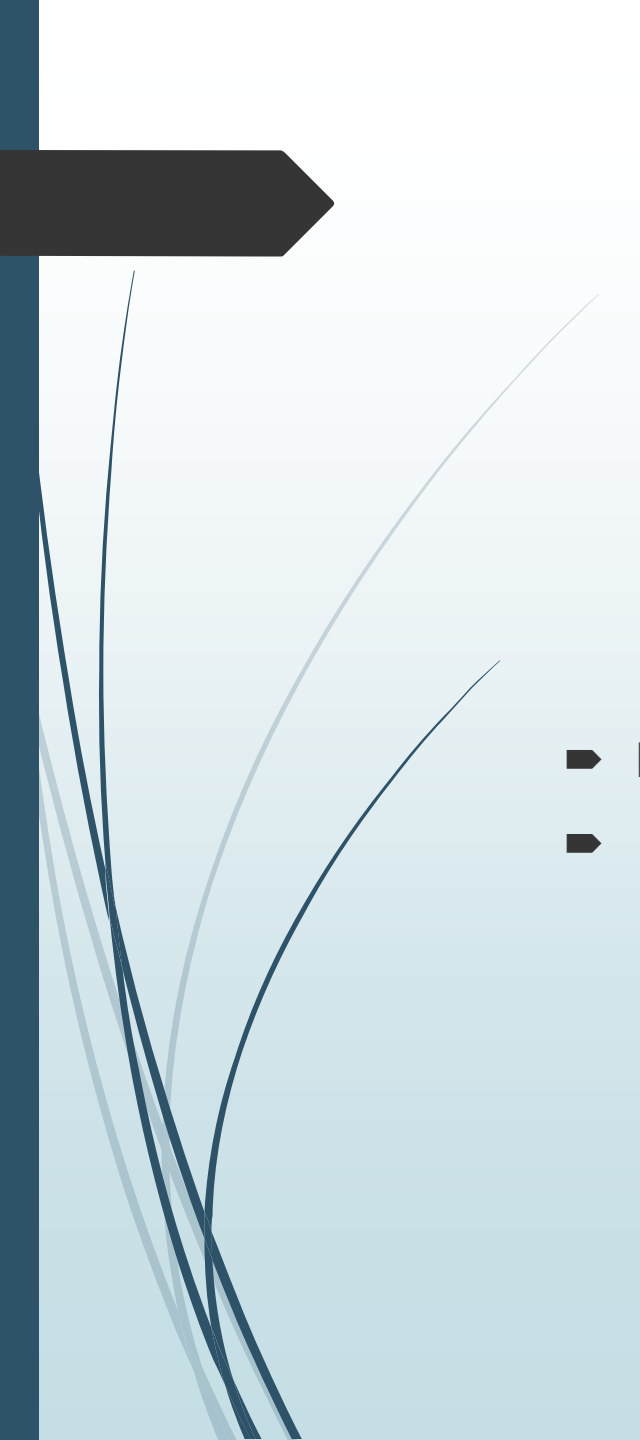


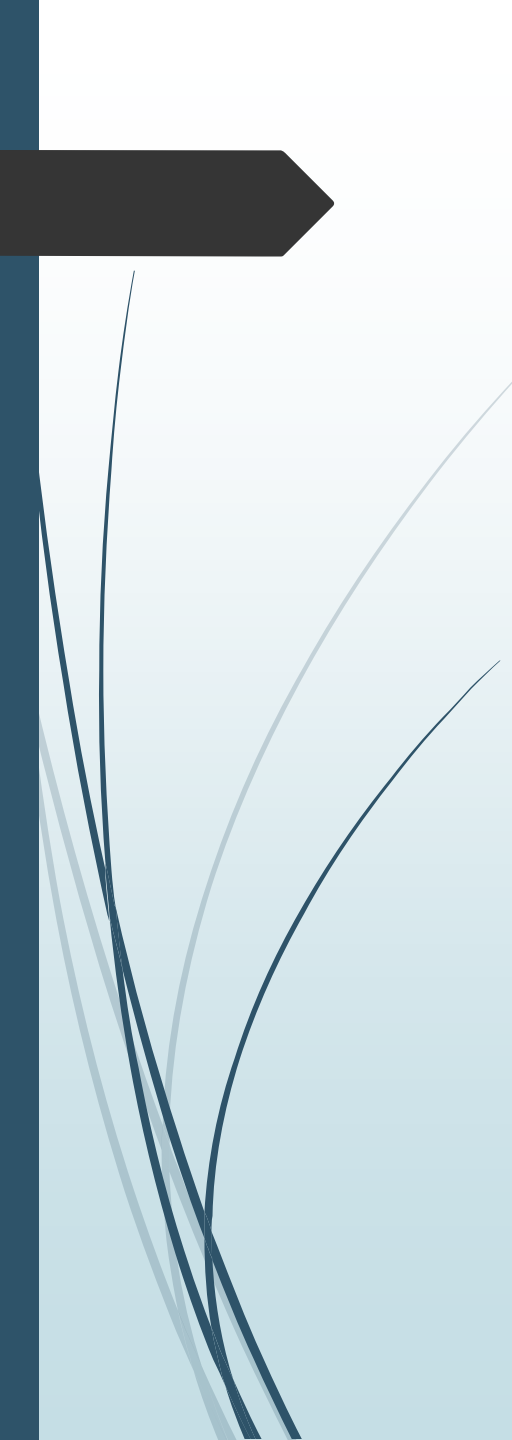
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OCCUPATIONAL EXPOSURES & PULMONARY DISEASE

- Dr. Noora Haj Jafari Nezhad
- pulmonologist





Why is knowledge of occupational or environmental etiology so important?

- **management and prognosis**

For example, patients with **occupational asthma or hypersensitivity pneumonitis** often cannot be managed adequately without cessation of exposure to the offending agent

- **a large number of individuals are at risk**

For example, **15–20% of the burden of adult asthma and chronic obstructive pulmonary disease (COPD)** has been estimated to be due to occupational factors.

- **OCCUPATIONAL RESPIRATORY CARCINOGENS**

Exposures at work have been estimated to contribute to **10% of all lung cancer cases**

>80% of mesotheliomas may be associated with asbestos exposure.

- **1) OCCUPATIONAL EXPOSURES**

- **Inorganic Dusts** : Asbestos, Silica, Coal dust, Beryllium, Other metals...

