

Respiratory 6 pneumoconioses

Dr H AWAD

FRCPath

Mineral dust pneumoconiosis

- Pneumoconiosis = non- neoplastic lung reactions to inhalation of mineral dust
- Mineral dust pneumoconioses are usually related to **occupational exposure**
- Mineral dust pneumoconioses : related to coal dust, silica and asbestos

Note:

- Most inhaled dust is entrapped in the mucus blanket and rapidly removed from the lung by ciliary movement,
- However, some of the particles become impacted at alveolar duct bifurcations, where macrophages accumulate and engulf the trapped particulates

PATHOGENESIS :

The reaction of the lung to mineral dusts depends on their size ,shape, solubility, and reactivity as well as purity, concentration and duration of exposure.

Effects of size

- a. 5 to 10 μm Particles are unlikely to reach distal airways,
- b. Particles smaller than 0.5 μm move into and out of alveoli, often without substantial deposition and injury
- c. **1 to 5 μm particles are the most dangerous**, because they get lodged at bifurcation of the distal airways.

Reactivity

- Coal dust is relatively inert, and large amounts must be deposited before lung disease is clinically detectable. .
- Silica, asbestos, and beryllium are more reactive than coal dust, resulting in fibrotic reactions at lower concentrations.

pathogenesis

- The alveolar macrophage is a key cellular element in the initiation and perpetuation of lung injury and fibrosis.
 - a. particles activate the **inflammasome and induce IL-1**
 - b. The more reactive particles trigger the macrophages to release a number of products that **mediate inflammation and initiate fibroblast proliferation and collagen deposition.**

Pathogenesis/ continued

Some of the inhaled particles may reach the lymphatics either by direct drainage or within migrating macrophages and thereby initiate an immune response to components of the particulates and/or to self-proteins that are modified by the particles and this then leads to an **amplification and extension of the local reaction.**

Note:

- Tobacco smoking worsens the effects of all inhaled mineral dusts, more with asbestos than other particles.

Coal workers pneumoconiosis



- Coal is mainly carbon
- Coal mine dust contains also: trace metals, inorganic minerals and silica

Coal workers pneumoconiosis: three types

- **Asymptomatic anthracosis** : pigment accumulates without any reaction
- **Simple coal workers pneumoconiosis (CWP)** : accumulation of macrophages with little or no pulmonary dysfunction. There is minimal fibrosis.
- **Complicated CWP** = progressive massive fibrosis (PMF)= extensive fibrosis with compromised lung function
- Each can progress to the more severe form

NOTE

- Less than 10% of simple CWP progress to PMF
- Carbon is relatively inert... so lung damage is less than that of silicosis or asbestosis.

Note:

- Once smoking-related risk has been taken into account, there is no increased frequency of lung carcinoma in coal miners, a feature that distinguishes CWP from both silica and asbestos exposures .

Morphology/Pulmonary anthracosis

- Carbon pigments engulfed by alveolar or interstitial macrophages
- These macrophages accumulate along the lymphatic vessels or in the lymph nodes
- Also seen in smokers and urban dwellers

