Occupational and Environmental Lung Diseases

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January 10th and 12th, 2018
Objectives

• Overview of various occupational lung diseases
• Things to consider with occupational lung diseases
• Preventive measures
Why is it important to know about occupational lung diseases?

• Disease progression
• Treatment
• Public health
• Financial and legal implications
• Disease models
• 46 y/o M surgical pathologist presents with increasing SOB over the previous 4 years.

• Cough and dyspnea within 30 minutes starting work. Continued through workweek and would progressively worsen until he left work.

• Intermittent rash on his upper extremities and torso, postnasal drip, progressive dyspnea on exertion.

• No seasonal variations to his symptoms.
Additional thoughts on history?
• 20 years prior, he noted a pruritic, erythematous rash on dorsal aspects of his hands when he wore latex gloves.
• Would apply steroid cream without relief.
• Noted heavy breathing if he “flipped” his gloves off.
• Use of xylene and formaldehyde exacerbated his symptoms.
• Seen by allergist, otorhinolaryngologist, and a dermatologist.
• Biopsy of rash consistent with acute urticaria.
• Latex skin prick tests were positive to latex glove extracts, dust, cat dander, and mold antigens.
• CT sinuses: nasal polyps in the maxillary sinus.
• Dx: chronic sinusitis, asthma, and allergic rhinitis.
• Tx: antibiotics, steroid taper, Serevent, Flovent, Proventil, and recommended to use a surgical mask while at work.
• Symptoms continued to progress.
• 2 months later: now c/o single flight dyspnea.
- PMH: HTN, nasal polyps, near syncope
- Family hx: Asthma in a sister and paternal uncle
- SH: No etoh, tobacco, of illicit drugs
- Allergies: Denied
- PE: VS WNL. BMI 30. Hyperemic conjunctivae, boggy nasal mucosa, erythematous urticarial rash on his right shoulder, and diffuse expiratory wheezing.
• ECG: normal
• Chest x-ray: poor inspiration
• CT chest: mild bronchial wall thickening consistent with mild airways disease
• RAST for latex IgE ab negative
• PFT: mild obstruction with acute bronchodilator response
Occupational Asthma

• Occupational asthma (OA) can be defined as the presence of variable airflow obstruction and bronchial hyperresponsiveness caused by a substance found in the workplace.

• Approximately 10% of adult asthma cases are attributed to an occupational etiology.
  
  • OA has been reported in 8% to 12% of laboratory animal workers, 7% to 9% of bakers, and 1.4% of healthcare workers exposed to natural rubber latex. Farmers, painters, plastic and rubber workers, and cleaners (caretakers, window cleaners, chimney and road sweepers) are at greatest risk for developing asthma.
  
  • The prevalence of latex sensitization has been estimated to be between 5 and 17% in health care workers versus between 5 and 10% in the general population.

• Atopic individuals are more easily sensitized to allergens and, as such, are at greater risk of developing a latex allergy than are individuals who are not atopic.

• Work aggravated asthma

http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/allergy/occupational-asthma/
Occupational Asthma

• Types
  • Immunologic
  • Non-Immunologic
  • Work aggravated asthma
Occupational Asthma

- HMW
- LMW
  - Isocyanates
    - Paints
    - Plastics
    - Rubber
    - Foam
      - Activated T-cells
      - HLA class II antigens
      - Beta-2 adrenergic receptors
      - Stimulate sensory nerves

LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.
## Major causes of occupational asthma

