

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

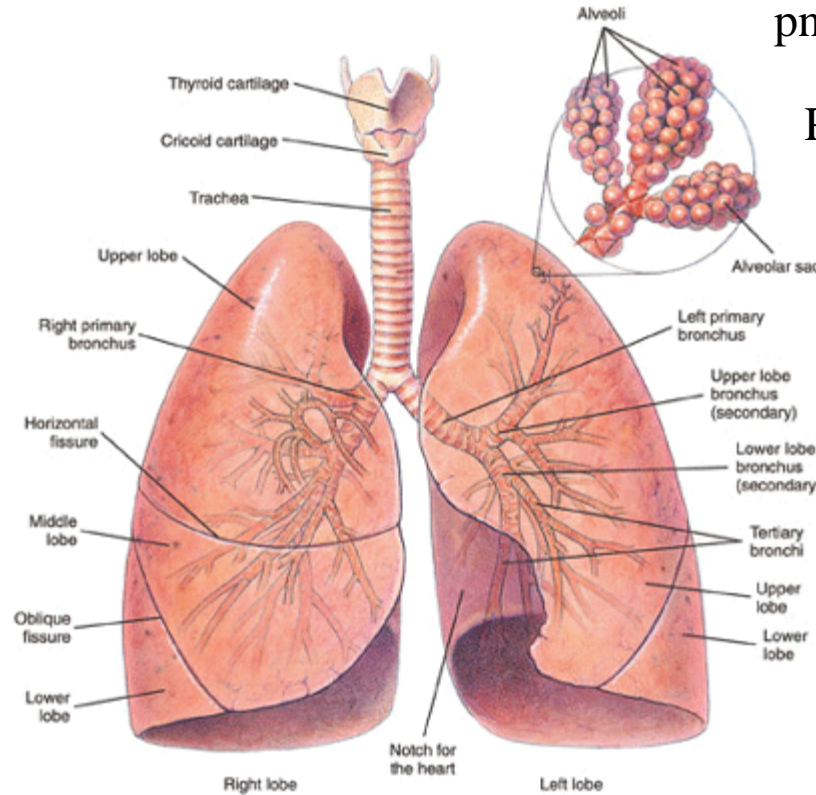


Respirology basics

rhinitis

Asthma

COPD



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)

```
graph TD; WRA[Work-related asthma (WRA)] --> OA[Occupational Asthma]; WRA --> WEA[Work-exacerbated Asthma (WEA)]; OA --> SI[Sensitizer-induced (allergic)]; OA --> IIR[Irritant-induced (reactive airways dysfunction syndrome: RADS)];
```

The diagram is a hierarchical flowchart. At the top level is a box labeled 'Work-related asthma (WRA)'. Two arrows point downwards from this box to two separate boxes: 'Occupational Asthma' on the left and 'Work-exacerbated Asthma (WEA)' on the right. From the 'Occupational Asthma' box, two more arrows point downwards to two sub-categories: 'Sensitizer-induced (allergic)' on the left and 'Irritant-induced (reactive airways dysfunction syndrome: RADS)' on the right. The text 'Occupational Asthma' is highlighted in red, while all other text is in black.

**Occupational
Asthma**

Work-exacerbated
Asthma (WEA)

Sensitizer-induced
(allergic)

Irritant-induced
(reactive airways
dysfunction syndrome:
RADS)

Work-related asthma (WRA)

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**Occupational
Asthma**

Work-exacerbated
Asthma (WEA)

**Sensitizer-induced
(allergic)**

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 (Gemboa et al, 1996)

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**Occupational
Asthma**

Work-exacerbated
Asthma (WEA)

Sensitizer-induced
(allergic)

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

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)

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graph TD; WRA[Work-related asthma (WRA)] --> OA[Occupational Asthma]; WRA --> WEA[Work-exacerbated / aggravated Asthma (WEA)]; OA --> SI[Sensitizer-induced (allergic)]; OA --> IIR[Irritant-induced (reactive airways dysfunction syndrome: RADS)];
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Occupational
Asthma