- **Organic Dusts**: Cotton dust, Grain dust, Other agricultural dusts(fungal spores, vegetable products, insect fragments, animal dander, bird and rodent feces, endotoxins, microorganisms, pollens)

- **2) Common Toxic Chemical Agents** That Affect the Lung

- Nitrogen dioxide , Ozone, Phosgene, Sulfur dioxide

- **3) OUTDOOR AIR POLLUTION**

- **4) INDOOR EXPOSURES**

- **5) OCCUPATIONAL RESPIRATORY CARCINOGENS**

HISTORY AND EXPOSURE ASSESSMENT

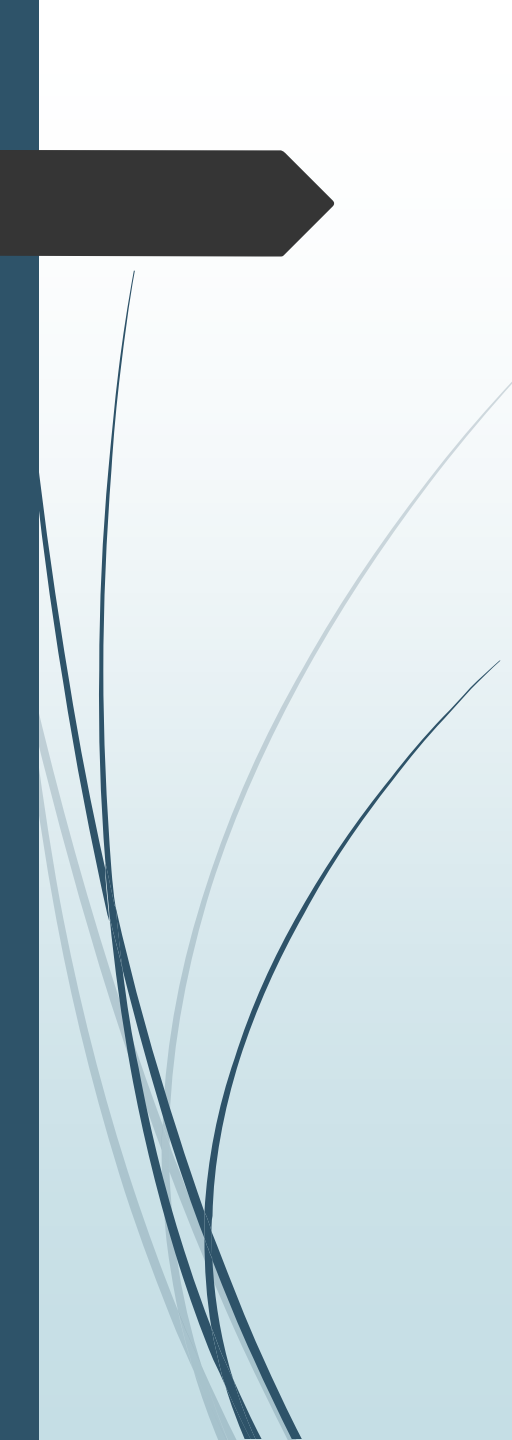
- careful **exposure history**
- questions about the specific contaminants involved, the presence of visible dust, chemical odors
- the use of **respiratory protective equipment**
- **Short-term** and **long-term** exposures





LABORATORY TESTS

- **interstitial lung disease & airway obstruction**
- Measurement of change in forced expiratory volume in 1 second (**FEV1**) before and after a working shift can be used to detect an acute bronchoconstrictive response
- conventional computed tomography (**CT**) is more sensitive for the detection of lung opacities and pleural thickening, and high-resolution CT (HRCT) improves the detection of interstitial changes
- **prick testing** or specific immunoglobulin type E (**IgE**) antibody titers
- bronchoscopy to obtain transbronchial **biopsies of lung tissue**



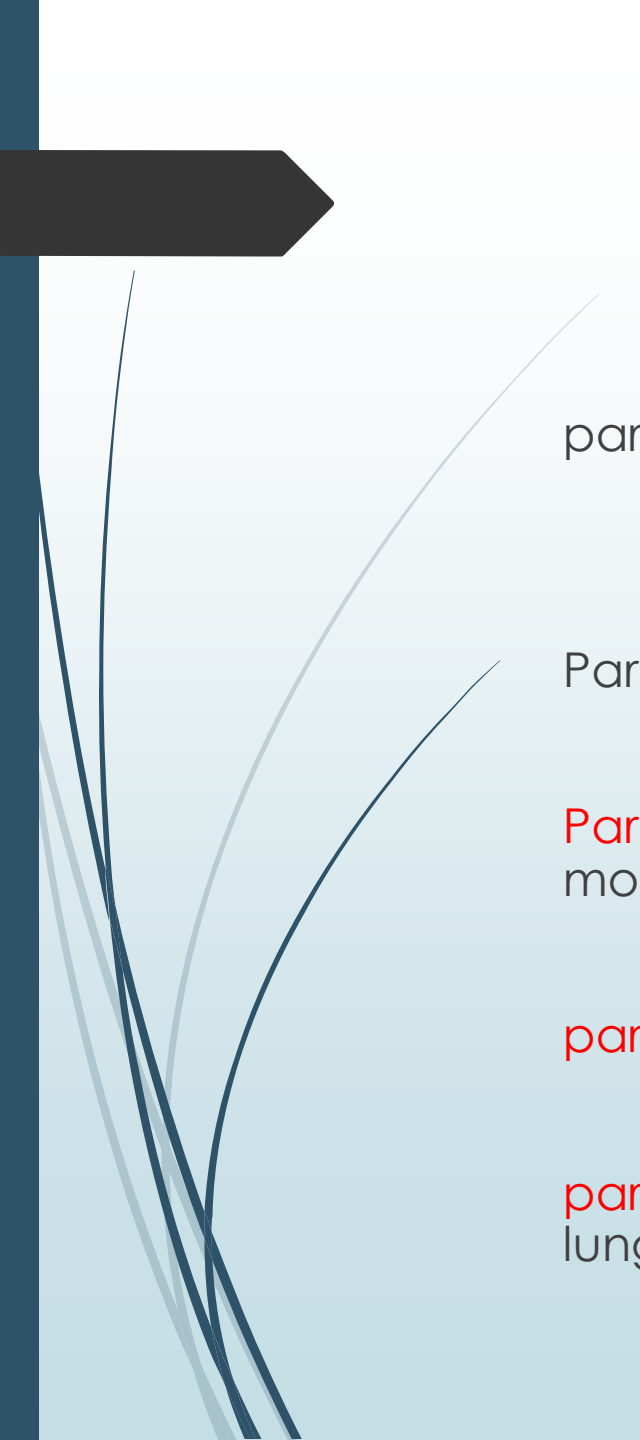
The chemical and physical characteristics of inhaled agents

- Water-soluble gases

ammonia and sulfur dioxide are absorbed in the lining fluid of the upper and proximal airways and thus tend to **produce irritative** responses

- which are less soluble

nitrogen dioxide and phosgene penetrate to the bronchioles and alveoli in sufficient quantities to produce **acute chemical pneumonitis**.