<table>
<thead>
<tr>
<th>Low molecular weight chemicals</th>
<th>Occupation at risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isocyanates (e.g., toluene diisocyanate, diphenylmethane diisocyanate, hexamethylene diisocyanate, methylene diphenyl diisocyanate)</td>
<td>Polyurethane workers, roofers, insulators, painters</td>
</tr>
<tr>
<td>Anhydrides (e.g., trimellitic anhydride, phthalic anhydride)</td>
<td>Manufacturers of paint, plastics, epoxy resins</td>
</tr>
<tr>
<td>Metals (e.g., chrome acid, potassium dichromate, nickel sulfate, vanadium, platinum salts)</td>
<td>Platers, welders, metal and chemical workers</td>
</tr>
<tr>
<td>Drugs (e.g., beta lactam agents, opiates, other)</td>
<td>Pharmaceutical workers, farm workers, health professionals</td>
</tr>
<tr>
<td>Wood dust (e.g., Western red cedar, maple, oak, exotic woods)</td>
<td>Carpenters, woodworkers</td>
</tr>
<tr>
<td>Dyes and bleaches (e.g., anthraquinone, carmine, hemna extract, persulfate, reactive dyes)</td>
<td>Fabric and fur dyers, hairdressers</td>
</tr>
<tr>
<td>Amines</td>
<td>Chemists, cleaners, plastic manufacturers</td>
</tr>
<tr>
<td>Glues and resins (e.g., acrylates, epoxy)</td>
<td>Plastic manufacturers</td>
</tr>
<tr>
<td>Miscellaneous (e.g., formaldehyde, glutaraldehyde, ethylene oxide, pyrethrin, polyvinyl chloride vapor)</td>
<td>Laboratory workers, textile workers, paint sprayers, health professionals</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>High molecular weight organic materials</th>
<th>Occupation at risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Animal proteins (e.g., domestic and laboratory animals, fish and seafood)</td>
<td>Farmers, veterinarians, poultry processors, fish and seafood processors</td>
</tr>
<tr>
<td>Flours and cereals</td>
<td>Bakers, food processors, dock workers</td>
</tr>
<tr>
<td>Enzymes (e.g., pancreatic extracts, papain, trypsin, Bacillus subtilis, bromelain, peptinase, amylase, lipase)</td>
<td>Bakers, food processors, pharmaceutical workers, plastic workers, detergent manufacturers</td>
</tr>
<tr>
<td>Plant proteins (e.g., wheat, grain dust, coffee beans, tobacco dust, cotton, tea, latex, psyllium, rancid flours)</td>
<td>Bakers, farmers, food and plant processors, health professionals, textile workers</td>
</tr>
</tbody>
</table>
Occupational Asthma

• Features of OA:
  • Nasal, ocular, or contact urticarial symptoms that precede asthma symptoms.
  • Association of prolonged exposure with worsening asthma symptoms at work.
  • Development of more pervasive symptoms while at work.
  • Presence of a latency period between initial exposures to the inciting agent where symptoms may develop weeks to more than 20 years after exposure.

Work relatedness?

• Was it present before beginning this job? Occupational history?
• Has it changed since beginning this job?
• Does it tend to go away with breaks?
• Has there been a change with use of new chemical or process?
• Does PPE change anything?
• Are coworkers affected?
Occupational Asthma

• Diagnosis
  • History
  • PEFR
    • Pre and post shift monitoring of lung function
    • Peak Expiratory Flow at and off work for period of several weeks - 4 times daily, preferable every 2 hours
    • Look for the pattern
    • Realize that PEF can be full of errors (malingering, poor technique, false positives, poor compliance)
  • Spirometry/PFT
  • Methacholine challenge testing
Treatment

- Disability from occupationally induced allergies is compensable under Workers’ Compensation law
- Prevention of exposure
  - Engineering and IH controls
  - PPE
- Removal from workplace

What happened to our patient?
Board Question

Occupational asthma is typically characterized by all of the following EXCEPT:

A) Positive methacholine challenge test  
B) Decreased peak expiratory flow rates associated with exposure  
C) Irreversible bronchoconstriction  
D) A decrease in FEV₁ of > 10 – 20% on pre- and post-shift spirometry testing
Board Question

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A) Positive methacholine challenge test
B) Decreased peak expiratory flow rates associated with exposure
C) Irreversible bronchoconstriction
D) A decrease in FEV₁ of > 10 – 20% on pre- and post-shift spirometry testing
Occupational asthma is characterized by reversible bronchospasm. Examples of agents associated with occupational asthma include: metal fumes and salts (e.g., nickel, chromium, cobalt, and platinum salts), organic chemicals (e.g., isocyanates, formaldehyde), cotton dust, wood dusts, grain dusts, etc.
Board Question

All of the following are true with respect to occupational asthma **EXCEPT:**

A) It accounts for about 2 – 15% of new cases of adult asthma
B) Workers with comparable exposures will be affected to the same degree
C) Typical symptoms include cough, chest tightness, wheezing, and dyspnea
D) Symptoms show improvement on weekends and vacations
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C) Typical symptoms include cough, chest tightness, wheezing, and dyspnea
D) Symptoms show improvement on weekends and vacations
B) Workers with comparable exposures will be affected to the same degree

