Occupational Pulmonary Disease: an Overview

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Conflicts of Interest/Disclosures
J.L. Lultschik, MD, MPH

• Board certified in Occupational Medicine
• Active clinical practice in Occupational Medicine, file reviews, and IMEs
• Consult on work-relatedness of COPD and other pulmonary diagnoses
• The opinions expressed in this presentation are my own and do not represent the opinions of WVU, WVU Medicine, or the WVU School of Public Health
• No conflicts of interest to disclose
Outline

• A Brief History of Occupational Pulmonary Disease
• The Occupational Pulmonary Exposure History
• Classification of Occupational Pulmonary Diseases
• Obstructive, Interstitial, Other
• A Few Caveats re Causation
• New Issues in Occupational Pulmonary Disease
Occupational lung disease

• Pulmonary disease due to an occupational exposure
• Exposure route is usually inhalational, but not always
• Temporal relationship to work exposure
• Often a dose-response relationship, but not always
• Removal from exposure usually helps – but not always
Occupational lung disease: from antiquity to the 18th Century

Pliny the Elder (Natural History XXXIII, 40):

- “Persons employed in the manufactories in preparing minium (red lead) protect the face with masks of loose bladder-skin, in order to avoid inhaling the dust, which is largely pernicious…”
- Noted that slaves working with asbestos became ill

Bernardo Ramazzini: De Morbis Artificum Diatriba (Diseases of Workers, 1700)

- Those who “shovel, smelt, cast, and refine the material that has been mined … are liable to the same diseases (as underground miners) … in the course of time the metallic fumes that they breathe make them short-winded … and in the end they pass into the class of consumptives.”
Occupational Lung Disease: Industrial Revolution to the present

1750–1900
- Deep mining in silica deposits
- Coal mining
- Cotton dust exposure

1900 – present
- Acute silicosis (Hawk’s Nest Tunnel, 1930-1935)
- Lung cancer in uranium miners
- Asbestos
Assessment of Possible Occupational Exposure

- Chronological list of all jobs
- Detailed list of exposures
- Actual job tasks
- Length of time spent on tasks
- Coworkers with same exposures and similar symptoms or disease?
The occupational history

WHACO:

- *What* do you do?
- *How* do you do it?
- *Acute* or chronic symptoms?
- *Coworkers* or family members, friends sick with the same illness?
- *Outside* of work: exposures via hobbies, pets, travel, unpaid work?

Complete chronological list of employment with past exposures

Hours of work per week; hours spent in specific tasks
Classification of occupational pulmonary disease

Obstructive Lung Disease
- Work-related Asthma; Work-exacerbated Asthma
- COPD in non-smokers
- Acute and Subacute Inhalation Injuries

Interstitial Lung Disease
- Nonspecific inflammatory response (Inorganic dusts)
- Immune sensitization (Organic dusts, Beryllium)
Classification of occupational pulmonary disease (continued)

- Hypersensitivity Pneumonitis (allergic extrinsic alveolitis)
- Occupational Infectious Pulmonary Disease
- Occupational Lung Disease related to High Altitude Work and Diving
- Occupational Lung Cancer
- Emerging Occupational Lung Diseases
Lung Anatomy

- Upper respiratory tract
- Lower respiratory tract
- Bronchi
- Bronchioles
- Alveoli
General approaches to management

- Primary prevention – avoid/minimize exposure
- Secondary prevention – workplace surveillance
- Investigations
- Accurate diagnosis
- Work-relatedness and Causation
- Medical management
Occupational obstructive lung diseases
Occupational Obstructive Lung Diseases

Asthma
- Occupational asthma
- Sensitizer-induced
- Irritant-induced
- Work-exacerbated asthma

COPD

Bronchiolitis obliterans (diacetyl, flavorings)
Asthma

- Reversible airway obstruction
- Reaction to irritants
- Immune reaction to allergen
Work-related Asthma