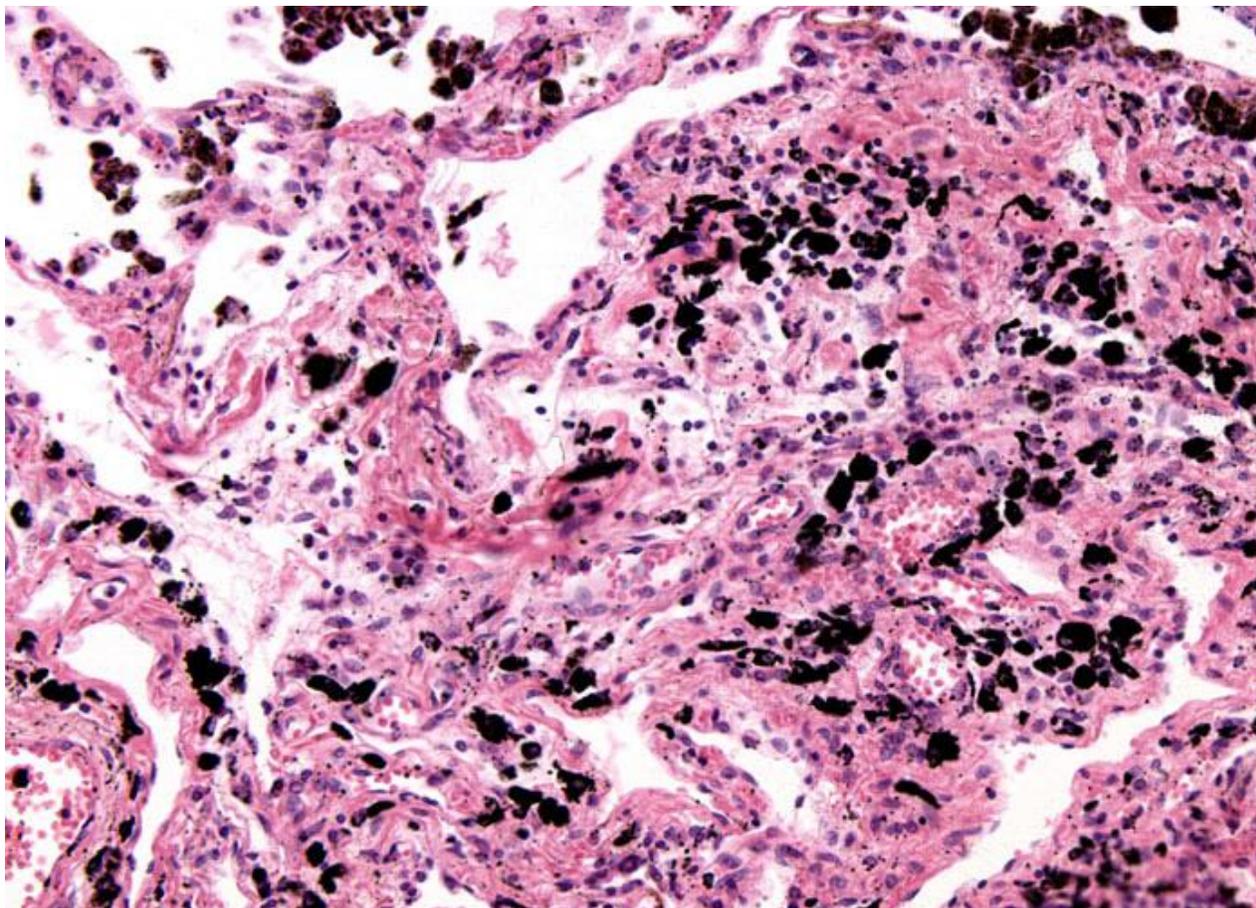
Note:

- PMF is a generic term that applies to a confluent fibrosing reaction in the lung; this can be a complication of any one of the pneumoconioses