Host factors, such as atopy and smoking, seem to play a role in predisposition of workers to occupational asthma. For affected individuals, job transfer to an area free from exposure to the offending agent is usually indicated.
Occupational Lung Diseases

- Rhinitis
- Laryngitis
- Tracheitis
- Bronchitis
- Bronchiolitis
- COPD
- Lung Cancer
- Interstitial diseases

- Particle size
- Site of deposition
- Water solubility
- Type of cell/structure damaged

LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.
Occupational Lung Diseases

- History
  - Work practices
  - Appropriate environmental controls
  - Respiratory protection gear
  - MSDS
  - Industrial hygiene data
  - Possible exposures outside of the home
• Physical
  • Not specific to occupational lung disease
  • Complete physical examination

• Imaging
  • CXRs often normal
  • ILO classification used for dust-exposed persons
  • HRCT>CT

• Pulmonary function testing
  • Spirometry: FEV₁, FVC, and FEV₁/FVC ratio
  • Peak expiratory flow rate (PEFR)
  • Bronchoprovocation tests
Occupational Lung Disease

• Prevention!
  • Elimination
  • Administrative controls
  • Engineering controls
  • PPE
  • Medical surveillance programs
  • IH surveys
Hierarchy of Controls

1. Elimination
   - Physically remove the hazard

2. Substitution
   - Replace the hazard

3. Engineering Controls
   - Isolate people from the hazard

4. Administrative Controls
   - Change the way people work

5. PPE
   - Protect the worker with Personal Protective Equipment

Source: NIOSH

Which Respirator?

- Particulate size and composition of particulate
- Other chemicals or exposures
- Concentration of exposures
- OSHA or Cal/OSHA regulations regarding chemicals
- Health of the patient (respiratory protection program)
Overview of Occupational Lung Diseases
Inhalation Fevers

- Short-term, flulike symptoms after exposure to organic dusts, polymer fumes, and metal fumes usually requiring a high level exposure usually after 3-10 hours after exposure, sx usually resolve in 1-2 days
- PE: crackles on lung auscultation
- CXR, PFT, and ABG usually normal
- Some common agents:
  - Zinc oxide
  - Polytetrafluoroethylene (Teflon)
    - Severe cases may lead to chemical pneumonitis with pulmonary edema
  - Cotton
  - Grain dust
- Symptomatic treatment and future prevention

LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.
COPD

- 15% of all cases of COPD is work-related


Figure 1: Proportion of patients with chronic obstructive pulmonary disease (COPD) who are non-smokers worldwide.

*Australia, Belgium, Denmark, France, Germany, Iceland, Ireland, Italy, Netherlands, New Zealand, Norway, Spain, Sweden, Switzerland, UK, and USA.*

• At risk:
  • Coal miners
  • Hard-rock miners
  • Tunnel workers
  • Concrete-manufacturing workers
  • Nonmining industrial workers
• Minerals
  • Coal
  • Oil mist
  • Silica
  • Silicates
  • Synthetic vitreous fibers
  • Portland cement
• Metals
  • Osmium
  • Vanadium
  • Welding fumes
• Organic dusts
  • Cotton
  • Grain
  • Wood
• Smoke
  • Tobacco smoke
  • Fire smoke
  • Engine exhaust

• 29 y/o M food flavoring worker, asymptomatic. Presents for workplace medical surveillance
• PMH significant for questionable childhood asthma
• Non-smoker
• Employed as a production worker at flavoring industry for 10 years
- PE includes normal vital signs and normal cardiopulmonary exam
- Spirometry: obstruction with no bronchodilator response

<table>
<thead>
<tr>
<th>Measurements</th>
<th>10/31/06</th>
<th>3/13/07</th>
<th>Repeated on 3/14/07</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pre-BD</td>
<td>Post-BD*</td>
<td>Pre-BD</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>6.38</td>
<td>5.96</td>
<td>5.77</td>
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<tr>
<td>FVC</td>
<td>113.5%</td>
<td>106.2%</td>
<td>102.8%</td>
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<tr>
<td>Percent predicted</td>
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<td></td>
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<tr>
<td>FEV₁ (L)</td>
<td>4.10</td>
<td>3.06</td>
<td>3.02</td>
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<tr>
<td>FEV₁</td>
<td>86.5%</td>
<td>64.5%</td>
<td>63.7%</td>
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<tr>
<td>Percent predicted</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV₁/FVC†</td>
<td>64.4%</td>
<td>51.3%</td>
<td>53.0%</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>52.3%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>53.3%</td>
</tr>
</tbody>
</table>
• HRCT chest:
  • inhomogeneity in the attenuation of the lung parenchyma and air trapping best seen on expiratory phase. There is diffuse involvement of both lungs. The findings of a mosaic pattern of patchy diffuse air trapping and fibrosis
What is the most likely diagnosis?