- **Occupational (work-related) asthma** – caused by exposure in the workplace
  - Irritant-induced
  - Immune sensitization

- **Work-exacerbated asthma** – pre-existing asthma exacerbated by an exposure in the workplace

- **RADS** – acute airway hyperreactivity secondary to an acute inhalation injury

- **Low-dose RADS** – ??? subacute airway hyperreactivity secondary to longer, lower-dose exposures to irritants
**Occupational asthma (OA): new onset adult asthma caused by workplace exposure**

- **Most common occupational lung disease**
- **10 to 25% of adult onset asthma**

**Sensitizer-induced (latency)**
- High-molecular weight agents: cereals, latex, animals, enzymes
- Low-molecular weight agents: isocyanates, metals, dyes, woods

**Irritant-induced (no latency)**
- Acids, alkalis, solvents, fumes, dusts, biocides
### Exposures and Occupational Asthma

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Occupation</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tomatoes</td>
<td>Greenhouses</td>
<td>Vandenplas et al</td>
</tr>
<tr>
<td>Neurospora sitophila</td>
<td>Coffee dispenser</td>
<td>Heffler et al</td>
</tr>
<tr>
<td>Hydroxylamine</td>
<td>Paper recycling</td>
<td>Tran et al</td>
</tr>
<tr>
<td>Methacrylate</td>
<td>Sculptured nails</td>
<td>Sauni et al</td>
</tr>
<tr>
<td>Diisocyanates</td>
<td>Manufacturing</td>
<td>Numerous</td>
</tr>
<tr>
<td>Wood fibers</td>
<td>Wood processing</td>
<td>Heikkila et al</td>
</tr>
<tr>
<td>Almond shells</td>
<td>Processor</td>
<td>Foti et al</td>
</tr>
<tr>
<td>Enzymes</td>
<td>Cheese making</td>
<td>Casper et al</td>
</tr>
</tbody>
</table>
Work-exacerbated Asthma (WEA)

- Pre-existing asthma triggered by specific exposures from the worksite
- Must document presence of conditions that can exacerbate asthma
- Document temporal relationship between work and asthma exacerbation
- Self-reporting has high sensitivity but low specificity
- Must confirm with objective testing
Diagnostic Criteria for RADS (ACCP 1995)

- Documented absence of preceding respiratory complaint
- Onset after single exposure incident/accident
- Exposure to very high concentration of gas, smoke, fumes, or vapors with irritant properties
- Onset of symptoms within 24 hours after exposure with persistence for at least 3 months
- Symptoms that simulate asthma with cough, wheeze, and dyspnea
- Presence of airflow obstruction on pulmonary function ± nonspecific bronchial hyper-responsiveness
- All other pulmonary disease excluded.
Asthma: Diagnostic Considerations

• > 300 known sensitizers exist; new ones found each year (Baur et al, Int Arch Occup Environ Health, 2014)

• Absence of a known sensitizer doesn’t rule out OA/WEA

• Differentiating OA from asthma that has had coincidental onset during work is challenging. BUT temporal relationship of symptoms at/away from work is helpful.

• IgE antibodies (i.e. allergy testing) only possible for a small fraction of known sensitizers; also not helpful for low molecular weight sensitizers

• Induced sputum may show eosinophilic or neutrophilic inflammation
Asthma: Diagnostic Workup

- Careful medical history including prior atopy and asthma history, and careful work exposure history with elements previously discussed
- Physical examination
- Imaging chiefly to establish or rule out comorbidities
- Pulmonary function tests: pre- and post-bronchodilator (FEV$_1$ increase $\geq$ 12% post-bronchodilator establishes asthma)
- Methacholine challenge: a negative test in a patient currently exposed to an agent at work makes a diagnosis of OA very unlikely
- Specific inhalation challenge – performed in Europe, but in North America, only in Quebec. A positive test $= \geq 15\%$ drop in FEV$_1$ at least 6 hours post-exposure.
Peak Expiratory Flow Rates: Caveats

- Typically used 4 times/day over 2 weeks at work, 2 weeks away from work; see if PEFR drops when away from work
- Can be falsified; need 3 supervised tests, reproducible
- Methacholine challenge should be performed to validate results
Asthma: Diagnostic Algorithm

1. PEFR ↓ at work; ↑ away from work; asthma present on spirometry and methacholine challenge + → likely OA. Discontinue occupational exposure.