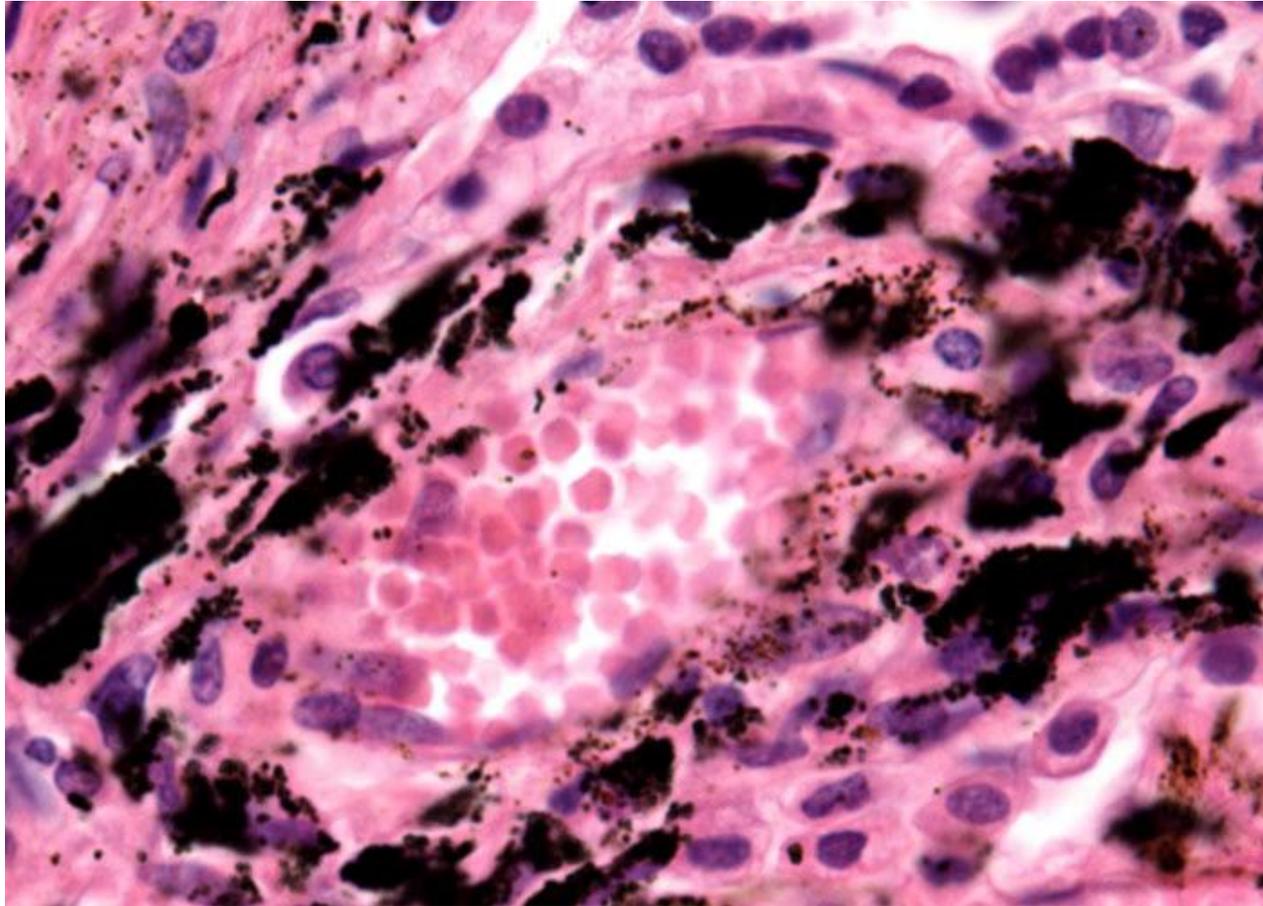
Anthracosis: accumulation of carbon pigment