A. Bronchiolitis Obliterans
B. Occupational asthma
C. Chronic obstructive pulmonary disease
D. Pneumoconiosis
Bronchiolitis Obliterans

- Characterized by fibrosis of the bronchioles
- Fixed airway obstruction
- Symptoms:
  - No symptoms
  - Cough
  - Dyspnea on exertion
  - Wheezing

https://www.cdph.ca.gov/Programs/CCDPHP/DEODC/OHB/Pages/FlavoringLungDisease.aspx
• Irritant gas inhalation
  • Oxides of nitrogen, chlorine, phosgene, ozone, hydrogen sulfide, and sulfur dioxide
• Cases reported in nylon flock workers, battery workers (exposed to thionyl chloride), and textile workers exposed to polyamide-amine dyes.
• Diacetyl?

LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.
Bronchiolitis Obliterans

- Popcorn lung disease
Diacetyl

Figure 1. 2-Dimensional representation of diacetyl [http://www.thewinemerchantinc.com/educational/WineAcid.html].
• Found in a group of 8 microwave popcorn plant workers in Missouri from 1993-2000.

• Cases also occurred in California flavor manufacturing plants in addition to New Jersey, Iowa, Ohio, and Illinois.

• Associated with inhalation of volatile butter-flavoring ingredients, namely diacetyl

• Cal OSHA passed the diacetyl standard which includes medical surveillance of employees exposed to food flavoring chemicals
Common findings consistent with BO seen on an HRCT of the chest include the following

- Cylindric bronchiectasis
- Bronchial wall thickening
- Heterogeneous air trapping during expiration
- Patchy ground glass opacities
- Mosaic pattern of attenuation
Computed tomogram of bronchiolitis obliterans

Computed tomography (CT) image of a patient with bronchiolitis obliterans. A mosaic pattern of attenuation is present in which higher attenuation indicates normal lung with relatively greater perfusion, whereas lower attenuation areas are caused by air trapping and hypoxic vasoconstriction in areas with narrowed small airways.

Graphic 91544 Version 1.0
Coffee Workers’ Concerns Brew Over Chemical’s Link To Lung Disease

April 15, 2016 · 5:15 AM ET
Heard on Morning Edition
In addition to concerns about vaping leading to smoking, the American Lung Association considers the vapors from e-cigarettes themselves unsafe. (iStock)
What should we do with our food flavoring worker now?

A. Tell him to quit his job

B. Continue doing the same work with a respirator after being fit-tested

C. Recommend that he be moved to an unexposed work area and obtain close medical monitoring
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<table>
<thead>
<tr>
<th>Dates:</th>
<th>FVC (L)</th>
<th>FVC % Predicted</th>
<th>FEV₁ (L)</th>
<th>FEV₁ % Predicted</th>
<th>FEV₁/FVC (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>04/17/07</td>
<td>5.33</td>
<td>95</td>
<td>2.91</td>
<td>62</td>
<td>56</td>
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<tr>
<td>05/22/07</td>
<td>5.62</td>
<td>101</td>
<td>3.09</td>
<td>65</td>
<td>55</td>
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<tr>
<td>06/22/07</td>
<td>5.64</td>
<td>102</td>
<td>3.00</td>
<td>64</td>
<td>55</td>
</tr>
<tr>
<td>06/29/07</td>
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<td>102</td>
<td>3.22</td>
<td>68</td>
<td>56</td>
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<td>07/31/07</td>
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<td>105</td>
<td>3.17</td>
<td>67</td>
<td>55</td>
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<td>09/11/07</td>
<td>6.07</td>
<td>109</td>
<td>3.27</td>
<td>70</td>
<td>54</td>
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<tr>
<td>10/17/07</td>
<td>5.81</td>
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<td>3.13</td>
<td>67</td>
<td>54</td>
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<td>3.09</td>
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<td>54</td>
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<td>103</td>
<td>3.08</td>
<td>65</td>
<td>54</td>
</tr>
<tr>
<td>04/01/08</td>
<td>5.54</td>
<td>100</td>
<td>2.95</td>
<td>63</td>
<td>53</td>
</tr>
<tr>
<td>05/16/08</td>
<td>5.53</td>
<td>100</td>
<td>2.99</td>
<td>64</td>
<td>54</td>
</tr>
</tbody>
</table>
Hypersensitivity Pneumonitis

