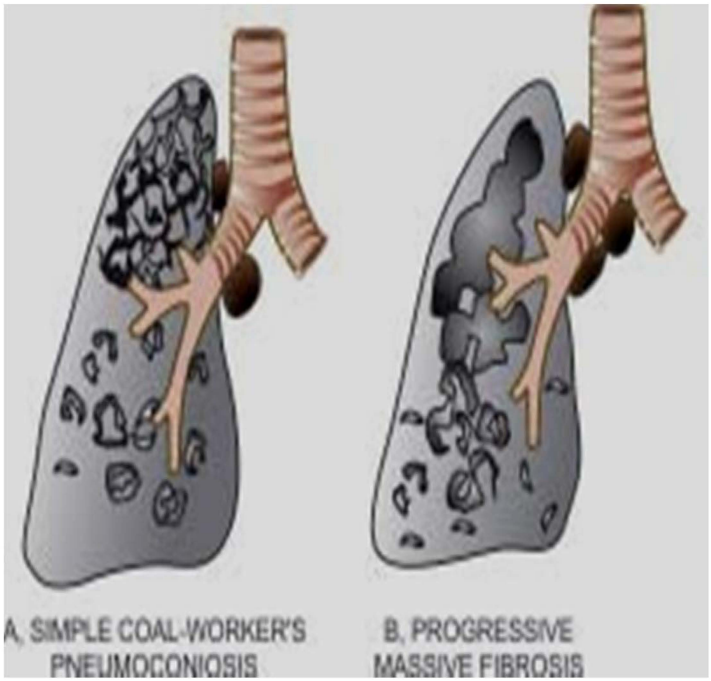
A, COAL-WORKERS' PNEUMOCONIOSIS

Morphology

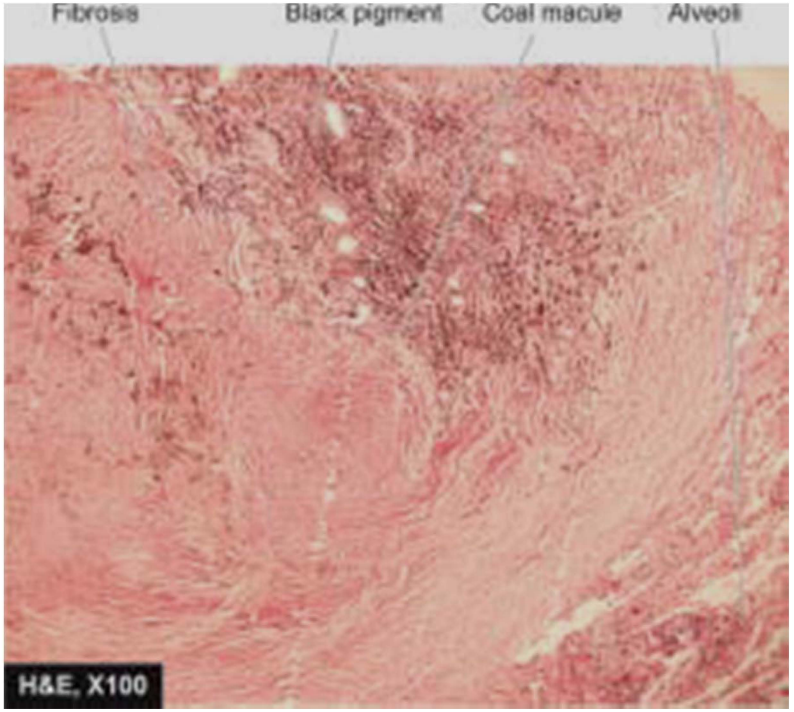
- Pathologic findings at autopsy of lungs in the major forms of coal-workers' pneumoconiosis are of 3 types
- Simple coalworkers' pneumoconiosis
- Progressive massive fibrosis
- Rheumatoid pneumoconiosis (Caplan's syndrome)

Simple coalworkers' pneumoconiosis:

- **G/A:**
- Lung parenchyma shows small, black focal lesions, measuring less than 5 mm in diameter and evenly distributed throughout the lung called *coal macules, and if palpable are called nodules.*


A, SIMPLE COAL-WORKER'S PNEUMOCONIOSIS
B, PROGRESSIVE MASSIVE FIBROSIS

- **M/E:**
- 1. Coal macules are composed of aggregates of dust laden macrophages, present in the alveoli and in the bronchiolar and alveolar walls.
- 2. There is some increase in the network of reticulin and collagen in the coal macules.
- 3. Respiratory bronchioles and alveoli surrounding the macules are distended without significant destruction of the alveolar walls.


Fibrosis
Black pigment
Coal macule
Alveoli
H&E, X100

Progressive massive Fibrosis:

- **G/A:**
- Besides coal macules and nodules of simple pneumoconiosis, there are larger, hard, black scattered areas measuring more than 2 cm in diameter and sometimes massive.
- Sometimes, these masses break down centrally due to ischaemic necrosis or due to tuberculosis forming cavities filled with black semifluid resembling India ink.
- Pleura and regional lymph nodes are also blackened and fibrotic

- **M/E:**
- 1. The fibrous lesions composed almost entirely of dense collagen and carbon pigment.
- 2. Wall of respiratory bronchioles and pulmonary vessels included in the massive scars are thickened and their lumina obliterated.
- 3. Scanty inflammatory infiltrate of lymphocytes and plasma cells around the areas of massive scars.
- 4. Alveoli surrounding scars are markedly dilated.

- **Rheumatoid Pneumoconiosis (CAPLAN'S Syndrome):**
- Development of rheumatoid arthritis in a few cases of coal-workers' pneumoconiosis, silicosis or asbestosis is called rheumatoid pneumoconiosis or Caplan's syndrome.
- **G/A:** Lungs have rounded, firm nodules with central necrosis, cavitation or calcification.
- **M/E:**
- Lung lesions are modified rheumatoid nodules with central zone of dust-laden fibrinoid necrosis enclosed by palisading fibroblasts and mononuclear cells.
- Lung lesions have immunological basis for their origin as there is + rheumatoid factor and antinuclear antibodies.