2. PEFR normal work and away; methacholine - → No OA.

3. Abnormal PEFR, methacholine - → Unlikely OA.

4. Abnormal PEFR, methacholine + → Inconclusive.
Occupational Asthma: Management

- As for non-work-related asthma with respect to education and pharmacotherapy
- BUT most often, employee must be removed completely from further occupational exposure
- Early diagnosis and early removal = best prognosis for resolution
- Older age, high molecular weight sensitizers = worse outcomes
- Majority improve after removal; improvement can continue for several years
- Immune modulation an area for future management
- Omalizumab used in small study: 7/10 employees were able to continue in job. Further studies are needed. Newer agents now being tested.
Spirometry: the ins and outs

- Spirometry is an effort-dependent test
- Criteria for acceptable spirometry:
  - 3 reproducible tests
  - Within 5% of each other
Occupational Chronic Obstructive Pulmonary Disease (COPD)

• Definition:
  • Presence of airflow obstruction that is not fully reversible
  • Usually progressive
  • Associated with an inflammatory response to particles or gases
Occupational COPD

- COPD affects 5-10% of the U.S. population
- 15% of total COPD cases due to occupational exposures (Eisner et al, 2010)
- No single study has found a direct causal relationship with a specific exposure
- Nevertheless, numerous epidemiological studies support a causal relationship between Vapors/Gases/Dusts/Fumes (VGDF) and COPD
## COPD: Risk Factors

<table>
<thead>
<tr>
<th>Non-Occupational</th>
<th>Occupational</th>
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<tbody>
<tr>
<td>• Smoking – 70-80% of COPD cases</td>
<td>• Coal dust (independent of CWP)</td>
</tr>
<tr>
<td>• Genetic (alpha-1 antitrypsin deficiency)</td>
<td>• Silica (independent of silicosis)</td>
</tr>
<tr>
<td>• Age, sex - related to exposures?</td>
<td>• Metals and inorganic dusts</td>
</tr>
<tr>
<td>• Likely factors: air pollution; second-hand smoke exposure; biomass smoke; chronic asthma; early life influences; particulate exposure during adult life</td>
<td>• Welding fumes and gases</td>
</tr>
<tr>
<td></td>
<td>• Organic dusts</td>
</tr>
<tr>
<td></td>
<td>• Generic VGDF</td>
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</tbody>
</table>
Diagnostic Workup: Occupational COPD

Based primarily on clinical status and lung function

1. Screen for alpha-1 antitrypsin deficiency

2. Pulmonary function tests
   • Obstruction; DLCO

3. Oximetry and ABGs

4. Exercise testing
   • Paced shuttle walk test, unpaced 6-minute walk test

5. Imaging
   • CXR, CT chiefly to exclude or establish comorbidities

6. Laboratory tests
   • Eosinophil count to assist in therapeutic decision-making
COPD: Diagnosis

- Definition of airflow limitation: $\text{FEV}_1/\text{FVC} < 70\%$

GOLD criteria for COPD diagnosis

- **GOLD* criteria:**
  - airflow limitation
  - not fully reversible.
  - usually both progressive
  - Usually associated with an abnormal lung inflammatory response to noxious particles or gases

+ Current GOLD definition of airflow limitation is $\text{FEV}_1/\text{FVC}$ ratio $< 70\%$