anthracosis

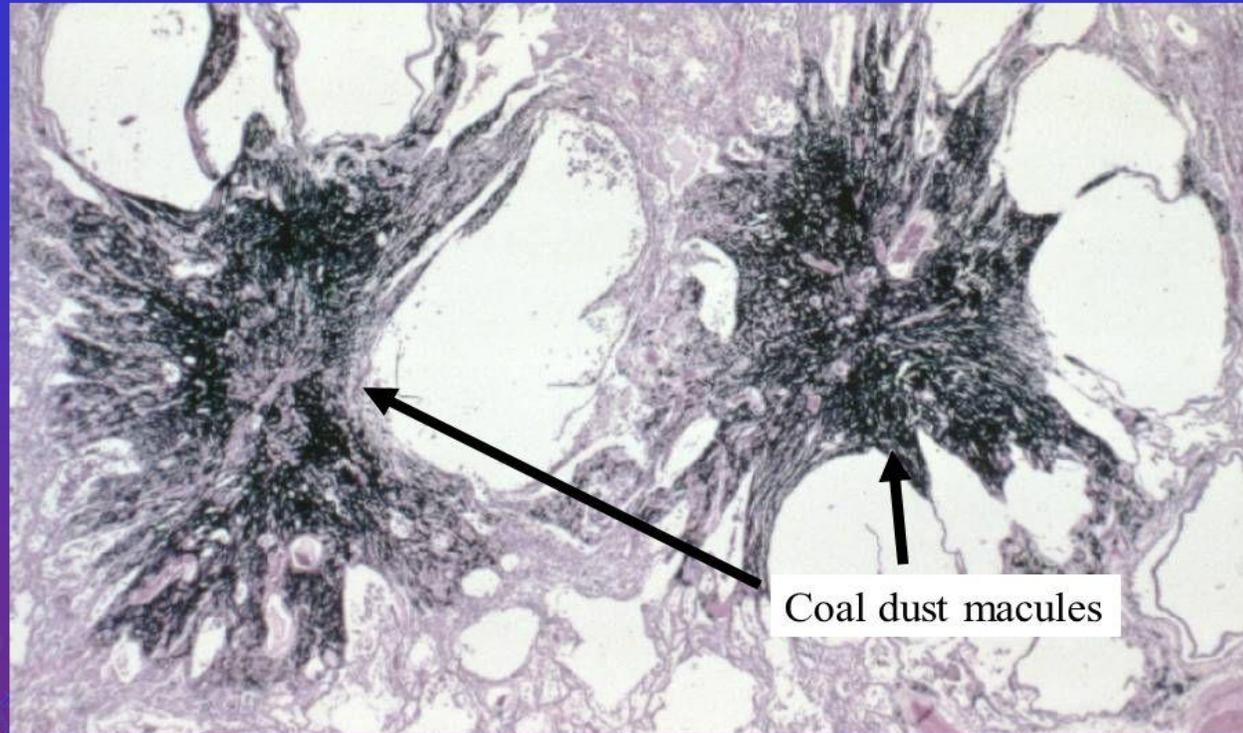


anthracosis



Simple CWP

- Characterized by coal macules and coal nodules
- Coal macules: carbon laden macrophages and small amount of collagen fibers arranged in a delicate network
- These are found mainly in the upper lung lobes
- Centrilobular emphysema can complicate these macules
- If the macules are large they are called coal nodules



Coal-worker's Pneumoconiosis

Complicated CWP= PMF

- There is coalescence of coal nodules .
- Multiple black scars
- Histologically: dense collagen and pigment
- This needs several years of exposure to develop

Coal worker's pneumoconiosis (CWP)

The simple form

- Focal aggregations of coal dust-laden macrophages (coal macules)
- Patients have slight cough and blackish sputum.



The complicated form

- Occurs after many years of underground mine work.
- fibrous scarring appears (complicated CWP) also called progressive massive fibrosis PMF



Clinical features

- Simple CWP is usually a benign disease that produces little effect on lung function
- PMF : pulmonary dysfunction, pulmonary hypertension and cor pulmonale
- Once PMF developed: it progresses even without additional exposure
- **No increased risk of lung cancer in relation to coal exposure.**

silicosis



silicosis

- The most prevalent chronic occupational disease in the world. Caused by inhalation of crystalline silica mostly in occupational settings
- Silica (Silicon dioxide)is a chemical compound that is an oxide of silicon with the chemical formula SiO_2 .
- Silica is most commonly found in nature as quartz and is a major constituent of sand.

Uses of silica

- Glass industry
- Sandblasting : the process used to clean a surface by means of an abrasive such as sand
- Hard rock mining

THROW AWAY
"DO NOT EAT"

DESICCANT
SILICA

GEL
THROW AWAY
"DO NOT EAT"

sandblasting



Silica Forms

- a. Crystalline (such as quartz) are the most toxic and fibrogenic .

NOTE: quartz is most commonly implicated in silicosis and when mixed with other minerals, it has a reduced fibrogenic effect (this is an example of importance of purity of the dust!!)

- b. Amorphous forms : less fibrogenic

MORPHOLOGY

Silicotic nodules :- Characterized grossly in early stages by barely palpable pale-to-blackened (if coal dust is present) nodules in the **upper zones** of the lungs.