*Work-exacerbated /
aggravated Asthma
(WEA)*

Sensitizer-induced
(allergic)

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 patient 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 studies (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)

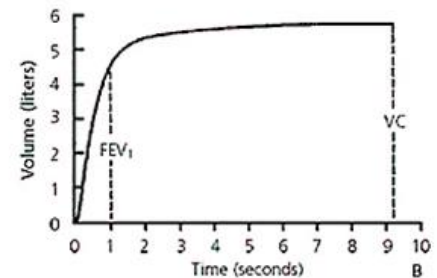
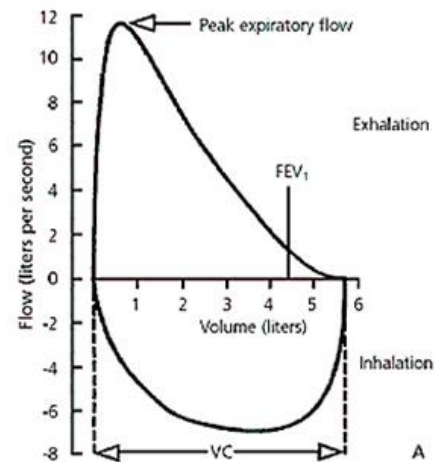
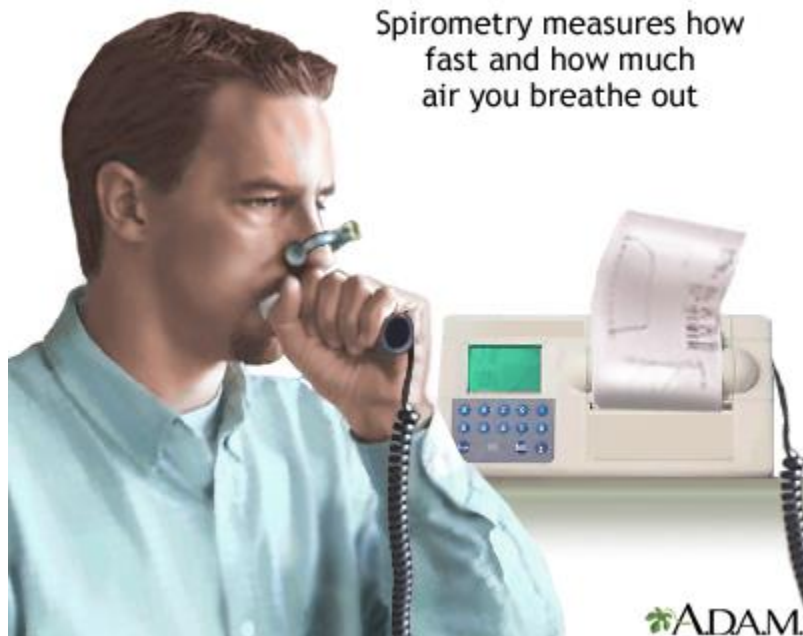