<table>
<thead>
<tr>
<th>Stage</th>
<th>$\text{FEV}_1$ Percentage</th>
</tr>
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<tbody>
<tr>
<td>I</td>
<td>$&gt; 80%$</td>
</tr>
<tr>
<td>IIA</td>
<td>$50-80%$</td>
</tr>
<tr>
<td>IIB</td>
<td>$30-50%$</td>
</tr>
<tr>
<td>III</td>
<td>$&lt; 30%$</td>
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*GOLD: Global initiative for Chronic Lung Disease
**Assessment of severity (GOLD)**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Spirometric Findings</th>
</tr>
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<tbody>
<tr>
<td>I: Mild</td>
<td>* FEV₁/FVC &lt; 0.70</td>
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<tr>
<td></td>
<td>* FEV₁ ≥ 80% predicted</td>
</tr>
<tr>
<td>II: Moderate</td>
<td>* FEV₁/FVC &lt; 0.70</td>
</tr>
<tr>
<td></td>
<td>* 50% ≤ FEV₁ &lt; 80% predicted</td>
</tr>
<tr>
<td>III: Severe</td>
<td>* FEV₁/FVC &lt; 0.70</td>
</tr>
<tr>
<td></td>
<td>* 30% ≤ FEV₁ ≤ 50% predicted</td>
</tr>
<tr>
<td>IV: Very severe</td>
<td>* FEV₁/FVC &lt; 0.70</td>
</tr>
<tr>
<td></td>
<td>* FEV₁ &lt; 30% predicted or FEV₁ &lt; 50% predicted</td>
</tr>
<tr>
<td></td>
<td>* predicted plus chronic respiratory failure²</td>
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**GOLD ranking of COPD severity**
Gold Abcd assessment tool

• Takes into account the severity, subjective symptoms, and history of exacerbations of disease to arrive at a more comprehensive assessment of disease burden
How is your COPD? Take the COPD Assessment Test™ (CAT)

This brief survey will help your healthcare professional measure the impact of COPD (Chronic Obstructive Pulmonary Disease) on your quality of life. Your answers and score can be used by you and your healthcare professional to help improve the management of your COPD and provide you with the best treatment.

For each item below, please circle a number (0-4) that best describes how you feel or how much you are affected by each problem.

**Examples:**
- I am very happy: 4
- I am very sad: 0

Here are some questions:

- I cough all the time
- I have no phlegm (mucus) in my chest at all
- My chest doesn’t hurt or feel tight at all
- My chest is not full of phlegm (mucus)
- My chest feels very tight
- When I walk up a hill or one flight of stairs I can’t get breathless
- When I walk up a hill or one flight of stairs I am very breathless
- I am not limited doing any activities at home
- I am very limited doing activities at home
- I am confident doing other people’s activities or exercise
- I am not confident doing other people’s activities or exercise
- I sleep soundly
- I wake up several times at night because of my breathlessness
- I have lots of energy
- I have no energy at all

**Score:** 5

**Total Score:**
The mMRC

• The mMRC, from Britain’s Medical Research Council, estimates burden of dyspnea in COPD

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description of Breathlessness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>I only get breathless with strenuous exercise</td>
</tr>
<tr>
<td>Grade 1</td>
<td>I get short of breath when hurrying on level ground or walking up a slight hill</td>
</tr>
<tr>
<td>Grade 2</td>
<td>On level ground, I walk slower than people of the same age because of breathlessness; or I have to stop for breath when walking at my own pace on the level</td>
</tr>
<tr>
<td>Grade 3</td>
<td>I stop for breath after walking about 100 yards or after a few minutes on level ground</td>
</tr>
<tr>
<td>Grade 4</td>
<td>I am too breathless to leave the house or I am breathless when dressing</td>
</tr>
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Gold 2019 Refined COPD Strategy

Spirometrically confirmed diagnosis → Assessment of airflow limitation → Assessment of symptoms/risk of exacerbations