- Hypersensitivity pneumonitis (HP), also called extrinsic allergic alveolitis, is a respiratory syndrome involving the lung parenchyma and specifically the alveoli, terminal bronchioli, and alveolar interstitium, due to a delayed allergic reaction.
  - Organic dusts
  - Chemicals
- At risk professions:
  - Farmers
  - Breeders
- First described in 1713 by the Italian researcher Bernardino Ramazzini in subjects belonging to 52 different professions, a repeated exposure to particles sufficiently small (diameter < 5 μm) to reach the alveoli and to trigger an immune response is necessary.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Acute/subacute HP</th>
<th>Chronic HP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure to causal antigen</td>
<td>Intermittent high-level exposure</td>
<td>Continuous low-level exposure</td>
</tr>
<tr>
<td>Onset of symptoms</td>
<td>2–9 h after exposure; may evolve to gradually increasing symptoms over days to weeks</td>
<td>Insidious, over weeks to months</td>
</tr>
<tr>
<td>Nature of symptoms</td>
<td>Cough and dyspnea, but predominantly influenza-like symptoms</td>
<td>Progressive symptoms (dyspnea, cough, and weight loss), sometimes punctuated by intermittent attacks of symptoms or slowly increasing</td>
</tr>
<tr>
<td>Physical signs</td>
<td>Fever</td>
<td>Inspiratory crackles; cyanosis; digital clubbing; cor pulmonale</td>
</tr>
<tr>
<td>Outcome</td>
<td>Symptoms peak within 6–24 h after exposure, lasting hours to days. Symptoms recur on re-exposure and may progress to severe dyspnea</td>
<td>End-stage fibrotic disease and/or emphysema. Exacerbations may occur despite avoidance of exposure</td>
</tr>
</tbody>
</table>

Chronic hypersensitivity pneumonia (extrinsic allergic alveolitis)

Chronic phase of hypersensitivity pneumonia (extrinsic allergic alveolitis) due to birds. Focal honeycombing is present in the right upper lobe and mild bilateral cylindrical bronchiectasis is visible.

Courtesy of Paul Stark, MD.
Chest CT scan in patient with acute hypersensitivity pneumonia

Chest computed tomogram in patient with acute hypersensitivity pneumonia demonstrating so-called "head cheese" pattern with mixture of ground-glass, consolidation, and air-trapping in adjoining secondary pulmonary lobules.
Hypersensitivity pneumonitides associated with plastic manufacturing, painting, electronics industry, and other chemicals

<table>
<thead>
<tr>
<th>Environmental source</th>
<th>Major causative antigen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemical worker's lung</td>
<td>Diphenylmethane diisocyanate (MDI)</td>
</tr>
<tr>
<td></td>
<td>Toluene diisocyanate (TDI)</td>
</tr>
<tr>
<td>Detergent worker's lung (washing powder lung)</td>
<td>Bacillus subtilis enzymes</td>
</tr>
<tr>
<td>Pauli’s reagent alveolitis</td>
<td>Sodium diazobenzene sulfate</td>
</tr>
<tr>
<td>Vineyard sprayer's lung</td>
<td>Copper sulfate (bordeaux mixture)</td>
</tr>
<tr>
<td>Pyrethrum (pesticide)</td>
<td>Pyrethrum</td>
</tr>
<tr>
<td>Epoxy resin lung</td>
<td>Phthalic anhydride (heated epoxy resin)</td>
</tr>
<tr>
<td>Bible printer's lung</td>
<td>Moldy typesetting water</td>
</tr>
<tr>
<td>Machine operator's lung</td>
<td>Pseudomonas fluorescens</td>
</tr>
<tr>
<td></td>
<td>Mycobacterium immunogenum</td>
</tr>
<tr>
<td></td>
<td>Aerosolized metal working fluid</td>
</tr>
</tbody>
</table>
### Hypersensitivity pneumonitides associated with ventilation and water-related contamination