Diagnosis: a multi-step process

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

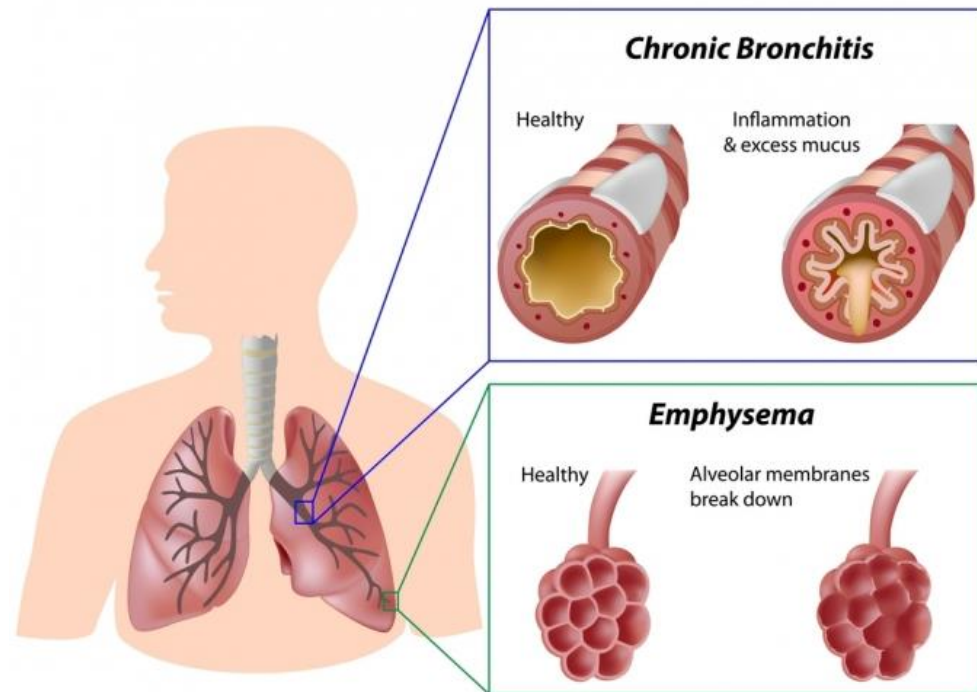
- Medical Surveillance
 - Specific program to assess for health effects from specific exposures at pre-set intervals (e.g. annual, semi-annual)
 - e.g. isocyanates
 - Trend analysis – is there a group change? If yes, what is it due to?
- PFTs, symptom questionnaire
- Identification of a case of sensitizer-induced 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

Chronic Obstructive Pulmonary Disease (COPD)



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



TABLE 1. Percentage Abnormality Attributable to Dust Exposure in a Cigarette Smoker with FEV₁, 65 Percent Predicted

Respirable Dust (mg/m ³)	Exposure (Years)			
	10	20	30	40
2	10	19	27	33
3	14	27	37	46
4	19	33	46	57
5	23	41	54	64
6	27	46	60	70

Original data from Marine *et al.*²

- 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

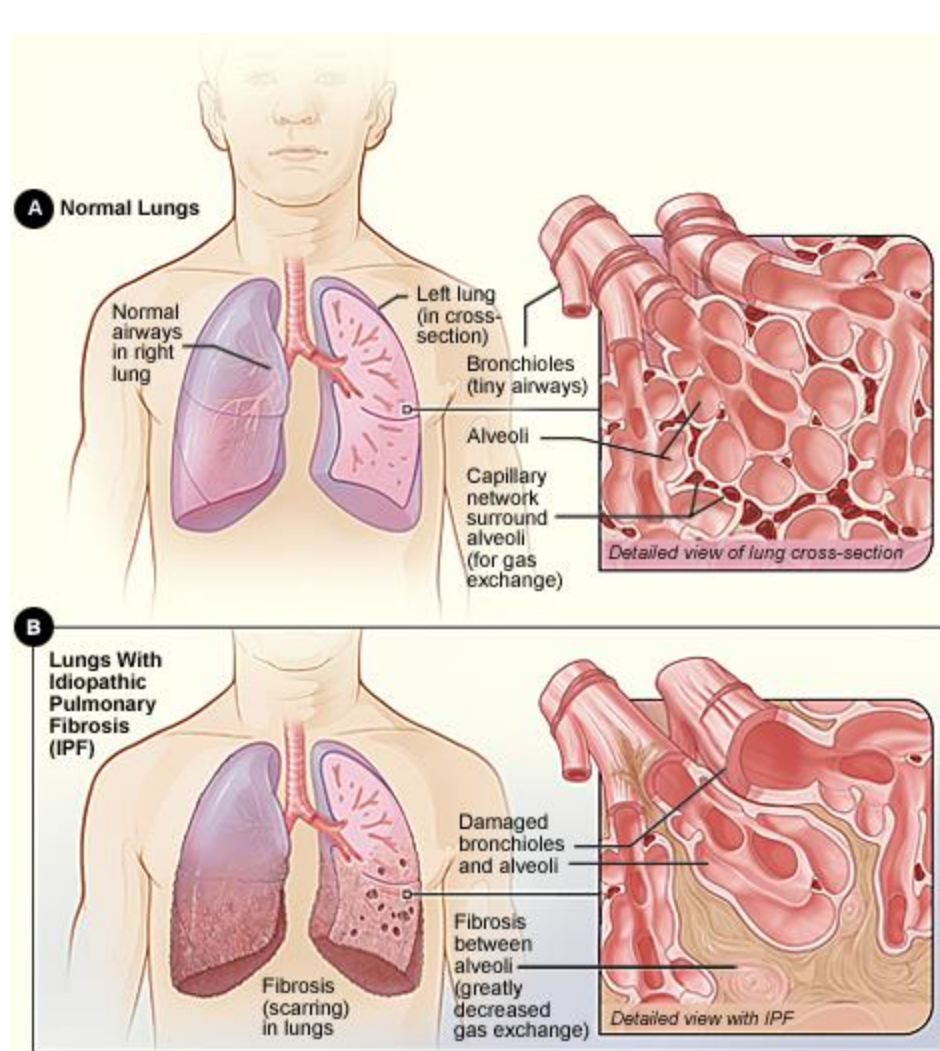
- Diagnosis depends largely on lung function testing
- Screening/early detection is of limited use
 - Once impairment is found, disease is usually well-established
- Management options:
 - Puffers
 - Oxygen
 - **PREVENTION IS KEY!**