Post-bronchodilator FEV₁/FVC <0.7

<table>
<thead>
<tr>
<th></th>
<th>GOLD 1</th>
<th>GOLD 2</th>
<th>GOLD 3</th>
<th>GOLD 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV₁ (% of predicted)</td>
<td>≥80</td>
<td>50–79</td>
<td>30–49</td>
<td>&lt;30</td>
</tr>
</tbody>
</table>

Moderate/severe exacerbation history

- ≥2 or ≥1 leading to hospital admission
- 0 or 1 (not leading to hospital admission)

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<tr>
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<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>mMRC 0–1</td>
<td>mMRC ≥2</td>
<td></td>
</tr>
<tr>
<td>CAT &lt;10</td>
<td>CAT ≥10</td>
<td></td>
</tr>
</tbody>
</table>

Symptoms

FEV₁ = forced expiratory volume in the first second; FVC = forced vital capacity; mMRC = modified Medical Research Council; CAT = COPD assessment test.
**Occupational COPD Management**

- Tobacco cessation and avoidance
- Decrease/eliminate risk factors/exposures
- Immunization (influenza, pneumococcal)
- Pharmacologic therapy (as for nonoccupational COPD)
- Pulmonary rehabilitation
- Long-term oxygen therapy
- Long-term noninvasive ventilation
- Surgical management (LVRS, Bronchial Thermoplasty, Lung Transplant)
- Palliative measures
Bronchiolitis Obliterans

- Irreversible small airways obstruction via destruction of small bronchioles
- Seen in workers exposed to diacetyl; also with exposures to burn pits, synthetic ‘flock’, some dyes
- Symptoms of cough, shortness of breath, wheezing, fatigue
- Chest x-ray often normal; PFTs show severe fixed obstruction; biopsy useful
- Treatment includes use of corticosteroids and immunosuppressants
- Lung transplantation
Occupational Interstitial Lung Disease (ILD)
Occupational Interstitial lung disease (ILD)

ILD due to a non-specific inflammatory response
- Inorganic dusts – coal dust, silica, asbestos

ILD due to immune sensitization
- Beryllium sensitization; Chronic Beryllium Disease
Coal Mine Dust Lung Disease

- Slide graphics in this section courtesy of Anna Allen, MD, MPH
- WVU School of Public Health
- Department of Occupational and Environmental Health Sciences
- Division of Occupational Medicine
**Active Mining Operations, 2015**

**Underground Coal**

Notes: Puerto Rico and U.S. Virgin Islands not shown. Mining operations that reported any mine operations/employment during the year are spatially visible within counties. Mines where only contractors were working did not show any employment and are not displayed.

Data source: MSHA

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**Active Surface Mining Operations, 2015**

**Coal Industry Sector**

Notes: Puerto Rico and U.S. Virgin Islands not shown. Mining operations that reported any mine operators/employment during the year are spatially visible within counties. Mines where only contractors were working did not show any employment and are not displayed.

Data source: MSHA
Coal Workers’ Pneumoconiosis

Percentage of examined miners with CWP Category 1 or greater by tenure in coal mining
(NIOSH Coal Workers’ Health Surveillance Program, 1970–2014)

Prevalence of CWP, %

Surveillance Period

Percentage profusion category 2 and 3

US excluding KY, VA, WV

KY, VA, WV

West Virginia University
Coal Mining Activities

- Roof bolting
- Face work
- Blasting/shooting
- Drilling
Coal Mining: Then and Now

- Historically, exposures were mostly coal dust.
- Coal dust is a heterogenous mixture composed of coal, quartz, silicates, pyrite, and calcite.
- Improved technology allows for mining coal seams and greater exposure to surrounding silica-containing rock.
Dust Exposure Variables

- Concentration
- Size (Aerodynamic diameter)
- Composition
- Air velocity (breathing rate)
- Recovery time
- Other exposures

From Geiser and Kreyling, Particle Fibre Toxicol, 2010, 7:2
Classic diseases seen with coal dust exposure

- Chronic bronchitis
- Chronic Obstructive Pulmonary Disease
- Coal Workers’ Pneumoconiosis
Coal and the Lung

Dust deposits in distal airways → Macrophage → Interstitum/lymph

Results in loss of surface lung surface area
Thickening of the alveolar surface
Fibrosis of the supporting structures

Inflammatory byproducts

West Virginia University
‘Medical’ Coal Workers’ Pneumoconiosis

Coal macules: anthracotic macrophages 1-2 mm, within bronchiolar walls. Accompanied by centrilobular emphysema.