Particle size

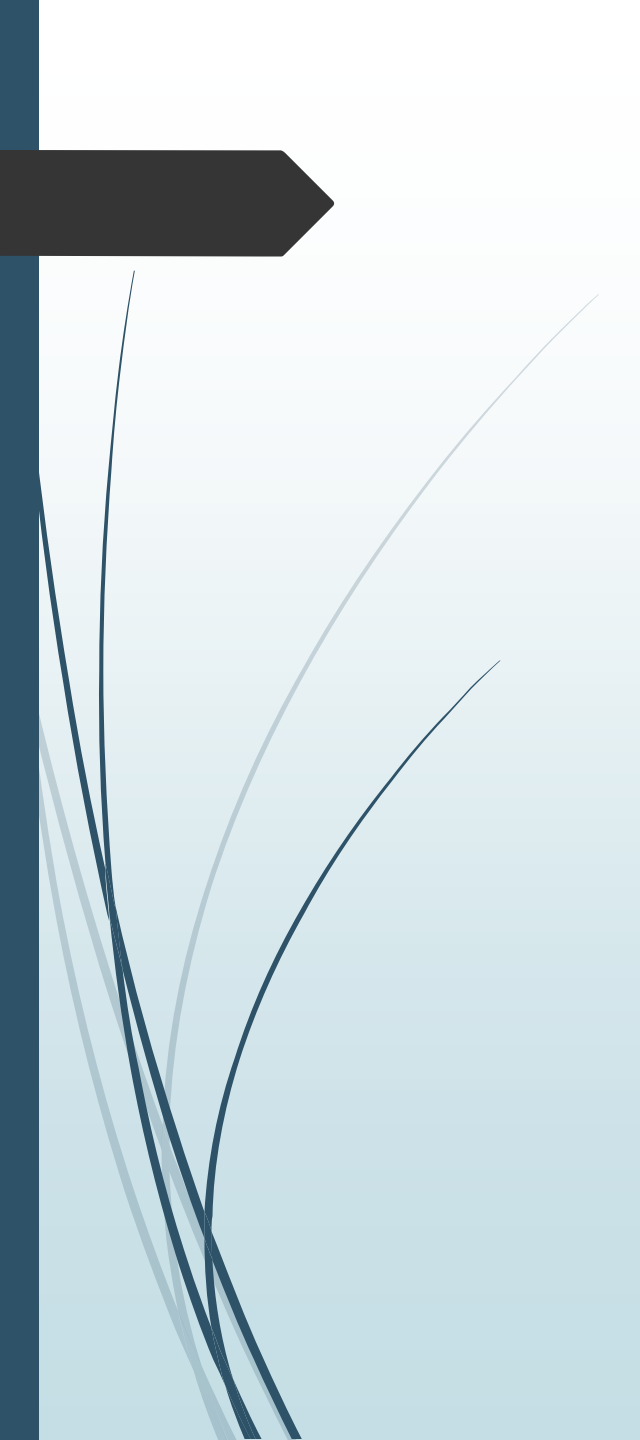
particles $>10\text{--}15\text{ }\mu\text{m}$ (nose and throat)

Particles $<10\text{ }\mu\text{m}$ in size are deposited below the larynx:

Particles $\sim 2.5\text{--}10\text{ }\mu\text{m}$ (coarse-mode fraction) such as silica, aluminum, and iron. mostly deposit relatively high **in the tracheobronchial tree**

particles $< 2\text{ }\mu\text{m}$ (fine-mode fraction) products from gases, fumes, or vapors

particles $< 0.1\text{ }\mu\text{m}$ (ultrafine fraction) remain **in the airstream**, deposit in the lung only on a random basis as they come into contact with the alveolar walls.



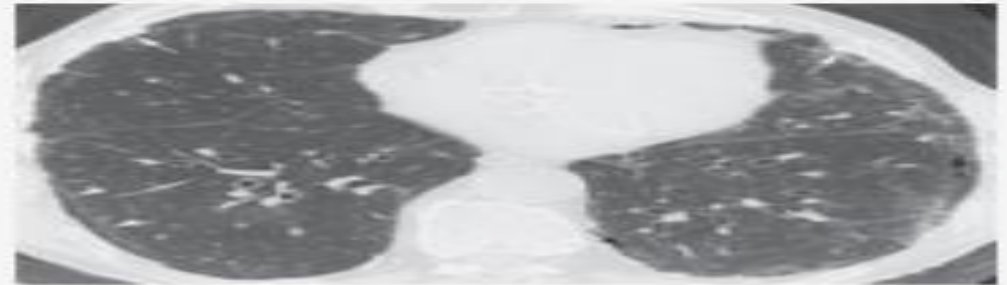
INORGANIC DUSTS

* ASBESTOS-RELATED DISEASES

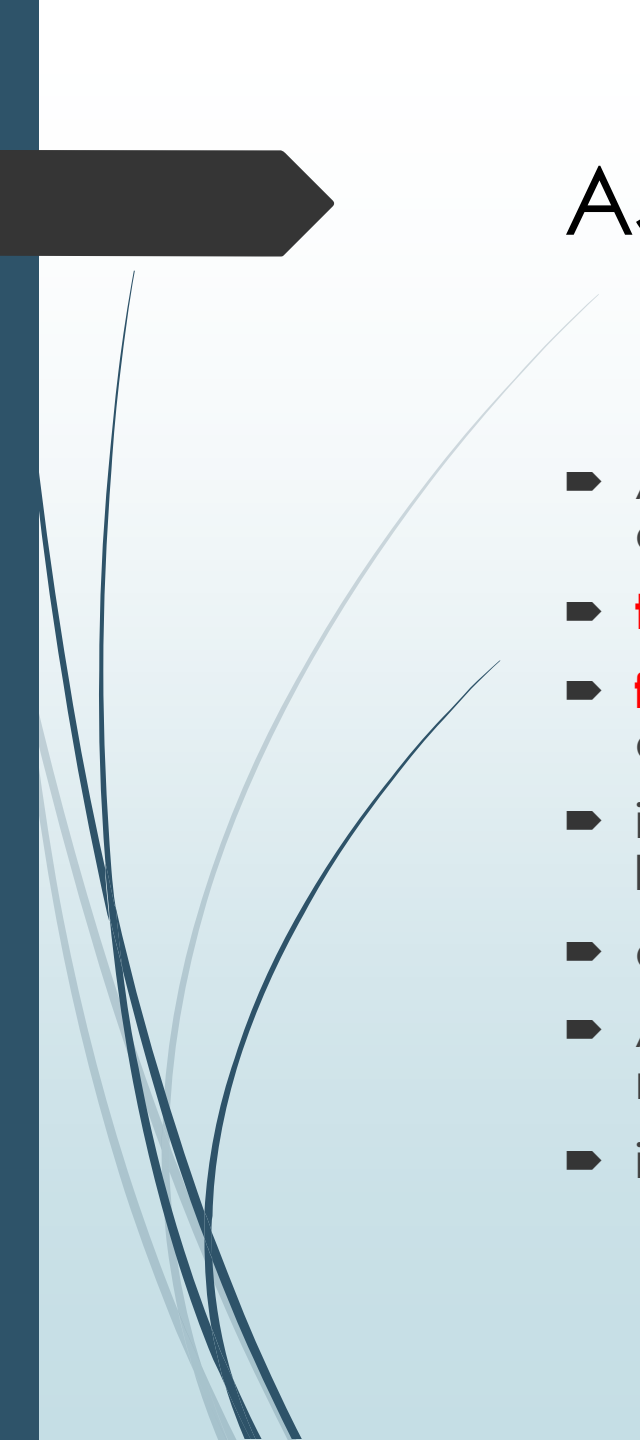
- ▶ pleural and pulmonary **fibrosis**
- ▶ **cancers** of the respiratory tract
- ▶ pleural and peritoneal **mesothelioma**.