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



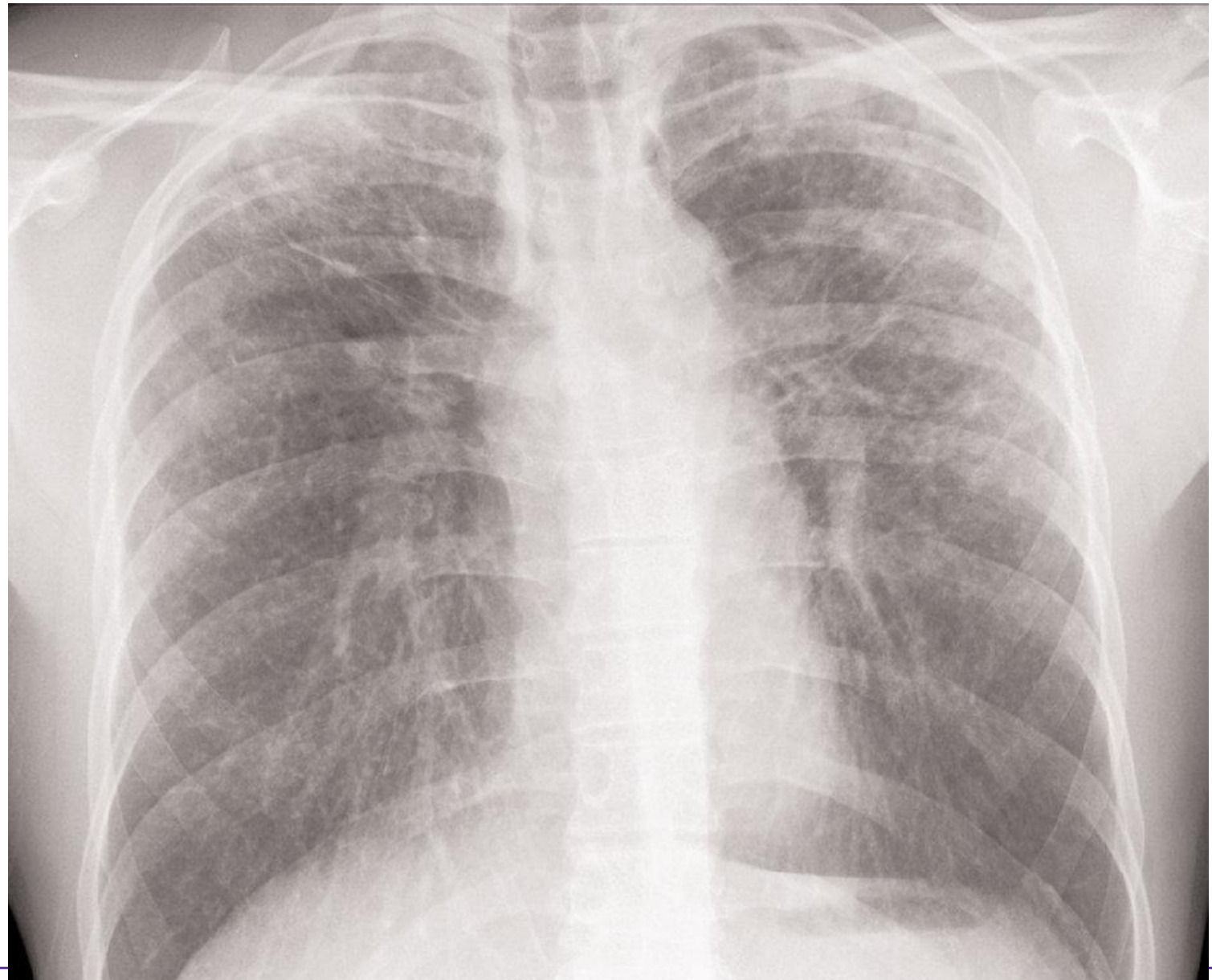
Clinical aspects of interstitial lung disease