Coal nodules: larger collections of macrophages, surrounded by more prominent emphysema, fibrosis.

If dust contains high silica content, nodules show typical silicosis pattern. May coalesce into massive fibrosis.

Large lesions may cavitate and become colonized with mycobacteria (TB) and fungi.
Normal

‘Simple’ Pneumoconiosis

PMF

Source: NIOSH Coal Workers’ X-ray Surveillance Program (CWXSP)

Coal Workers’ Pneumoconiosis (CWP)

Progressive massive fibrosis
Complicated pneumoconiosis
‘Legal’ Coal Workers’ Pneumoconiosis (CWP)

**Diagnosis**
- Characteristic radiological findings
- History of coal dust exposure

**Simple**
- Opacities less than 10 mm

**Complicated**
- At least one opacity $\geq 10$ mm
- Progressive Massive Fibrosis
CWP: Radiological Diagnosis

- ILO ‘B’ Read
  - Film quality
  - Small round opacities – p, q, r (< 1.5, 1.5-3, 3-10 mm)
  - Small irregular opacities – s, t, u (same sizes)
  - Profusion (0 – 3 scale, 0/- to 3/+)
  - Large opacities – A,B,C
  - Pleural and other abnormalities
CWP: Small and Large Opacities
Silica and the Lung: Respirable Crystalline Silica

- **Acute Silicosis**
  - Occurs within weeks to months (Hawk’s Nest Tunnel disaster)

- **Accelerated Silicosis**
  - Occurs within 5-10 years

- **Chronic Silicosis**
  - 10+ years
Silicosis: Symptoms and Diagnosis

- Gradual onset progressive shortness of breath, +/- cough; wheezing not prominent
- Imaging shows ‘ground glass’ opacities, hazy infiltrates
- Histology shows inflammation and alveolar filling similar to pulmonary alveolar proteinosis
- Significantly increased risk for mycobacterial (TB) coinfection; treat latent and active infection promptly
- Lung cancer and scleroderma can also be attributed to silica exposure: to meet criteria for causation, the patient should also meet criteria for a diagnosis of silicosis.
Silicosis: Silicotic Nodule, Eggshell Calcifications on Chest X-ray
Asbestos

- Hydrated magnesium silicates that readily separate into long, flexible fibers
  - Amphibole
    - Crocidolite, Amosite, Tremolite, Actinolite, Anthophyllite
  - Serpentine
    - Chrysotile (95% of production)
Asbestos and the Lung: Clinical Effects

- Pleural Plaques
- Asbestosis (diffuse parenchymal fibrosis)
- Lung cancer
- Pleural mesothelioma
Asbestos: Effect on Lung Tissue

- Fibers not cleared by mucociliary process attract macrophages
- Macrophages stimulate inflammatory response
- Cytokines and inflammation stimulate fibrosis
- Pleural plaques and thickening seen within 10 years; benign
- Asbestosis reflects parenchymal fibrosis; latency 20+ years.
- Lung cancer
  - Adenocarcinoma most common type
  - Requires substantially higher fiber burden/exposure than asbestosis
  - Cannot be attributed to asbestos exposure in absence of asbestosis
- Mesothelioma
  - Latency 30-50 years
  - Fiber burden less than for asbestosis but not 3x that for pleural plaques
Asbestos-related pulmonary disease: Diagnosis