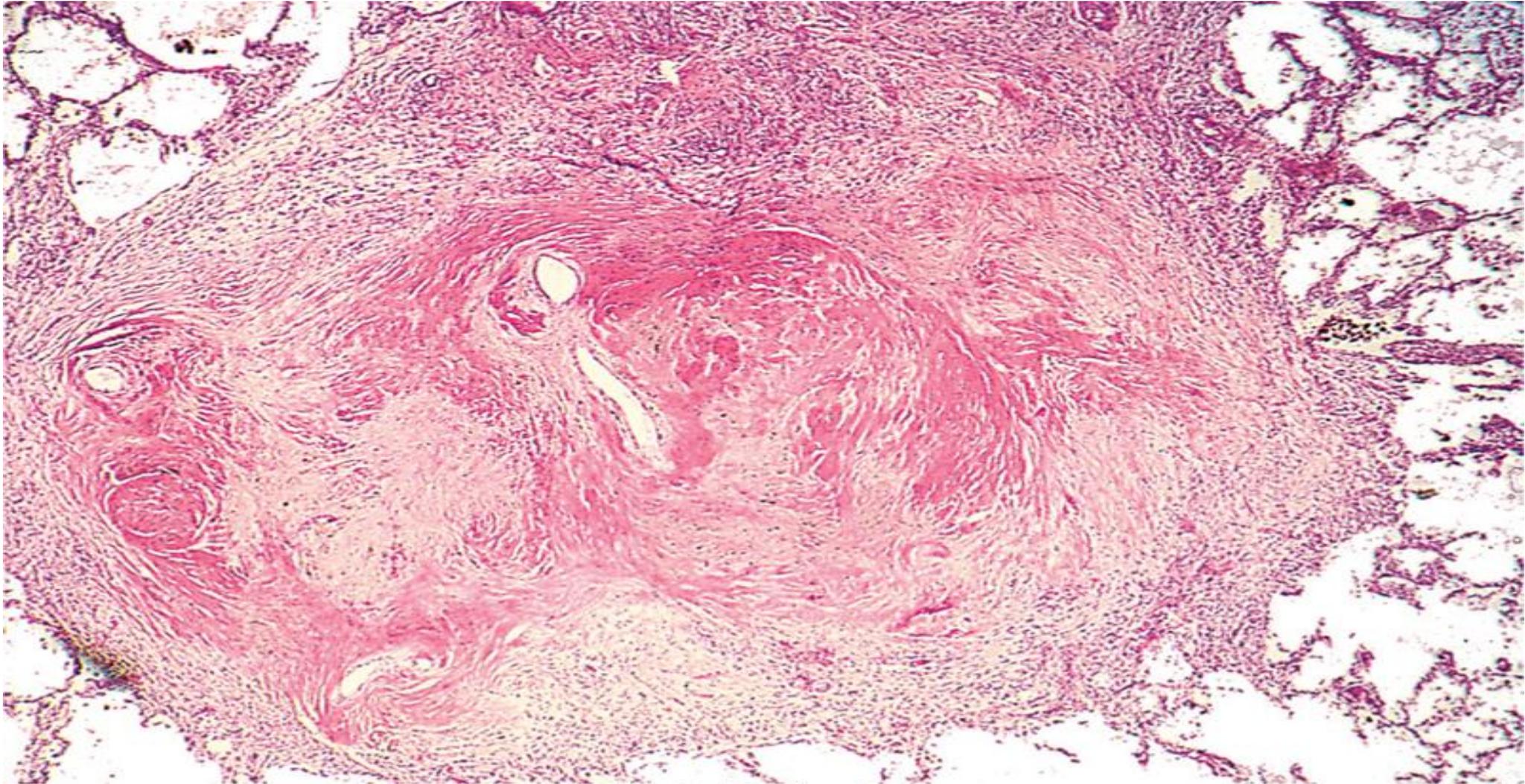
Microscopically,

- concentrically arranged hyalinized collagen fibers surrounding amorphous center
- this “whorled” appearance of the collagen fibers is distinctive for silicosis

Morphology:

- As the disease progresses, the individual nodules may coalesce into hard, collagenous scars, with eventual **progression to PMF**
- Fibrotic lesions may also occur in the hilar lymph nodes and pleura.

silicosis



Silicosis... radiology

- Sometimes, thin sheets of calcification occur in the lymph nodes and are appreciated radiographically as "eggshell" calcification (e.g., calcium surrounding a zone lacking calcification).

Egg-shell calcification



Clinical Features:

- Silicosis usually is detected on routine chest radiographs obtained in asymptomatic workers.
- The radiographs shows a fine nodularity in the upper zones of the lung, but pulmonary function is either normal or only moderately affected.
- Most patients do not develop shortness of breath until late in the course, after PMF is present.

Clinical features

- Once PMF develops , the disease may be progressive, even if the person is no longer exposed.
- Many patients with PMF develop pulmonary hypertension and cor pulmonale.
- The disease is slow to kill, but impaired pulmonary function may severely limit activity

NOTE: Silicosis is associated with an increased susceptibility to tuberculosis.

- It is postulated that crystalline silica may inhibit the ability of pulmonary macrophages to kill phagocytosed mycobacteria.
- Nodules of silicotuberculosis often contain a central zone of caseation

The relationship between silica and *lung cancer* has been a contentious issue.