<table>
<thead>
<tr>
<th>Environmental source</th>
<th>Major causative antigen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Humidifier fever</td>
<td>Thermoactinomyces (T vulgaris, T sacchari, T candidus)</td>
</tr>
<tr>
<td></td>
<td>Klebsiella oxytoca</td>
</tr>
<tr>
<td></td>
<td>Neoglena gruberi</td>
</tr>
<tr>
<td></td>
<td>Acanthamoeba polyphaga</td>
</tr>
<tr>
<td></td>
<td>Acanthamoeba castellani</td>
</tr>
<tr>
<td>Unventilated shower</td>
<td>Epicoccum nigrum</td>
</tr>
<tr>
<td>Hot-tub lung (mists; mold on ceiling and around tub)</td>
<td>Cladosporium sp</td>
</tr>
<tr>
<td></td>
<td>Mycobacterium avium complex</td>
</tr>
<tr>
<td>Sauna taker's lung</td>
<td>Aureobasidium sp, other sources</td>
</tr>
<tr>
<td>Summer-type pneumonitis</td>
<td>Trichosporon cutaneum</td>
</tr>
<tr>
<td>Lifeguard lung</td>
<td>Aerosolized endotoxin from pool-water sprays and fountains</td>
</tr>
<tr>
<td>Contaminated basement (sewage) pneumonitis</td>
<td>Cephalosporium</td>
</tr>
</tbody>
</table>
### Hypersensitivity pneumonitides associated with farming

<table>
<thead>
<tr>
<th>Environmental source</th>
<th>Major causative antigen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moldy hay, grain, silage</td>
<td>Thermophilic actinomycetes, such as <em>Faenia recti</em>, Virgula (also known as <em>Micropolyspora faeni</em> or <em>Saccharopolyspora rectivirgula</em>) <em>Fungus, such as Aspergillus umbrosus</em></td>
</tr>
<tr>
<td>Mold on pressed sugar cane (bagassosis, very rare cases)</td>
<td>Thermoactinomyces sacchari, <em>T. vulgaris</em></td>
</tr>
<tr>
<td>Tobacco plants (tobacco grower’s lung; “Blackfoot” tobacco)</td>
<td>Aspergillus sp</td>
</tr>
<tr>
<td></td>
<td><em>Scopulariopsis brevicaulis</em></td>
</tr>
<tr>
<td>Mushroom worker’s lung</td>
<td>Mushroom spores</td>
</tr>
<tr>
<td></td>
<td>Thermophilic actinomycetes</td>
</tr>
<tr>
<td>Potato riddle’s lung (moldy hay around potatoes)</td>
<td>Thermophilic actinomycetes</td>
</tr>
<tr>
<td></td>
<td><em>T. vulgaris</em></td>
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<tr>
<td></td>
<td><em>F. rectivirgula</em></td>
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<td></td>
<td>Aspergillus sp</td>
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<tr>
<td>Paprika slicer’s lung (moldy paprika pods)</td>
<td><em>Mucor zoosporifer</em></td>
</tr>
<tr>
<td>Wine maker’s lung (mold on grapes)</td>
<td><em>Botrytis cinerea</em></td>
</tr>
<tr>
<td>Cheese washer’s lung (moldy cheese)</td>
<td><em>Penicillium casei</em></td>
</tr>
<tr>
<td></td>
<td><em>Aspergillus clavatus</em></td>
</tr>
<tr>
<td>Coffee worker’s lung</td>
<td><em>Coffee-bean dust</em></td>
</tr>
<tr>
<td>Tea grower’s lung</td>
<td><em>Tea plants</em></td>
</tr>
</tbody>
</table>
Hypersensitivity pneumonitis is caused by exposure to:

A) Oxides of heavy metals  
B) Irritant gases  
C) Plastic constituents  
D) Organic dust
Hypersensitivity pneumonitis is caused by exposure to:
A) Oxides of heavy metals
B) Irritant gases
C) Plastic constituents
D) Organic dust
Various organic dusts can cause hypersensitivity pneumonitis with acute symptoms of fever, cough, dyspnea, malaise.