- History: progressive shortness of breath; dry cough; exposures
- Physical: dry crackles at lung bases; clubbing
- Imaging: Chest x-ray B read; lower lung fields affected; HRCT useful
- PFTs: Decreased FVC and DLCO (confounder: smoking)
- Pathology: Presence of asbestos bodies
This PA radiograph shows some of the typical findings of asbestosis including a "shaggy heart", pleural plaques and diaphragm calcification
ILD due to immune sensitization: Chronic Beryllium Disease (CBD)

- Granulomatous lung disease only distinguishable from sarcoidosis by the presence of Beryllium (Be) sensitization
- Caused by delayed-type (Type IV) hypersensitivity to Be
- Be Lymphocyte Proliferation Test (BeLPT) indicates whether sensitization has occurred
- Be is more rigid than steel, lighter than aluminum, high melting point, excellent conductor of heat and electricity
- Exposures in mining; in bauxite smelting; in aerospace, electronics, alloy manufacture, recycling of e-waste, many industries
Chronic Beryllium Disease (CBD)

- Genetic susceptibility to sensitization exists (HLA DPB1 glu69)
- Exposure is inhalational or transdermal
- Sensitization and subsequent immune response results in development of noncaseating granulomas that evolve to cause pulmonary fibrosis
- No evidence that removal from exposure reverses or halts the process; however, removal is considered best practice.
- Symptoms of fever, night sweats, weight loss, cough, shortness of breath, fatigue. Latency 3 months – 30 years.
Chronic Beryllium Disease

- Granulomas
- Hilar lymphadenopathy
- Fibrosis
Diagnostic Workup: Occupational ILD

Based primarily on radiological findings; other tests support diagnosis

1. Imaging
   - CXR with B read
   - HRCT

2. Pulmonary function tests
   - Obstruction or mixed picture may occur

3. Oximetry and ABGs

4. Laboratory tests
   - Be lymphocyte proliferation test

5. Pathology
General management of ILD

- Removal from exposure (especially for nonspecific inflammatory response types)
- Pneumococcal vaccination
- Annual influenza vaccination
- Pulmonary rehabilitation
- Oxygen therapy to keep saturation > 89%
Specific approaches: non-specific inflammatory response ILD

- Screen for tuberculosis

- Treat latent TB if PPD > 10 mm or IGRA is positive and no active infection
  
  Treat for 8 months rather than 6 months to reduce risk of recurrence

- General principles
Specific approaches: ILD due to immune sensitization

- Similar to that for sarcoidosis
- Disease remission is unlikely with CBD
- Inhaled corticosteroids (ICS)
- Short-acting beta-agonist bronchodilators (SABA)
- Oral corticosteroids (relapse common during tapering)
- Lung transplantation for end-stage CBD
Hypersensitivity Pneumonitis (Extrinsic Allergic Alveolitis)

- Immunological responses of the lung causing symptoms of cough, wheeze, shortness of breath, myalgia, arthralgia, malaise
  - Frequently mistaken for viral illness; often misdiagnosed
- Caused by organic dusts, microbial agents, chemicals
- Usually self-limiting, with improvement in 12-24 hours and recovery within a few days
- Subacute form goes on for weeks to months
- Progression to lung fibrosis after several years
### Some Causes of HP/Extrinsic Allergic Alveolitis

<table>
<thead>
<tr>
<th>Name</th>
<th>Exposure</th>
<th>Antigen</th>
</tr>
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<tbody>
<tr>
<td>Farmer’s lung</td>
<td>Moldy hay</td>
<td>Aspergillus, Candida, etc.</td>
</tr>
<tr>
<td>Lifeguard lung</td>
<td>Contaminated water</td>
<td>Pseudomonas</td>
</tr>
<tr>
<td>Bird fanciers’ lung</td>
<td>All types of birds</td>
<td>Proteins from feathers, GI</td>
</tr>
<tr>
<td>Coffee workers’ lung</td>
<td>Coffee bean dust</td>
<td>Coffee</td>
</tr>
<tr>
<td>Mushroom workers’ lung</td>
<td>Moldy compost</td>
<td>Thermophilic actinomyces</td>
</tr>
<tr>
<td>Tractor lung</td>
<td>Tractor cabs</td>
<td>Rhizopus spp</td>
</tr>
<tr>
<td>Pyrethrum alveolitis</td>
<td>Pesticide</td>
<td>Pyrethrum</td>
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Hypersensitivity pneumonitis: management