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ASBESTOS-RELATED DISEASES

- Asbestos: a generic term for several different mineral silicates(chrysolite, amosite, anthophyllite, and crocidolite)
- **thermal** and **electrical** insulation properties
- **fire-resistant textiles**, in **cement and floor tiles**, and in friction materials such as **brake and clutch** linings
- intensity and duration of exposure: Usually, exposure has taken place for **at least 10 years before the disease** becomes manifest
- directly handle, bystander exposure,
- Asbestos has largely been replaced in the **developed world** with synthetic mineral fibers
- in the **developing world** ...



Asbestos exposure

- Past exposure to asbestos: **pleural plaques** on chest radiographs,
- thickening or calcification along the parietal pleura, lower lung fields, the diaphragm, and the cardiac border.
- Without additional manifestations, pleural plaques imply only exposure, **not pulmonary impairment**.
- **Benign pleural effusions** also may occur.





Asbestosis

- **Irregular or linear opacities** that usually are first noted in the lower lung fields are the chest radiographic hallmark of asbestosis.
- HRCT : **subpleural curvilinear lines 5–10 mm in length** that appear to be parallel to the pleural surface
- 1- **Restrictive** pattern with a decrease in both lung volumes
- 2- **Lung cancer** (15–19 years between first exposure, smoking)
- 3- **Mesothelioma** of both pleural and peritoneal (>80%)



** SILICOSIS

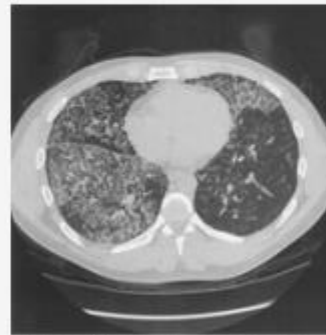
- **oldest** known occupational pulmonary hazards
- **free silica (SiO₂), or crystalline quartz**
- The major occupational exposures include **mining; stonecutting**; sand blasting; **glass and cement** manufacturing; foundry work; packing of silica flour; and quarrying, particularly of granite
- **Acute, Accelerated, Chronic**

Acute silicosis

- ▶ chest radiograph : profuse miliary infiltration or consolidation
- ▶ HRCT pattern known as “**crazy paving**”
- ▶ pulmonary alveolar proteinosis

FIGURE 300-2

Acute silicosis. This high-resolution computed tomography scan shows multiple small nodules consistent with silicosis but also diffuse ground-glass densities with thickened intralobular and interlobular septa producing polygonal shapes. This has been referred to as "crazy paving."

[illegible]

Chronic

► 1- simple silicosis:

less intense exposure, small rounded opacities in the upper lobes may appear on the chest radiograph after 15– 20 years of exposure, without associated impairment of lung function

Calcification of hilar, 20% of cases, “eggshell” pattern

► 2- complicated silicosis:

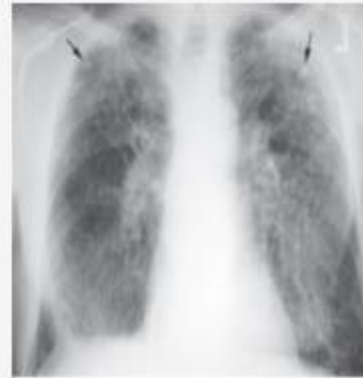
nodular fibrosis may be progressive in the absence of further exposure, with coalescence and formation of nonsegmental conglomerates of irregular masses >1 cm in diameter

► 3- PMF:

Significant functional impairment with both restrictive and obstructive components may be associated with PMF.

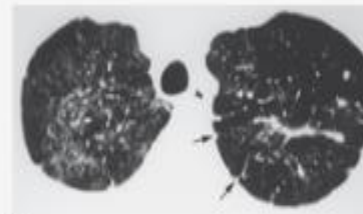
FIGURE 300-3

Chronic silicosis. **A.** Frontal chest radiograph in a patient with silicosis shows variably sized, poorly defined nodules (*arrows*) predominating in the upper lobes. **B.** Axial thoracic computed tomography image through the lung apices shows numerous small nodules, more pronounced in the right upper lobe. A number of the nodules are subpleural in location (*arrows*).



A

Source: Longo D, Fauci A, Kasper D, Hauser S, Jameson JL, Loscalzo J, Hazzard S, Longford C. Harrison's Principles of Internal Medicine, 22nd Edition. Copyright © McGraw-Hill. All rights reserved.



B

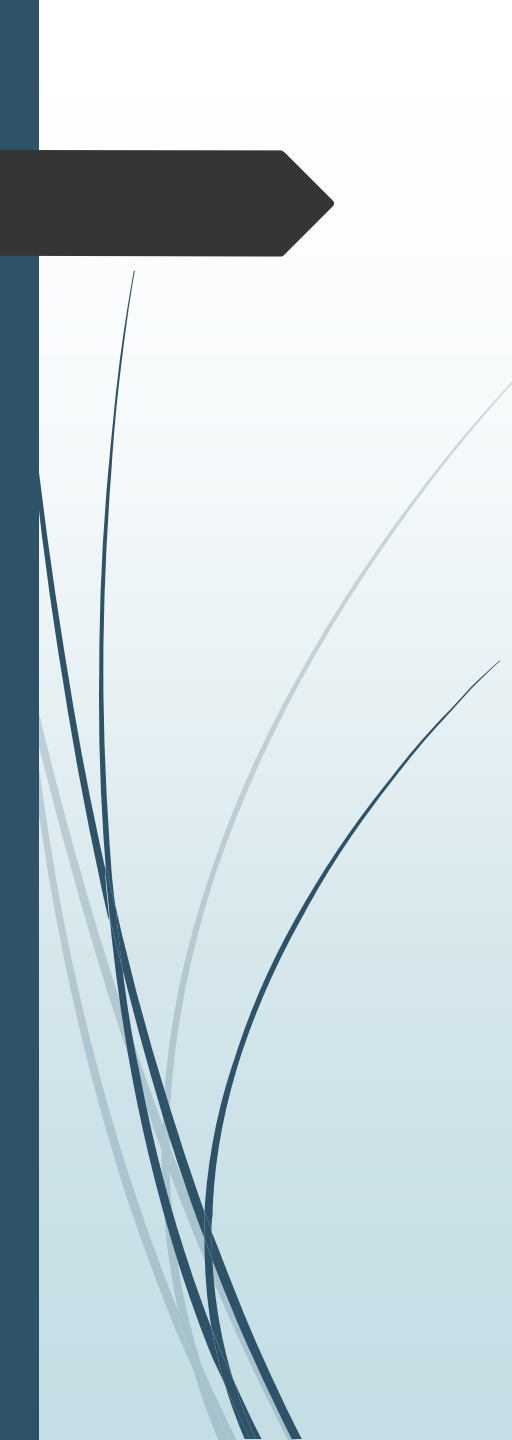
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- ▀ **alveolar macrophage dysfunction**

