Occupational lung disease
In the mining industry

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Objectives

• Review pathophysiology of various lung diseases
• Understand the relative occurrence/burden of these lung diseases in mining
• Improve identification and recognition of occupational lung diseases
• Understand that primary prevention plays a key role in managing these conditions
Importance of respirology issues in mining

• The lungs are prime route of exposure for many circumstances
• Mining involves a broad range of potential inhalation toxins
• Respiratory pathology is a likely consequence
• Work-relatedness of lung issues may not be recognized due to latency, varied etiologies, etc.
• Diseases may be forgotten due to emphasis on injury
Respirology basics

- Asthma
- COPD
- rhinitis
- Fibrotic lung disease (interstitial lung disease, pneumoconiosis)
- Pleural plaques
- mesothelioma
- Lung cancer
- Acute pneumonitis
- Inhalation exposures
- Irritant reactions
- Infectious disease
Etiology of diseases in the workplace

• Deterministic: the higher the exposure, the worse the disease
  • direct effects from radiation
  • hearing loss
  • pneumonitis
• Stochastic: the higher the exposure, the higher the probability of disease
  • Lung cancer
• Predilective: demonstrates predilection +/- dose-dependency
  • Allergic conditions (asthma, dermatitis, rhinitis)
WORK-RELATED ASTHMA
What is asthma?

- Chronic lung disease affecting over 3 million Canadians
- Three hallmarks:
  - Reversible
  - Airway inflammation (swelling)
  - Airway constriction (tightening)
- Brought on and aggravated by certain triggers (family history, allergy, allergens, infections, smoking, etc.)
Epidemiology
Work-related asthma is not uncommon (10-15%) but often under-recognized
Asthma in mining

- McHugh et al (2010) found that workers in mining had the highest prevalence of asthma compared to other occupations in the US
Classification
Work-related asthma (WRA)

Occupational Asthma

- Sensitizer-induced (allergic)
- Irritant-induced (reactive airways dysfunction syndrome: RADS)

Work-exacerbated Asthma (WEA)
Work-related asthma (WRA)

Occupational Asthma

Sensitizer-induced (allergic)

Work-exacerbated Asthma (WEA)

Irritant-induced (reactive airways dysfunction syndrome: RADS)
Sensitizer-induced occupational asthma

- Represents the majority of occupational asthma (>90%)

- Clinical features:
  - fulfils the classic criteria for an allergic response:
    - asthma usually occurs in a minority of those exposed
    - asthma develops only after an initial symptom-free period of exposure
  - latency period of sensitization may be weeks to many years
  - Symptoms worsen at work or shortly after work
  - Symptoms improve when away from work (initially – if exposure continues, improvement away may not happen)
Sensitizers

- There are over 300 known sensitizers, with more every year
  - **high molecular weight** – generally proteins
    - e.g. latex, flour, animal dander
    - more likely to act as antigens through an IgE mechanism
  - **low molecular weight** – generally chemicals
    - e.g. isocyanates, metals
    - pathophysiology of mechanism is not well understood
- at particular risk are those with general risk factors for asthma:
  - Atopy, co-existing infections
Potential sensitizers in mining

- Isocyanate and phenol-based resins have been used for tunnelling support and have been associated with asthma (Bertrand et al, 2007)
- Paint and cement was reported to cause asthma in gold miners (Cowie et al, 1996)
- Exposure to Rhizopus nigricans, a fungal contaminant, in a coal mine (Gembooa et al, 1996)
Work-related asthma (WRA)

Occupational Asthma

- Sensitizer-induced (allergic)
- Irritant-induced (reactive airways dysfunction syndrome: RADS)

Work-exacerbated Asthma (WEA)
Irritant-induced occupational asthma

• Represents a small fraction of OA (approx. 6%)
• Onset typically occurs within 24 hours of exposure to a large quantity of a respiratory irritant. That is, unlike sensitizer-induced OA, there is typically no latency period.
• Most will recover after a toxic inhalation injury; some do not
• There is persistence of symptoms beyond 12 weeks, possibly lasting years
• Pulmonary testing shows objective evidence of asthma
• There is some evidence to suggest that chronic, low-level exposure to irritants (dusts, gases, mists, fumes, smoke) that are irritating to the respiratory tract may cause asthma (Balmes 2002)
IIA in mining

- Piirila et al (1996) report on 9 miners who were accidentally exposed to sulfur dioxide in a mine explosion
- A number of these workers went on to develop a persisting airway hyperreactivity, consistent with a history of IIA
Work-related asthma (WRA)

Occupational Asthma

Sensitizer-induced (allergic)

Work-exacerbated / aggravated Asthma (WEA)

Irritant-induced (reactive airways dysfunction syndrome: RADS)
CAUSES

Work-Exacerbated Asthma
(also called work-aggravated asthma)

Exposure to workplace irritants or exertion at work may aggravate pre-existing or concurrent asthma, particularly in patients who have moderate or severe asthma, or who are uncontrolled, because they are not receiving optimal treatment.