- 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)



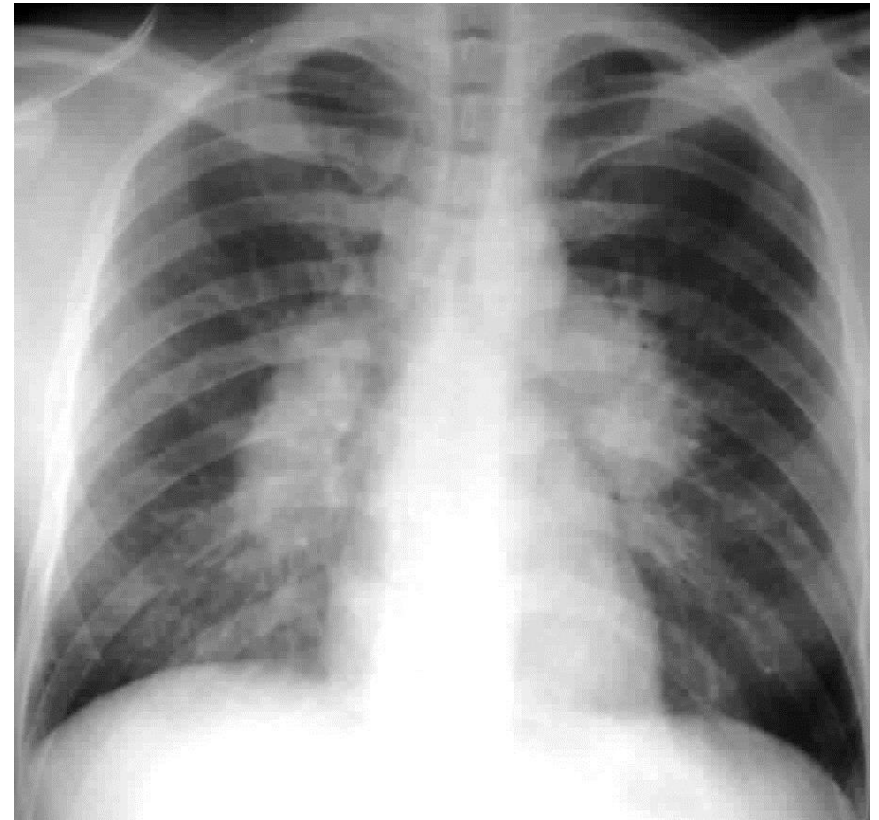
Silicosis

- 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

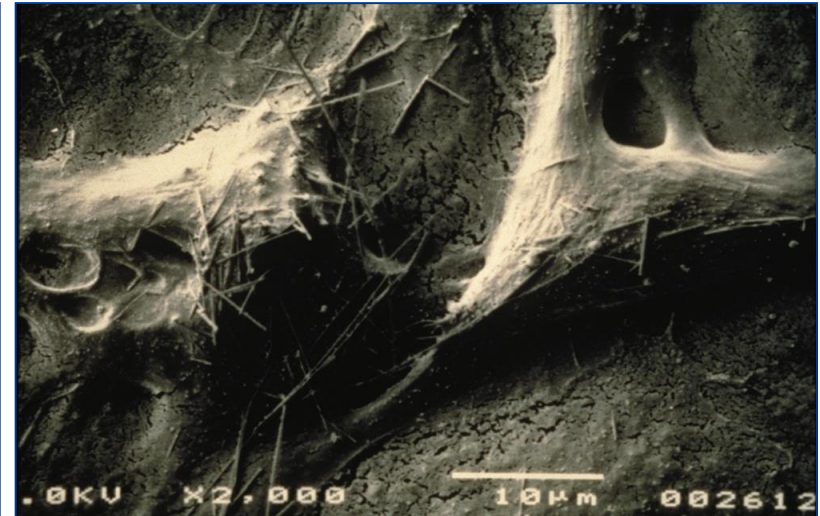


Sarcoidosis

- 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