D) Organic dust
Metal-Induced Lung Disease

• Hard metal
  • Cemented alloy of tungsten carbide with cobalt (or other metals).
  • Very heat resistant
  • Used in cutting tools and drill-tip surfaces
  • At risk for development for interstitial lung disease and occupational asthma
  • Dx usually made by pathological examination rather than clinical evaluation. Interstitial pneumonitis, frequently giant-cell type and interstitial fibrosis.
  • Tx: Removal from further exposure. Consider empirical therapy with corticosteroids due to progressive nature of the disease

LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.
Metal-Induced Lung Disease

• Beryllium
  • Commonly used in ceramics, electronics, aerospace, and nuclear weapons/power industries.
  • Acute beryllium-induced pneumonitis – high intensity exposure
  • Chronic beryllium disease
    • Sensitization through cell-mediated (type IV) mechanism. Low level exposure. Phagocytosed by macrophages → beryllium-specific CD4+ T cells → release of cytokines → granuloma formation.
    • Granulomatous inflammatory disorder similar to sarcoidosis.
    • Usually only affects lungs but extrapulmonary involvement can occur.
    • Need to demonstrate beryllium sensitivity to confirm diagnosis.

LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.
Pneumoconiosis

- Group of interstitial lung diseases caused by the inhalation of certain dusts and the lung tissue’s reaction to the dust.
  - Asbestosis
  - Silicosis
  - Coal Worker’s Pneumoconiosis (CWP or black lung)
  - Other dusts:
    - Aluminum
    - Antimony
    - Barium
    - Graphite
    - Iron
    - Kaolin
    - Mica
    - Talc

https://www.cdc.gov/niosh/topics/pneumoconioses/default.html
Asbestosis

• Diffuse pulmonary fibrosis caused by the inhalation of excessive amounts of asbestos fibers.

• Ddx: Idiopathic pulmonary fibrosis and respiratory bronchiolitis
• *History of moderate to heavy asbestos exposure
• Clinical signs of interstitial fibrosis
  • End-inspiratory crackles in the lower zones
  • *Reticular-linear diffuse opacities in the lower zones of the lungs on radiological exam
  • Restrictive impairment of lung function
  • Usually, but not always, parietal pleural fibrous plaques and/or diffuse pleural fibrosis

Diffuse pleural calcification in a patient with asbestosis

Chest radiograph from a patient with asbestosis showing a combination of extensively calcified pleural plaques and interstitial fibrosis.
Malignant Mesothelioma

- Aggressive
- Debate over which types of asbestos cause it
- Treatment is palliative
- Amphibole group more potent inducers
Claire's pulls children's makeup kits over asbestos concerns

By Sandee LaMotte, CNN

Updated 7:16 PM ET, Fri December 29, 2017

Claire's has retained an independent lab to test its children's makeup products for asbestos.
Board Question

Which of the following is the most potent agent in the production of malignant mesothelioma?

A) Silica dust  
B) Chrysotile fibers  
C) Amphibole fibers  
D) Chromium
Board Question

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A) Silica dust
B) Chrysotile fibers
C) Amphibole fibers
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Board Question

Which of the following statements about asbestos-related disease is TRUE?

A) Asbestos exposure can produce chronic obstructive pulmonary disease
B) The radiologic findings specific for asbestos exposure is rounded atelectasis
C) The demonstration of asbestos bodies in the sputum is diagnostic proof of asbestosis
D) The synergism between smoking and asbestos exposure yields a 50-fold risk for bronchogenic carcinoma
Which of the following statements about asbestos-related disease is TRUE?

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D) The synergism between smoking and asbestos exposure yields a 50-fold risk for bronchogenic carcinoma.

Interstitial fibrosis may produce a reduction in vital capacity and impaired diffusing capacity, but not COPD. Asbestos bodies in the sputum are a sign of exposure, but not asbestosis. The profound synergism between cigarette smoking and asbestos exposure results in increased risk of bronchogenic carcinoma by 50-fold.
Silicosis

- Disease produced by inhalation of one of the forms of crystalline silica, most commonly quartz.
  - Simple (nodular) silicosis
  - Acute silicosis (silicoproteinosis)
  - Complicated pneumoconiosis (progressive massive fibrosis)

Silicosis

- At risk occupations/exposures:
  - Sandblasting
  - Quarrying
  - Stone dressing
  - Refractory manufacture
  - Foundry work
  - Mining
  - Pottery making
  - Abundant in soil as well

Simple Silicosis

- Exposure history
- Radiography: fine nodules, tends to be upper zonal but lower zones may be involved in severe cases
- Nodules: 1 cm or less. If larger, classified as complicated pneumoconiosis
- Usually asymptomatic
- Pulmonary function: usually normal, sometimes reduced vital capacity, possible chronic airflow obstruction
- Like asbestosis, long latency period. With extremely high level of silica exposure can develop accelerated silicosis

LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.
Simple silicosis

There is a profusion of small rounded densities, predominantly within the upper lung zones.
Accelerated/Acute Silicosis

- Results from exposure to relatively high levels of silica.
- Characterized by pulmonary edema, interstitial inflammation, and the accumulation within the alveoli of proteinaceous fluid rich in surfactant.

LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.
Acute silicosis (silicoproteinosis)

There is a bilateral alveolar filling process present in both lower lung zones.

Courtesy Dr. E. L. Petsonk.
Acute silicosis (silicoproteinosis)

There is prominent alveolar filling with eosinophilic proteinaceous material. Mild interstitial thickening is also present.

Courtesy of Dr. J Parker.
Complicated pneumoconiosis (progressive massive fibrosis)

- Coalescence and agglomeration of several smaller nodules. Profusion of nodular lesions increases and results in progressive massive fibrosis (PMF).
- Cavitation and extensive destruction of the lung parenchyma.
- PMF lesions greater than 2 cm in diameter.
Progressive massive fibrosis (PMF)

Patient with end-stage silicosis complicated by respiratory failure. There is upward retraction of hila, and the lower zones are hyperinflated.

Courtesy of Dr. J. Parker.
Silicosis complications

- Tuberculosis
- Rheumatoid complications
- Pulmonary hypertension
- Cor pulmonale
- Glomerulonephritis
- Bronchogenic carcinoma
Coal Workers

- CWP
  - Simple
  - Complicated
- Bronchitis
- Emphysema
- Caplan Syndrome
- Silicosis

Coal Worker’s Pneumoconiosis

• Simple CWP
  • Formation of black coal dust macules centered around the respiratory bronchioles, mostly in the upper lobes of the lungs.
  • Macules range from 1-6 mm in diameter.
  • Focal emphysema.

• Complicated CWP
  • Nodules may converge and coalesce to produce lesions larger than 2 cm with a fibrous nature. Turns into PMF.

Lung Cancer

**Occupational Exposure**
- Several occupational carcinogens
- Attributable risk 7.4% males and 3.1% females

**Environmental exposures**
- Outdoor particulate matter
- Residential radon exposure (attributable risk 7% in US)
- Environmental tobacco smoke
- Environmental exposure to occupational carcinogens

Lung Cancer

- Arsenic
- Asbestos
- BCME
- Beryllium
- Cadmium
- Chromium 6
- Silica dust
- Nickel

- Ionizing radiation
- Occupational exposure to strong inorganic acids
- Sulfur mustard
- Polycyclic aromatic hydrocarbons
- Soot
- Coal tar pitch
- Diesel exhaust
An epidemiological study of lung cancer among workers exposed to bis(chloromethyl)ether

- Epidemiological study of lung cancer carried out in dyestuff factories in Japan, Factory K and Factory U.
- Subjects: 35 men in each factory who had been exposed to BCME
- Results: Total 13 cases of lung cancer. SMR (standardized mortality ratio) for Factory K: 23.5 and Factory U: 17.9. Excess death due to lung cancer seen in those exposed to BCME.
  - Of 8 cancer cases:
    - Small cell carcinoma: 5 cases
    - Adenocarcinoma: 3 cases
    - Large cell carcinoma: 1 case
What did we learn today?

• How to approach and considerations of occupational lung diseases
  • **History**
  • Overview of occupational and environmental lung diseases
    • Occupational Asthma
    • Inhalation Fevers
    • COPD
    • Bronchiolitis Obliterans
    • Hypersensitivity Pneumonitis
    • Metal-Induced Lung Disease
    • Pneumoconioses
      • Asbestosis
      • Silicosis
      • CWP
    • Lung Cancer
  • Prevention!
Thank You!
And special thanks to Drs. Joseph Fedoruk and Catherine Boomus for their slides from previous years

References:


LaDou J, Harrison, R. Current Diagnosis & Treatment Occupational & Environmental Medicine, 5th Edition. 2014.


https://www.cdc.gov/niosh/topics/pneumoconioses/default.html
https://www.cdph.ca.gov/Programs/CCDPHP/DEODC/OHB/Pages/FlavoringLungDisease.aspx
http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/allergy/occupational-asthma/