(Mycobacterium tuberculosis, atypical mycobacteria, and fungi)

treatment of latent tuberculosis in these patients is longer

- ▀ silica as a probable **lung carcinogen**
- ▀ another potential clinical complication of silicosis: **CTD** (RA, SSC)



*** COAL WORKER'S PNEUMOCONIOSIS (CWP)

- exposure to coal dust can lead to CWP

- # Simple radiographically identified CWP is seen **in ~10%** of all coal miners

- # in as many as **50%** of anthracite miners with >20 years of work on the coal face.

- The prevalence of disease is lower in workers in bituminous coal mines.

► **simple CWP:**

With prolonged exposure to coal dust (i.e., 15–20 years), small, rounded opacities **similar to those of silicosis** may develop

► **Chronic bronchitis and COPD:**

In addition to CWP, coal dust can cause COPD. The effects of coal dust are **additive to those of cigarette smoking**.

► **Complicated CWP:**

≥1 cm in diameter , upper half of the lungs

► **can progress to PMF:**

accompanied by severe lung function deficits and associated with premature mortality.

► **Caplan syndrome:**

first described in coal miners but subsequently in patients with silicosis

the combination of **pneumoconiotic nodules** and **seropositive RA**

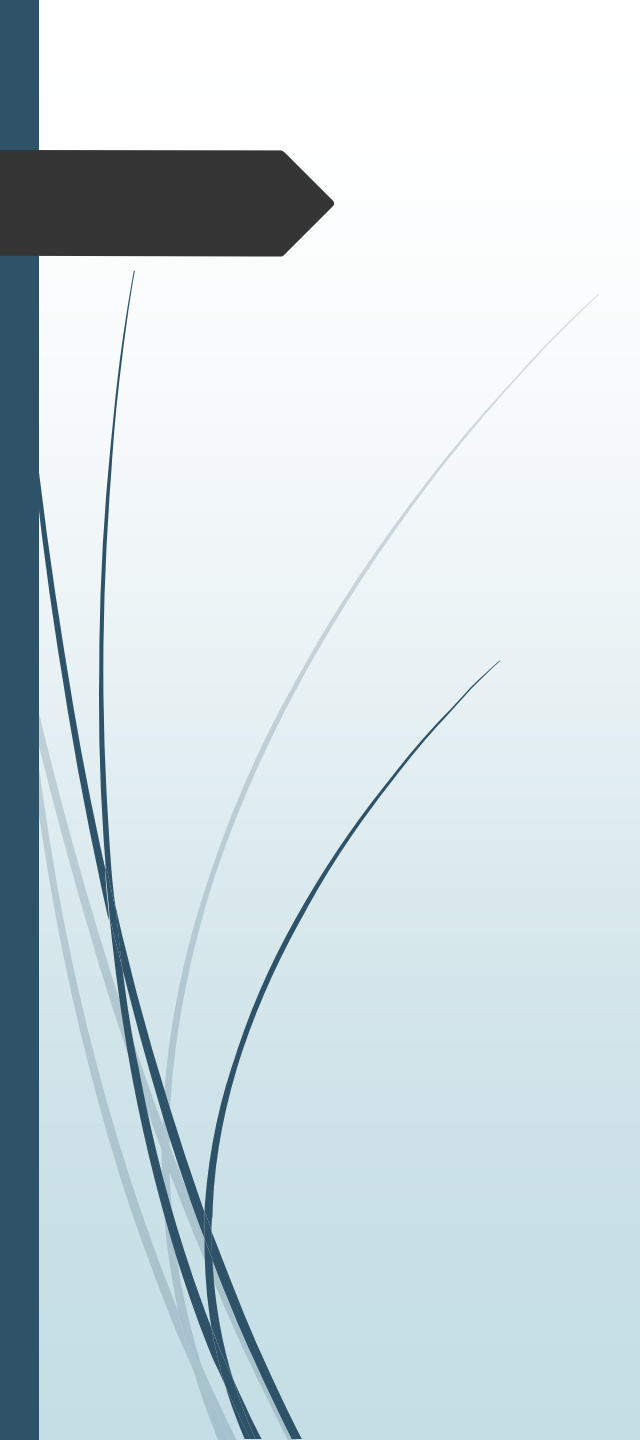
**** CHRONIC BERYLLIUM DISEASE

- **lightweight metal** , good electrical conductivity, and value in the control of nuclear reactions
- manufacture of **alloys**, ceramics, or **high-technology electronics**
- Combat-related embedded metal fragments (shrapnel) in military veterans may also contain beryllium
- Chest imaging findings are similar to those of **sarcoidosis** (nodules along septal lines) except that hilar adenopathy is somewhat less common
- **granulomatous inflammation** seen on lung biopsy
- **BeLPT**: proliferation of lymphocytes from blood or bronchoalveolar lavage in the presence of beryllium salts