- Cornerstone: early identification and antigen avoidance
- Corticosteroids provide rapid improvement
  - Do not influence long-term outcome
- Steroid-sparing agents (mycophenolate mofetil, azathioprine)
  - Improvements in DLCO but not FVC in chronic HP
- Biological therapies (rituximab) in severe, refractory disease
- Referral for lung transplantation if failure to respond
Infectious occupational pulmonary diseases

Infections with a zoonotic source – Hantavirus, Psittacosis, Q Fever, Tularemia, Plague, Anthrax, Human Avian Influenza, Leptospirosis, SARS and MERS

Infections with an environmental source – Histoplasmosis and other fungal infections, Legionnaires’ Disease, Melioidosis

Infections with a human source – Tuberculosis; infections in healthcare workers; invasive pneumococcal disease
Emerging challenges in occupational lung disease

Known agents, novel occupational settings
- Isocyanates, methacrylate, cyanoacrylate, linseed oil, silica, coal dusts, fluoropolymers

Novel agents not previously known to cause lung disease
- Machine cooling fluids; chamomile flower; peptide coupling reagents; short-length synthetic fibers; burn pits; indium-tin oxide; nanoparticles; cerium
## Known Agents: Novel Occupational Settings

<table>
<thead>
<tr>
<th>Condition</th>
<th>Exposure</th>
<th>Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>Healthcare techs working with casting material</td>
<td>Isocyanates</td>
</tr>
<tr>
<td></td>
<td>Nail salon workers</td>
<td>Methacrylates</td>
</tr>
<tr>
<td></td>
<td>Research chemists</td>
<td>Linseed oil</td>
</tr>
<tr>
<td>Silicosis</td>
<td>Sandblasting denim</td>
<td>Silica</td>
</tr>
<tr>
<td></td>
<td>Stone countertop fabrication</td>
<td>Silica</td>
</tr>
<tr>
<td>Acute lung injury</td>
<td>Leather protectant users, floor sealant users</td>
<td>Fluoropolymers</td>
</tr>
</tbody>
</table>
## Novel Agents: Emerging Issues

<table>
<thead>
<tr>
<th>Condition</th>
<th>Exposure</th>
<th>Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypersensitivity Pneumonitis</td>
<td>Animal feed industry</td>
<td>Phytase enzymes</td>
</tr>
<tr>
<td>Lymphocytic bronchiolitis</td>
<td>Nylon workers</td>
<td>Short-length synthetic fibers</td>
</tr>
<tr>
<td>(Flock workers’ lung)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bronchiolitis obliterans</td>
<td>Flavoring industry workers</td>
<td>Diacetyl</td>
</tr>
<tr>
<td>Constrictive bronchiolitis</td>
<td>Deployed soldiers</td>
<td>Burn pits</td>
</tr>
<tr>
<td>Pulmonary alveolar proteinosis</td>
<td>Indium processing, liquid crystal panels</td>
<td>Indium-tin oxide</td>
</tr>
</tbody>
</table>
Thank you!

Questions?
Selected References

- Parke et al. Parkes’ Occupational Lung Diseases, CRC Press, 2017 by Taylor & Francis Group LLC.
Selected References (continued)


- NIOSH Coal Workers’ Surveillance Program [https://www.cdc.gov/niosh/topics/cwhsp/default.html](https://www.cdc.gov/niosh/topics/cwhsp/default.html)