- In 1997, based on evidence from several epidemiologic studies, the International Agency for Research on Cancer concluded that *crystalline silica* is carcinogenic in humans.
- However, this subject continues to be controversial

asbestosis

- a heat-resistant fibrous silicate mineral that can be woven into fabrics, and is used in fire-resistant and insulating materials such as brake linings.

ASBESTOS



CHRYBOTILE



AMOSITE



CROCIDOLITE



TREMOLITE



ACTINOLITE

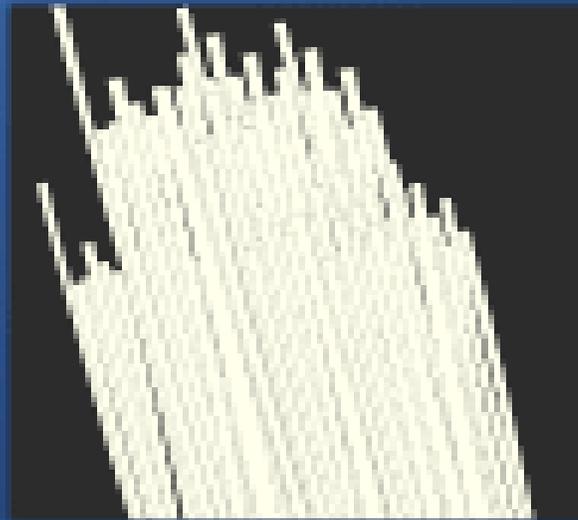


ANTHOPHYLLITE

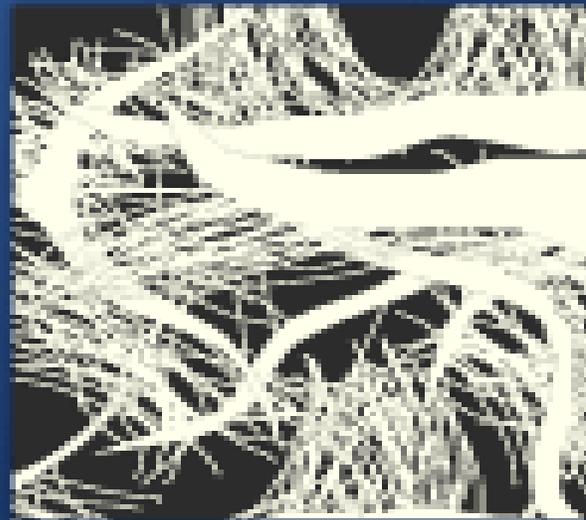
Asbestos Types



Amphibole



Serpentine



AMPHIBOLES

The Big Three made up about 5% of all asbestos commercially used



Anthophyllite



Amosite



crocidolite

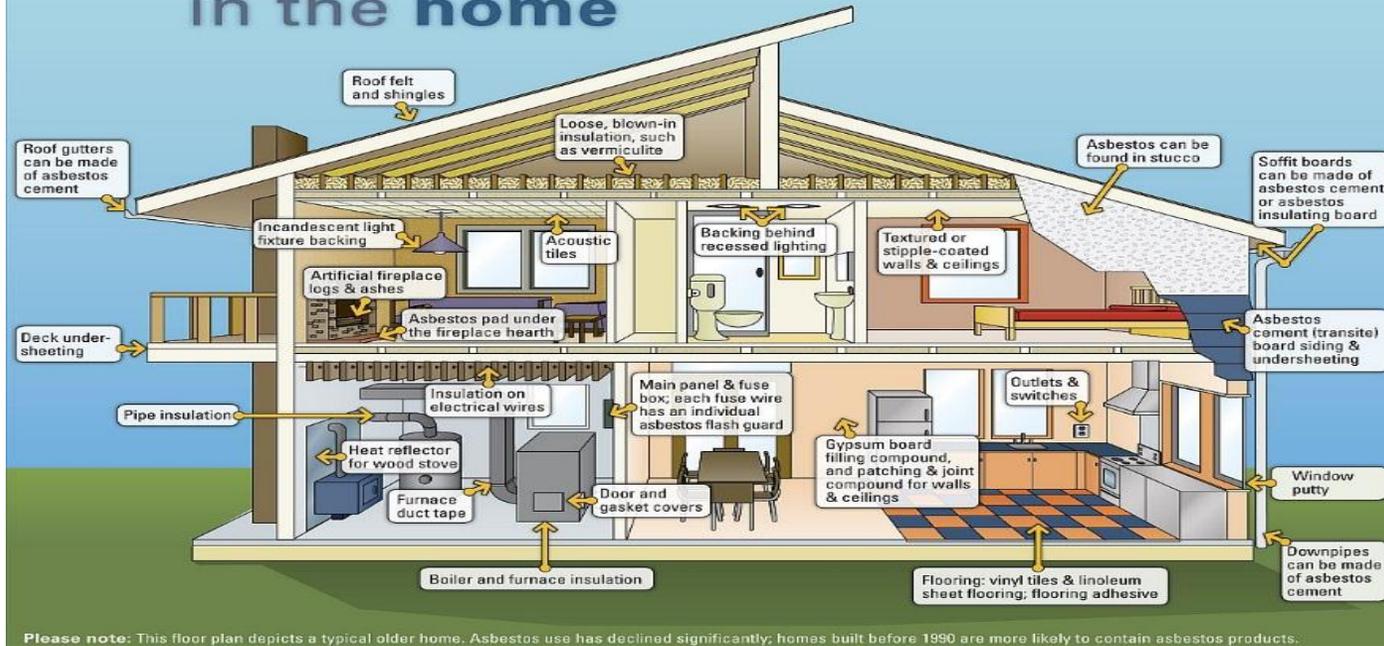
While its evil cousin Tremolite is blamed as the toxic contaminate of chrysotile and thus makes chrysotile toxic.