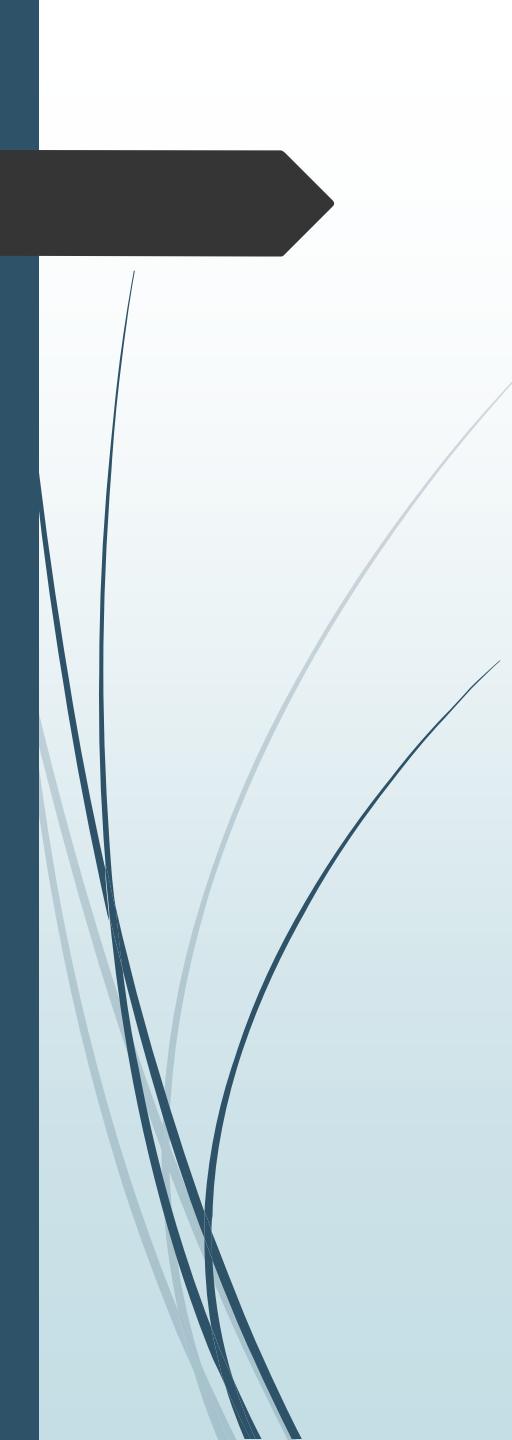


***** OTHER METALS

- **Aluminum and titanium dioxide** have been rarely associated with a sarcoid-like reaction in lung tissue
- dust containing **tungsten** carbide, also known as “hard metal,” may produce giant cell interstitial pneumonitis
- **Cobalt** is a constituent of tungsten carbide and is the likely etiologic agent of both the interstitial pneumonitis and the occupational asthma that may occur



ORGANIC DUSTS



ORGANIC DUSTS

- asthma and hypersensitivity pneumonitis
- farmer's lung, malt worker's disease, and mushroom worker's disease

- Cotton Dust (Byssinosis)
- Grain Dust
- Farmer's Lung



* Cotton Dust (Byssinosis)

- cotton dust (but also to flax, hemp, or jute dust)
- an **asthma-like syndrome** known as byssinosis
- “**Monday chest tightness**”
- Initially the symptoms do not recur on subsequent days of the week, but in a subset of workers, chest tightness may recur or persist throughout the workweek.
- **spirometry before and after the work shift**
- After >10 years of exposure, workers with recurrent symptoms are more likely to have an obstructive pattern on pulmonary function testing.
- All workers with **persistent symptoms or significantly reduced levels of pulmonary function** should be moved to areas of lower risk of exposure.



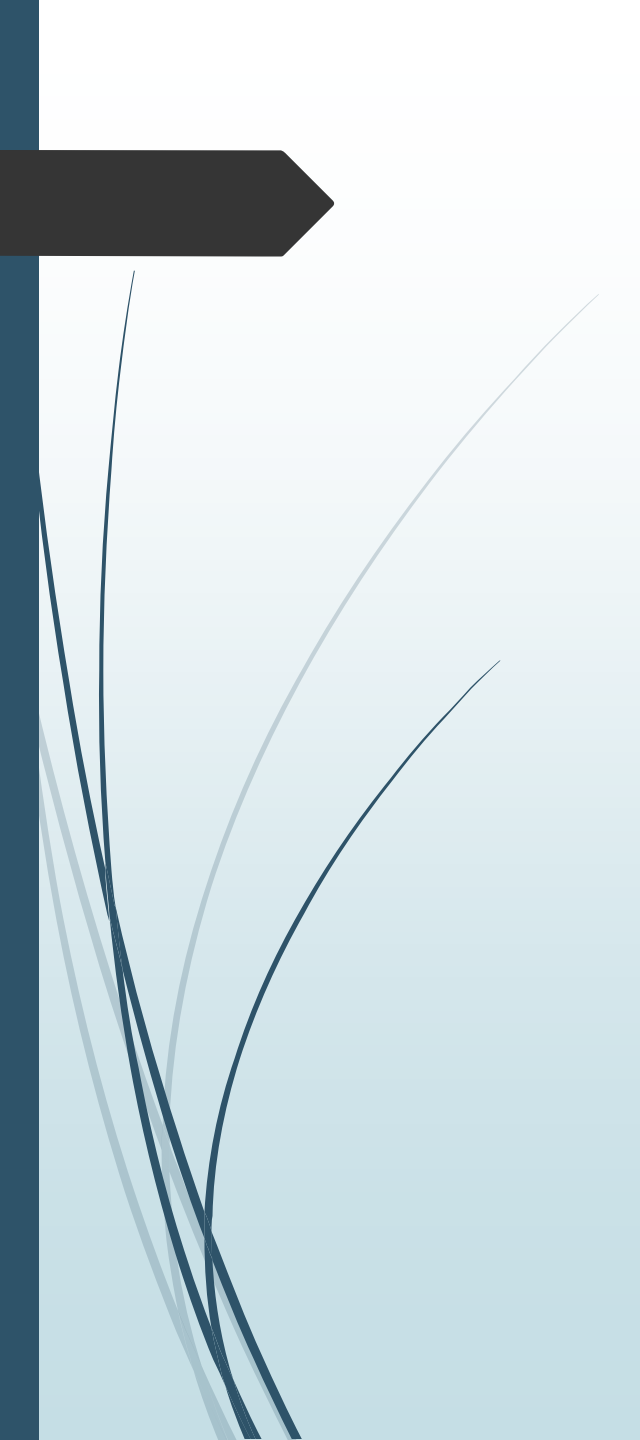
** Grain Dust

- The presentation of **obstructive airway disease** in grain dust-exposed workers is virtually identical to the characteristic findings in cigarette smokers, i.e., **persistent cough, mucus hypersecretion, wheeze and dyspnea on exertion, and reduced FEV1 and FEV1 /FVC** (forced vital capacity) ratio.
- The effect of grain dust exposure is **additive to that of cigarette smoking**
- ~50% of workers who smoke having symptoms



*** Farmer's Lung

- This condition results from exposure to **moldy hay containing spores of thermophilic actinomyces** that produce a hypersensitivity pneumonitis.
- **4–8 h after exposure with fever, chills, malaise, cough, and dyspnea without wheezing**
- influenza or pneumonia with similar symptoms
- **chronic form of the disease, the history of repeated attacks** after similar exposure is important in differentiating this syndrome from other causes of patchy fibrosis (e.g., sarcoidosis).