Serpentine (93% of commercial use)	Amphibole (7% of commercial use)
Chrysotile	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)

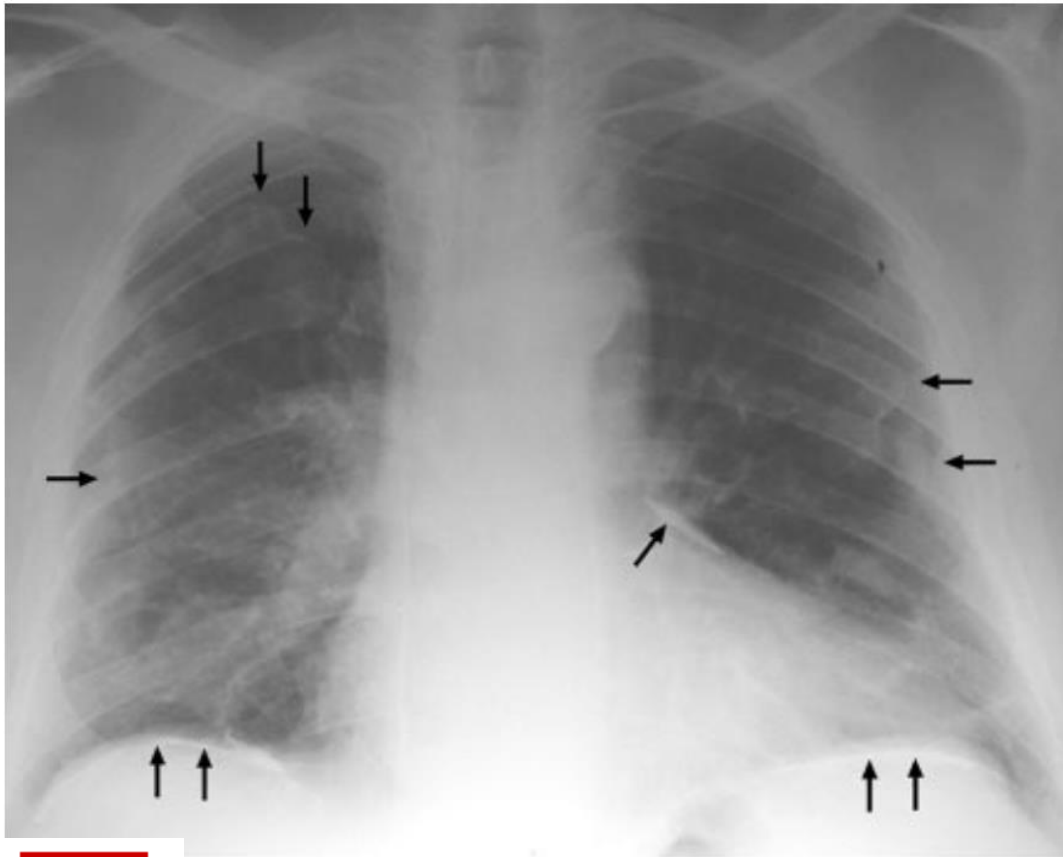
- He has questions:
 - Will he get cancer?
 - Does he need more tests?
 - Can he apply for compensation?
 - Should he be followed into the future?