Work-exacerbated asthma represented approximately half of work-related asthma seen in a Canadian clinic study (Tarlo, 2000).

People with pre-existing asthma are still susceptible to sensitizers in the workplace.

THIS IS COMPENSABLE IN ONTARIO
WEA in mining

- Mining is one of the most common industries that causes worsening of underlying asthma (Henneberger et al, 2002)
Diagnosis: a multi-step process

1. DIAGNOSE ASTHMA
2. SUSPECT WORK-RELATEDNESS
3. DETERMINE WORK-RELATEDNESS
Spirometry 101

- Measure of airflow and volume of lungs
- Most commonly consider FEV1 and FVC
Management of WRA

The three main components of the management of WRA are:
1. Treat the asthma as per the usual guidelines
2. Address issues of workplace exposure
3. Initiate compensation claim, if applicable
Management of WRA

- OA (sensitizer-induced)
  - Remove from exposure:
    - Longer duration of exposure leads to increased risk of permanence and increased severity of disease
    - Workers can react to very small amounts of exposure

- Irritant induced asthma (RADS)
  - Remove from work until symptoms resolve
  - Return to work should be considered a trial – may react to exposures for long period (some cases up to 2 years)

- Work Exacerbated Asthma (WEA):
  - Control exposure - engineering efforts, modified work
  - Respirator is not a solution
Management of WRA

• Initiate a compensation claim

• Sentinel health event: consider that others may be similarly affected

• All workers need education and information about managing their asthma, recognition of triggers and what to do about them ++ + support.

• Employers and workplace parties also need this information as well as support in determining how they will manage the worker and address exposure issues
Secondary prevention (early recognition)

Workplace – Occupational Health Program

- Medical Surveillance
  - Specific program to assess for health effects from specific exposures at pre-set intervals (e.g. annual, semi-annual)
    - e.g. isocyanates
    - Trend analysis – is there a group change? If yes, what is it due to?
- PFTs, symptom questionnaire
- Identification of a case of sensitizer-induced asthma should sound an alarm within the workplace – hygiene measures should be implemented to control exposure
COPD
COPD (chronic obstructive pulmonary disease)

- Also called emphysema, chronic bronchitis
- Similar to asthma but irreversible
- Often caused by smoking: occupational relationship is often ignored/unrecognized
- Compensated in smokers: attenuate compensation based on smoking
COPD and mining

- Cumulative dust exposure may cause COPD
- Sampatakakis et al (2013) found that mining dust was associated with greater risk of COPD
- Graber et al (2014) found elevated rates of COPD mortality in miners (HR = 1.84, 95% CI = 1.05-3.22)
- Mohner et al (2013) reported on declining lung function in miners
- Etc.
COPD and mining

- Oxman et al (1993) reviewed the epidemiology regarding occupational dust exposure and COPD
- They identified a number of studies reporting an association between dust exposure and obstructive lung impairment (COPD)
- 35 years of work at a dust level of 2 mg/m3 can lead to significant impairment in lung functioning
• WSIB threshold (40mg/m³-years) developed from British coal mining cohort (1988): corrected for smoking
• Higher risks had been reported in silica workers; this extrapolated table did not take into account Si effect of quartz mining
COPD and occupation

- Largely associated with “dust” levels: may occur in any industry but most common in foundries, mining, heavy industry, welding
- More recent cohort studies are implicating lower levels of dust (e.g. Santo Tomas, 2011)
- Synergy with smoking (Blanc et al, 2009): dust + smoking OR = 14.1 (95% CI = 9.33-21.2)
Clinical aspects of COPD

- Diagnosis depends largely on lung function testing
- Screening/early detection is of limited use
  - Once impairment is found, disease is usually well-established
- Management options:
  - Puffers
  - Oxygen
- PREVENTION IS KEY!
INTERSTITIAL LUNG DISEASE
Interstitial lung disease

- Scarring of lungs (interstitium)
- Also called pneumoconiosis, pulmonary fibrosis, restrictive lung disease
- Classic work-related lung diseases: asbestosis, silicosis,
- Any adult presenting with ILD should be asked about occupational exposures
- Usually strong dose-response: higher the exposure, the more chance of disease
Common types

- Asbestosis
- Silicosis
- Hard metal disease
- Berylliosis
- Hypersensitivity pneumonitis
- Coal worker’s pneumoconiosis
- Siderosis
- Aluminum oxide fibrosis
- Talc pneumoconiosis
Clinical aspects of interstitial lung disease