OUTDOOR AIR POLLUTION

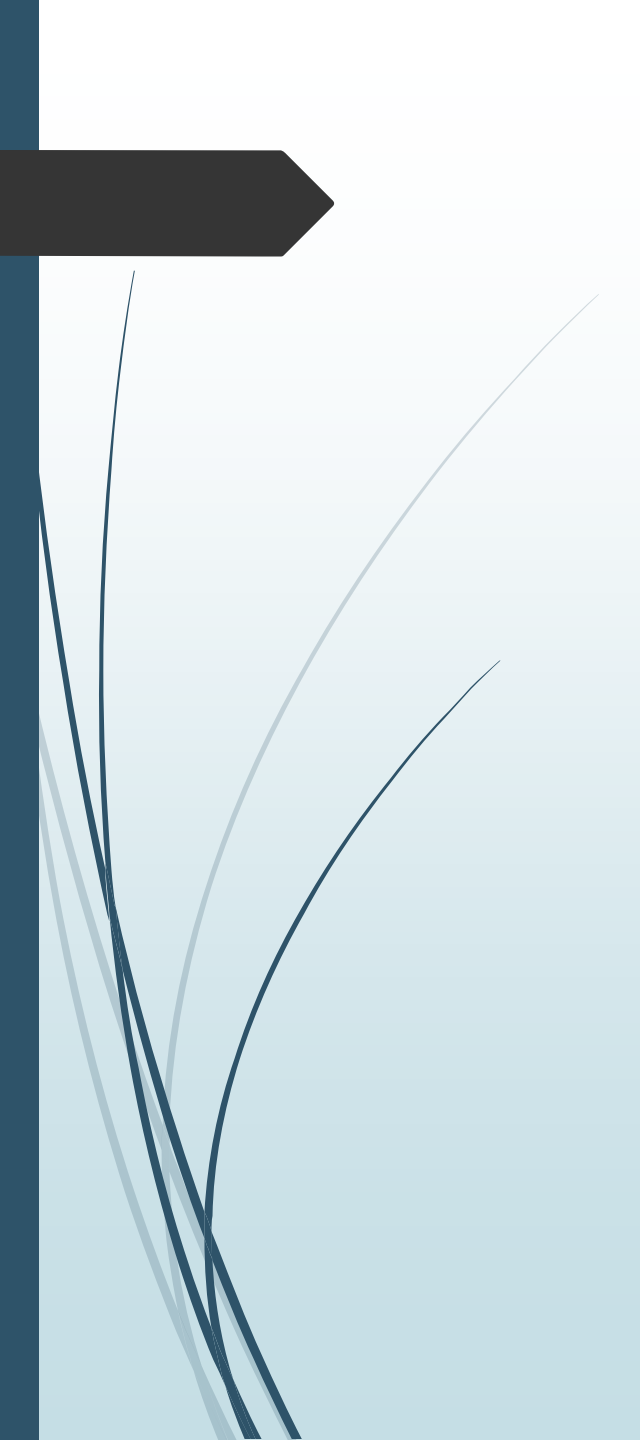
- 
- stationary sources (power plants and industrial facilities)
 - mobile sources (motor vehicles),
 - sulfur dioxide (PM)
 - nitrogen dioxide
 - Ozone
 - lead
 - carbon monoxide.

Selected Common Toxic Chemical Agents That Affect the Lung

AGENT(s)	SELECTED EXPOSURES	ACUTE EFFECTS FROM HIGH OR ACCIDENTAL EXPOSURE	CHRONIC EFFECTS FROM RELATIVELY LOW EXPOSURE
Acid anhydrides	Manufacture of resin esters, polyester resins, thermoactivated adhesives	Nasal irritation, cough	Asthma, chronic bronchitis, hypersensitivity pneumonitis
Acid fumes: H_2SO_4 , HNO_3	Manufacture of fertilizers, chlorinated organic compounds, dyes, explosives, rubber products, metal etching, plastics	Mucous membrane irritation, followed by chemical pneumonitis 2–3 days later	Bronchitis and suggestion of mildly reduced pulmonary function in children with lifelong residential exposure to high levels
Acrolein and other aldehydes	By-product of burning plastics, woods, tobacco smoke	Mucous membrane irritant, decrease in lung function	Upper respiratory tract irritation
Ammonia	Refrigeration; petroleum refining; manufacture of fertilizers, explosives, plastics, and other chemicals	Same as for acid fumes, but bronchiectasis also has been reported	Upper respiratory tract irritation, chronic bronchitis
Cadmium fumes	Smelting, soldering, battery production	Mucous membrane irritant, acute respiratory distress syndrome (ARDS)	Chronic obstructive pulmonary disease (COPD)
Formaldehyde	Manufacture of resins, leathers, rubber, metals, and woods; laboratory workers, embalmers; emission from urethane foam insulation	Same as for acid fumes	Nasopharyngeal cancer
Halides and acid salts (Cl, Br, F)	Bleaching in pulp, paper, textile industry; manufacture of chemical compounds; synthetic rubber, plastics, disinfectant, rocket fuel, gasoline	Mucous membrane irritation, pulmonary edema; possible reduced forced vital capacity (FVC) 1–2 years after exposure	Upper respiratory tract irritation, epistaxis, tracheobronchitis

Hydrogen sulfide	By-product of many industrial processes, oil, other petroleum processes and storage	Increase in respiratory rate followed by respiratory arrest, lactic acidosis, pulmonary edema, death	Conjunctival irritation, chronic bronchitis, recurrent pneumonitis
Isocyanates (TDI, HDI, MDI)	Production of polyurethane foams, plastics, adhesives, surface coatings	Mucous membrane irritation, dyspnea, cough, wheeze, pulmonary edema	Upper respiratory tract irritation, cough, asthma, hypersensitivity pneumonitis, reduced lung function
Nitrogen dioxide	Silage, metal etching, explosives, rocket fuels, welding, by-product of burning fossil fuels	Cough, dyspnea, pulmonary edema may be delayed 4–12 h; possible result from acute exposure: bronchiolitis obliterans in 2–6 weeks	Emphysema in animals, chronic bronchitis, associated with reduced lung function growth in children with lifelong residential exposure
Ozone	Arc welding, flour bleaching, deodorizing, emissions from copying equipment, photochemical air pollutant	Mucous membrane irritant, reduced pulmonary function transiently in children and adults, asthma exacerbation	Excess cardiopulmonary mortality rates, increased risk for new-onset asthma in children

AGENT(s)	SELECTED EXPOSURES	ACUTE EFFECTS FROM HIGH OR ACCIDENTAL EXPOSURE	CHRONIC EFFECTS FROM RELATIVELY LOW EXPOSURE
Phosgene	Organic compound, metallurgy, volatilization of chlorine-containing compounds	Delayed onset of bronchiolitis and pulmonary edema	Chronic bronchitis
Sulfur dioxide	Manufacture of sulfuric acid, bleaches, coating of nonferrous metals, food processing, refrigerant, burning of fossil fuels, wood pulp industry	Mucous membrane irritant, epistaxis, bronchospasm (especially in people with asthma)	Chronic bronchitis