Tremolite

Potential sources of asbestos in the home

WORK SAFE BC
WORKING TO MAKE A DIFFERENCE



Please note: This floor plan depicts a typical older home. Asbestos use has declined significantly; homes built before 1990 are more likely to contain asbestos products.

- Occupational exposure to asbestos is linked to
 1. Parenchymal interstitial fibrosis (*asbestosis*);
 2. Localized fibrous plaques and rarely, diffuse fibrosis in the pleura;
 3. Pleural effusions;
 4. Lung carcinomas;
 5. Malignant pleural and peritoneal mesotheliomas;
 6. Laryngeal carcinoma.

Important note

- There is an increased incidence of asbestos-related cancers in family members of asbestos workers
- Asbestos can affect people in non-occupational settings

PATHOGENESIS: Forms of asbestos:

A. Serpentine :

- the fiber is curly and flexible, so they are likely to become impacted in the upper respiratory passages and removed by the mucociliary elevator
- Those that are trapped in the lungs are gradually leached from the tissues, because they are more soluble than amphiboles.
- chrysotile (a serpentine fiber) accounts for most of the asbestos used in industry

Second form of asbestos:

b. Amphibole, in which the fiber is straight, stiff

- Amphiboles, are less prevalent but more pathogenic than the serpentine and align themselves in the airstream and are delivered deeper into the lungs where they may penetrate epithelial cells to reach the interstitium

- Asbestos also functions as both a tumor initiator and a promoter
- Some of the oncogenic effects of asbestos on the mesothelium are mediated by reactive free radicals generated by asbestos fibers