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?

- Taskar and Coultas (2006): meta-analysis of occupational risk factors for IPF

Exposure	OR
agriculture	1.65 (1.20, 2.26)
wood dust	1.94 (1.34, 2.81)
metal dust	2.44 (1.74, 3.40)
stone/sand	1.97 (1.09, 3.55)



Are cases of IPF actually misdiagnoses of occupational conditions?

- Baumgartner et al (2000): occupational risk factors for IPF

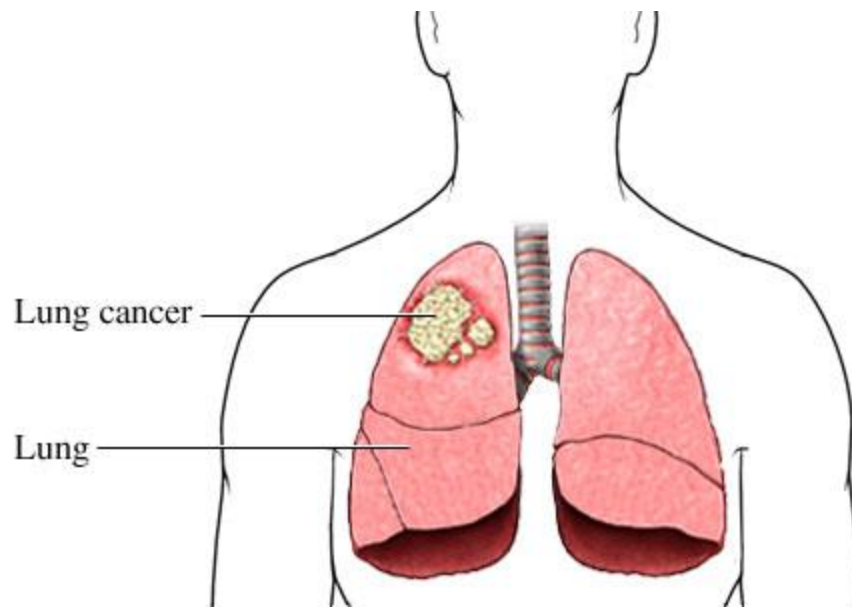
Exposure	OR
asbestos	1.1 (0.6, 1.9)
metal dust	2.0 (1.0, 4.0)
talc	2.8 (0.7, 11.2)
animal dust	4.7 (2.1, 10.4)



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
- Edwards et al (2014)
 - Radon exposures related to lung cancer



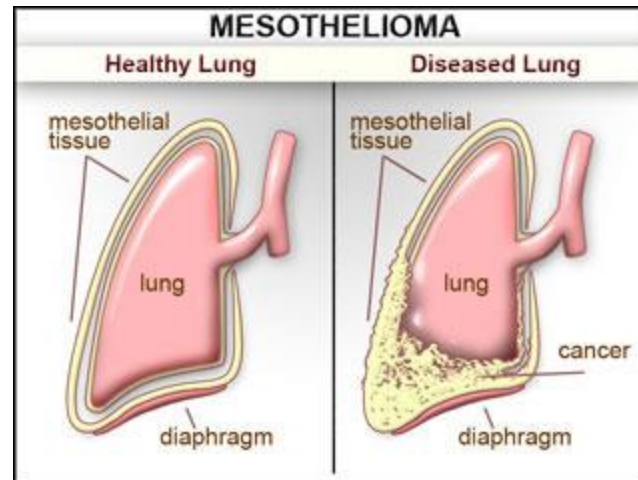
Radon

- 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

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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

<http://www.thoracic.org/statements/resources/eoh/asthma-in-the-workplace.pdf>

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:

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