- Can be difficult to clearly diagnose
  - Based on radiographic (CT, CXR) findings
  - Based on history of exposure
  - Occasionally biopsy/autopsy may help
- Screening is of limited value
  - Once impairment is found, it is likely established
- Management is limited; there is no cure
  - Oxygen, lung transplant
  - PREVENTION IS KEY!
Epidemiology of pneumoconiosis in mining

- Annual incidence of 25.7 per 100,000 in mining industry (Meyer et al, 2001) in the UK
- Silicosis mortality rate dropped from 0.74 per million in 2001 to 0.39 per million in 2010
- Diesel Exhaust in Miners Study (DEMS):
  - Significantly elevated risk of pneumoconiosis (RR=12.20, 95% CI = 6.82-20.12)
Silicosis

- Industries: mining, foundries, construction, masonry, mining
- Typically nodular disease, predominance of upper lobes
- Progressive disease with no real cure
- Associated with tuberculosis: TB is 2 to 30x more common in those with silicosis (Cowie, 1994)
- Silica has been associated with connective tissue disorders (scleroderma, lupus, RA)
- CXR: symmetric nodules, initially upper zones
Sarcoidosis

- Granulomatous disease often causing lung lesions, including fibrosis, as well as other organ involvement
- Unlike other ILD, onset maybe more rapid
- Cause is unknown:
  - ? Infectious
  - ? Autoimmune
  - ? beryllium
## Asbestos: Types

<table>
<thead>
<tr>
<th>Serpentine</th>
<th>Amphibole</th>
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<tbody>
<tr>
<td>(93% of commercial use)</td>
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<td>Chrysotile</td>
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- **Serpentine** (93% of commercial use)
- **Amphibole** (7% of commercial use)
  - Actinolite, Amosite, Anthophyllite, Crocidolite, Richterite, Tremolite
Asbestosis

- Thought to occur from all subtypes of asbestos: fairly ubiquitous material
- Industries: construction (industrial, commercial, residential), mining, heavy industry, trades, insulation
- Long latency (20 to 40 years)
- Diffuse interstitial fibrosis with:
  - Restrictive pattern of disease on pulmonary function testing (but can see mixed pattern)
  - Impaired gas exchange
  - Progressive exertional dyspnea
  - Not a necessary pre-cursor of lung cancer (Finkelstein, 2010)
Case (pleural plaques)

- Mr. B. (76 years old) is referred by the family MD for pleural plaques found incidentally on CXR
- He worked as a millwright from 1956 to 1994 and describes extensive asbestos exposure
- He has no respiratory complaints
- Pulmonary function is normal:
  - FEV1 = 101% pred; FVC = 102% pred
Case (pleural plaques)

- He has questions:
  - Will he get cancer?
  - Does he need more tests?
  - Can he apply for compensation?
  - Should he be followed into the future?
Pleural plaques

- Marker of exposure
- *Not* asbestosis
- latency = 20-30 years
- prevalence = 3-14% or more
- usually incidental findings
- ? not associated with impairment
- does not prognosticate for mesothelioma

(Greillier, 2008)
Berylliosis (chronic beryllium disease)

- Industries: used for alloying:
  - aerospace, electronics, metalwork, welding, nuclear
- Found in the mining and extraction industry (Deubner et al, 2001)
- 3-10% of workers sensitized; difficult to establish safe level
- **Granuloma formation that mimics sarcoidosis**
- BeLPT (Sens = 0.6 to 0.7; Spec = 0.99) (Middleton et al, 2010)
- Management requires removal from Be exposure
- Be may also cause acute pneumonitis in high doses
Ribeiro et al, 2011

- Searched for CBD amongst 121 sarcoidosis patients
- 17/121 (14%) had beryllium exposure (alloying, construction, mining, nuclear)
- None had positive BeLPT (although false negative rates = 31.7% (Stange, 2004)
- Conclusions: need for careful history taking in sarcoid; Be exposure was not uncommon

- Fireman et al, 2004 did a similar study in Israel and found 3 of 47 sarcoidosis patients had CBD
- Muller-Quernheim, 2006 found that 34 of 84 Be-exposed sarcoidosis patients likely had CBD based on BeLPT
Hard metal disease

- From tungsten-carbide/cobalt alloy: extremely strong
- Occupations: grinding, rolling, cutting/tunneling tools, aerospace
- May be related to cobalt mining (Kerfoot et al, 1975)
- Prevalence = 0.7 to 13% (Fontenot, 2008)
- No clear dose-response reported
- Pathology = ? giant cell interstitial pneumonitis, desquamative interstitial pneumonitis
Idiopathic pulmonary fibrosis

• ATS Statement on IPF (2011):
  • Diagnosis of IPF requires:
    ▪ “Exclusion of other known causes of interstitial lung disease (ILD) (e.g. domestic and occupational environmental exposures...)”
    ▪ “presence of a UIP pattern on high-resolution computed tomography (HRCT) in patients not subjected to surgical lung biopsy”: similar to asbestosis, HP
    ▪ “accuracy of the diagnosis of IPF increases with multidisciplinary discussion between pulmonologists, radiologists, and pathologists experienced in the diagnosis of ILD”
  • “the natural history is variable and unpredictable”: unable to diagnosis IPF on clinical grounds
Are cases of IPF actually misdiagnoses of occupational conditions?