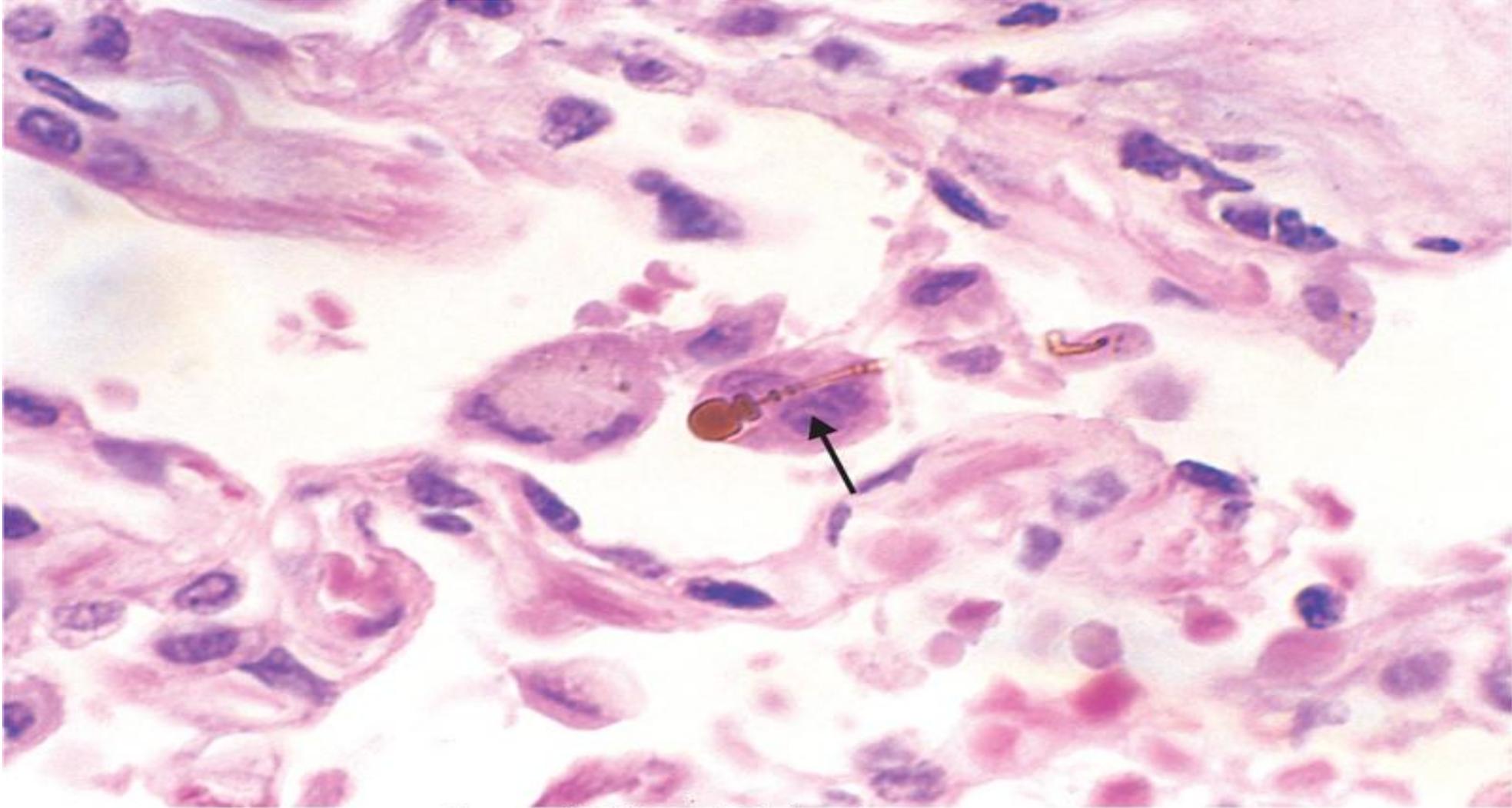
- MORPHOLOGY

1. Asbestosis is marked by diffuse pulmonary interstitial fibrosis which is indistinguishable from UIP, except for the presence of asbestos bodies,

Asbestos bodies

- = golden brown, fusiform or beaded rods with a translucent center and consists of asbestos fibers coated with an iron-containing material are formed when macrophages attempt to phagocytose asbestos fibers; the iron is derived from phagocyte ferritin.

Asbestos body



- Asbestos bodies sometimes can be found in the lungs of normal persons, but usually in much lower concentrations and **without** an accompanying interstitial fibrosis.

- Note
- - In contrast with CWP and silicosis, **asbestosis begins in the lower lobes and subpleurally**

Morphology 2. Pleural plaques :

- Are the most common manifestation of asbestos exposure
- Are well-circumscribed plaques of dense collagen , often containing calcium.



- Pleural plaques develop most frequently on the anterior and posterolateral aspects of the parietal pleura and do not contain asbestos bodies, and rarely do they occur in persons with no history or evidence of asbestos exposure.

NOTE: Uncommonly, asbestos exposure induces pleural effusion or diffuse pleural fibrosis

Note

- Both lung carcinoma and malignant mesothelioma (pleural and peritoneal) develop in workers exposed to asbestos.
- The risk of lung carcinoma is increased about five-fold for asbestos workers; and the relative risk for mesothelioma, normally a very rare tumor (2 to 17 cases per 1 million persons), is more than 1000 times greater.

Important note

- Concomitant cigarette smoking greatly increases the risk of lung carcinoma but not that of mesothelioma .

Peritoneal Mesothelioma

- 2nd most common site of mesothelioma is the peritoneum
 - 10-30% of cases of mesothelioma
 - 300-400/cases in U.S. year