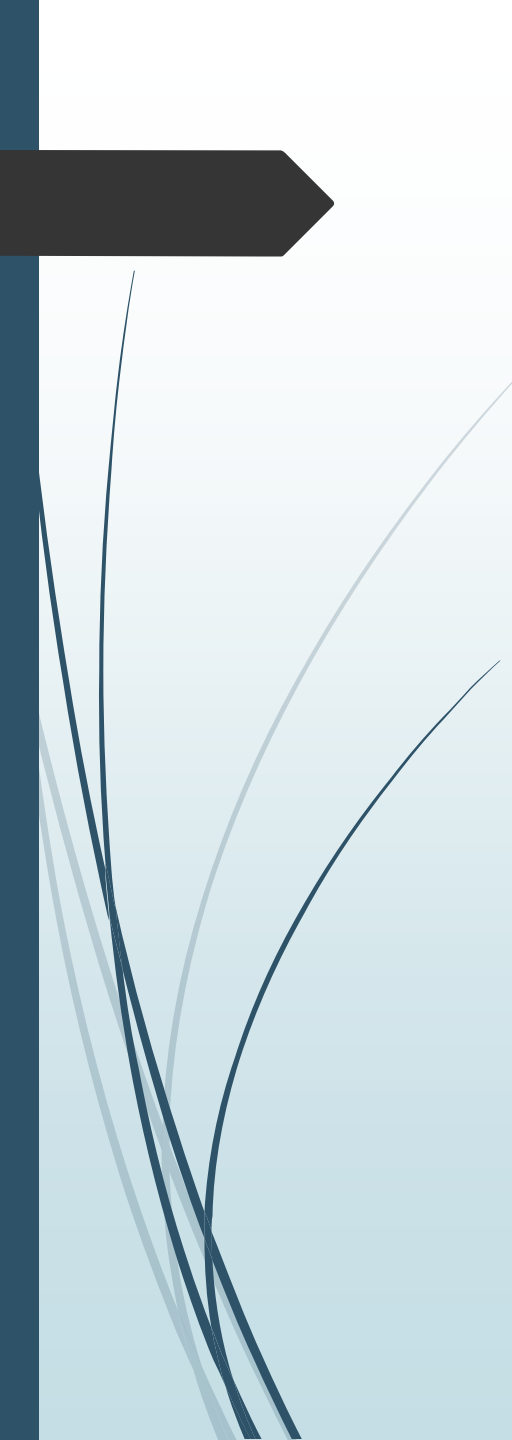


INDOOR EXPOSURES

- 
- **Secondhand tobacco smoke** , radon gas, wood smoke, and other biologic agents generated indoors must be considered.
 - respiratory illnesses, **especially asthma**, and reduced levels of pulmonary function have been found **in the children of smoking parents** in a number of studies.
 - **~25% increase in relative risk for lung cancer and cardiopulmonary diseases**, in secondhand tobacco smoke



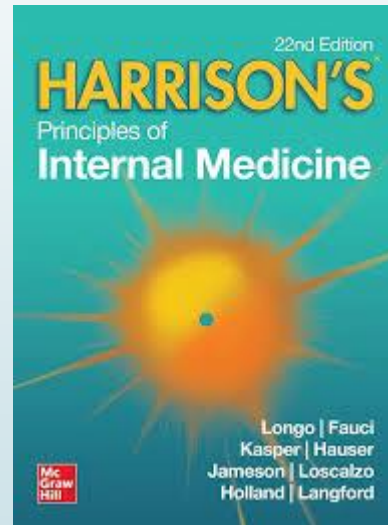
OCCUPATIONAL RESPIRATORY CARCINOGENS

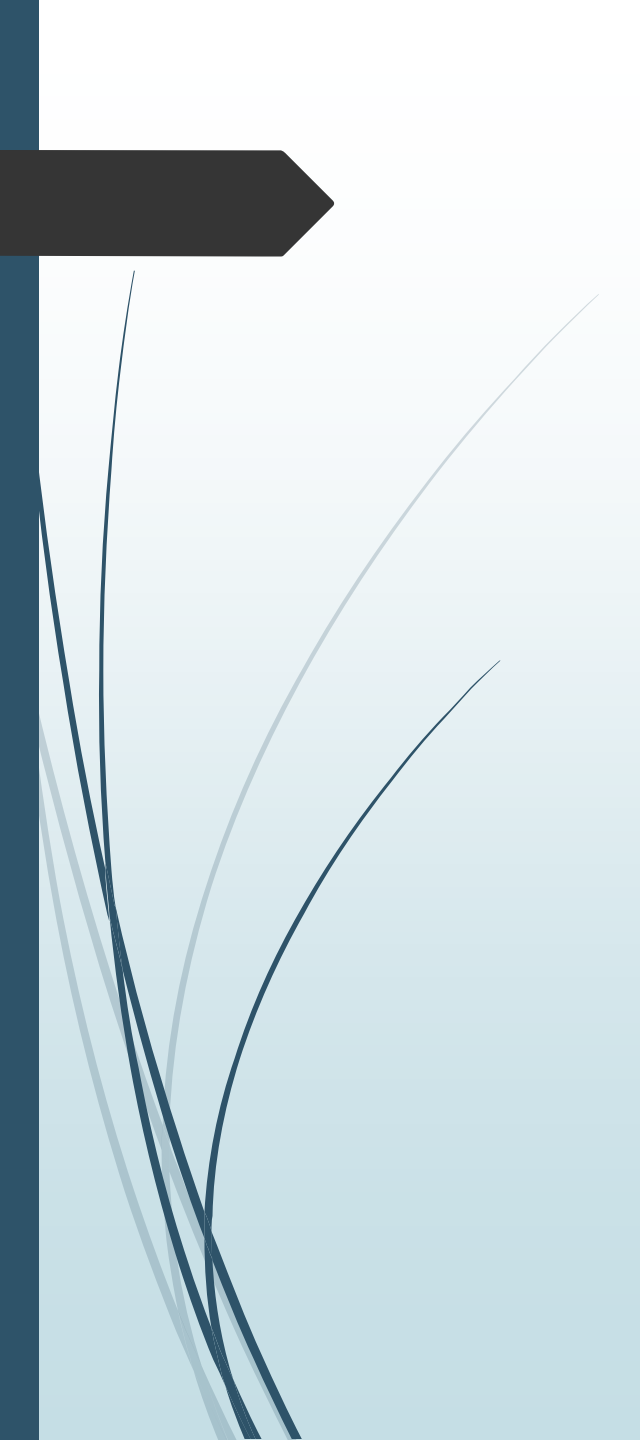


OCCUPATIONAL RESPIRATORY CARCINOGENS

- Exposures at work have been estimated to contribute to **10% of all lung cancer cases**
- **Asbestos**
- acrylonitrile, arsenic compounds, **beryllium**, bis(chloromethyl) ether, chromium (hexavalent), formaldehyde (nasal), isopropanol (nasal sinuses), mustard gas, nickel carbonyl (nickel smelting), polycyclic aromatic hydrocarbons (coke oven emissions and diesel exhaust), secondhand tobacco smoke, **silica** (both mining and processing), talc (possible asbestos contamination in both mining and milling), vinyl chloride (sarcomas), wood (nasal), and uranium

REFERENCE





*WITH
THANKS*