<table>
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<tr>
<th>Exposure</th>
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<tr>
<td>agriculture</td>
<td>1.65 (1.20, 2.26)</td>
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<tr>
<td>wood dust</td>
<td>1.94 (1.34, 2.81)</td>
</tr>
<tr>
<td>metal dust</td>
<td>2.44 (1.74, 3.40)</td>
</tr>
<tr>
<td>stone/sand</td>
<td>1.97 (1.09, 3.55)</td>
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<tr>
<td>metal dust</td>
<td>2.0 (1.0, 4.0)</td>
</tr>
<tr>
<td>talc</td>
<td>2.8 (0.7, 11.2)</td>
</tr>
<tr>
<td>animal dust</td>
<td>4.7 (2.1, 10.4)</td>
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LUNG CANCER
Lung cancer

- Attributable fraction to work: 6-29%
- Only a fraction of these are being reported in Ontario: 250 to 1200 new cases per year; 152 submitted in 2003
Lung cancer and mining

- The following are IARC Group 1 (carcinogenic to humans) carcinogens that may be found in mining:
  - Asbestos
  - Beryllium
  - Cadmium
  - Chromium
  - Diesel exhaust
  - Nickel
  - Radon
  - Silica
Lung cancer and mining

- Diesel Exhaust in Miners Study (DEMS), 2012
  - 12,315 miners in US
  - Elevated risk of lung cancer (RR=1.26, 95% CI = 1.09-1.44) and esophageal cancer (RR=1.83, 95% CI = 1.16-2.75)

- Rage et al (2014)
  - Uranium workers: elevated lung and kidney cancer rates

  - Radon exposures related to lung cancer
Radon

- decay product of Uranium 238
- Naturally occurring
- Two of radon’s decay daughters (Po214 and Po218) emit alpha particles
- Alpha particles are carcinogenic, but limited penetrance
- Radon becomes attached to dust particles, then are inhaled; they cause local lung damage leading to carcinogenesis
Mesothelioma

- Almost all are work-related (95%)
- Minimal exposure seems to be able to cause disease: *even brief, intermittent exposure*
- Many claims are being missed (Pichora, 2009)
Clinical aspects of cancer

- Early diagnosis is paramount if there is to be possibility of cure
- All cancers are not the same: prognosis/diagnosis/treatment varies considerably depending on cancer type and location
- Screening for cancers is very difficult; there are few studies that have clearly established the usefulness of tests, such as CXR, CT, blood tests, etc.
- Management is generally limited; outcomes are poor
- PREVENTION IS KEY!
Prevention
Prevention of respiratory disease

- Primary prevention is clearly the goal for effective management of respiratory disease in the mining industry.
- By the time disease is found, management is relatively futile.
- Early detection (such as medical screening) is usually very limited, except in the case of asthma.
PREVENTION CONTINUUM

Agent Exposure Early Symptoms Clinical Signs Disability
chemical odour annoyance cough/tight chest ↓ lung function asthma

Primary Prevention

Secondary Prevention

Tertiary Prevention

At the source Along the path At the worker

Medical surveillance Assessment of symptoms

Managing disability
KEY POINTS:

• Mining work involves potential exposure to many lung toxins
• The entire spectrum of disease may be seen in this industry
  • Asthma, fibrosis, cancer, COPD
• Understand the different lung diseases that may be encountered
• Prevention is clearly the most important management strategy
References / Resources

**Recommended Reading:**
Diagnosis and Management of Work-related Asthma: ACCP Consensus
http://chestjournal.chestpubs.org/content/134/3_suppl/1S.full.html
An Official ATS Proceedings: Asthma in the Workplace
Bernstein IL, et al. Asthma in the workplace… 2006
Asthmagen? UK Health and Safety Executive. 2001
http://www.hse.gov.uk/asthma/asthmagen.pdf

**Recommended Clinics:**
Occupational Health Clinics for Ontario Workers
www.ohcow.on.ca  (905) 549-2552
St. Mikes Department of Occupational and Environmental Health
www.stmichaelshospital.com/programs/occupationalhealth
(416) 864-5074

**Other** Ministry of Labour Health & Safety Contact Centre
www.labour.gov.on.ca  1-877-202-0008
Contact:
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With thanks to: