Work-related asthma, 2016

Dr Ryan Hoy
Alfred Hospital, Melbourne
Monash Centre for Occupational and Environmental Health
Overview

- Asthma in the workplace
- Is it asthma?
- Work-exacerbated asthma
- Occupational asthma
- Diagnosis
- Prevention

Dr Ryan Hoy
Asthma in the workplace

- Work exposures can:
  - cause asthma (occupational asthma),
  - worsen asthma control (work-exacerbated asthma),
  - cause asthma-variants (eosinophilic bronchitis, aluminum potroom asthma)
  - cause mimics of asthma such as irritable larynx syndrome (ILS).

- Asthma can also affect ability to work and may need to be considered as part of assessing a workers fitness for a role eg occupational diving, military recruits, firefighters
Is it asthma?
Asthma epidemiology

- Asthma is a common disease.
  - In Victoria, 21% of people aged 18 years and over have ever been diagnosed with asthma
  - Over 2 million Australians have currently diagnosed asthma
    - 11%-13% of children
    - 9%-11% of adults
Asthma is a common disease in Australia

Dr Ryan Hoy
What else could it be?

- Vocal cord dysfunction (VCD)
- COPD
- Congestive cardiac failure
- Lack of fitness
- Pulmonary embolism
- Cystic fibrosis
- Bronchiectasis
- Bronchiolitis obliterans
- Hypersensitivity pneumonitis
- Aspiration – gastro oesophageal reflux
- Neoplasms - Central airway obstruction
- Extrathoracic airway obstruction
- Rhinosinusitis
- Hyperventilation syndrome
- Anxiety
- Respiratory tract irritation
What is asthma?

- A **chronic inflammatory disorder of the airways** in which many cells and cellular elements play a role (mast cells, eosinophils, T lymphocytes, neutrophils, and epithelial cells).

- In susceptible individuals, this inflammation causes **recurrent episodes** of wheezing, breathlessness, chest tightness, and cough, particularly at night and in the early morning.

- These episodes are usually associated with widespread but **variable airflow obstruction that is often reversible** either spontaneously or with treatment.

- The inflammation also causes an associated increase in **airway hyperresponsiveness** to a variety of stimuli. *GINA 1995*
Normal airway

- Intact surface pseudostratified ciliated columnar epithelium
- Indistinct underlying reticular basement membrane
- Few inflammatory cells
- Small amounts of bronchial smooth muscle
Mild asthma

- Goblet cell hyperplasia in epithelial cell lining
- Sub-basement membrane thickening
- Collagen deposition in submucosal area
- Cellular infiltrate
Severe asthma

- Small airway of a patient with severe asthma showing a combination of inflammation and remodelling.
- Inset: submucosal fibrosis and increase in smooth muscle.

Dr Ryan Hoy
Asthma diagnosis

- No gold standard for investigation of asthma

- **History**
  - Symptoms may vary widely.
  - Typical symptoms include some combination of wheezing, dyspnea, chest tightness and cough.
  - Chronic symptoms may also include sputum production.
  - Symptoms generally episodic.
  - History of atopic conditions (allergic rhinitis) or family history
Asthma triggers

- Symptoms provoked by a trigger
  - Allergens, especially indoor perennial allergens including house dust mite, cat, mould
  - Chronic rhinosinusitis – including post nasal drip
  - Respiratory tract infections
  - Respiratory irritants - perfumes, passive smoke, air pollution
  - Cold air
  - Drugs, especially beta blockers and aspirin
  - Emotion – including stress and laughter,
  - Exercise
  - Reflux
  - Occupation
Investigations

- Never assume dyspnoea is asthma without lung function tests.

- **Respiratory function tests** - identification of airflow obstruction that is at least partially reversible and possibly confirmation of bronchial hyper-responsiveness.
  - **Spirometry with bronchodilator testing**
    - Peak flow charts (for diagnosis if spirometry not available)
    - Bronchial provocation tests, eg methacholine, histamine or mannitol challenge.
    - Allergy tests, eg skin prick tests or RAST tests.
Spirometry

- Patient (effort), operator and interpreter dependent.

- Video showing correct technique for spirometry and other resources available at www.nationalasthma.org.au
What is spirometry?

Spirometry is a method of assessing lung function by measuring timed inspired and expired lung volumes. These values are used to:

1. Calculate how effectively and how quickly the lungs can be filled and emptied and
2. Measure the severity of airway obstruction by comparing them with predicted normal values

Dr Ryan Hoy
Desktop Electronic Spirometers

Dr Ryan Hoy
Box 3: Guide to performing spirometry

- The patient is seated (for safety) with upright posture
- Explain and demonstrate the manoeuvre
- Instruct the patient to inhale as deeply as possible until full (“Breathe in all the way”)
- Immediately after, instruct the patient to seal their lips tightly around the mouthpiece and exhale as rapidly as possible until no more air is able to be expelled (“Blast out” and “Keep going”)
- Always use vigorous demonstration and encouragement
- Repeat until a minimum of three acceptable manoeuvres (see box 4) are obtained
- From these, check for repeatability (that is, the two largest FVC and FEV₁ values are within 150 mL of each other), otherwise continue testing
- The manoeuvre with the largest FEV₁+FVC sum is reported for interpretation
- To assess reversibility, repeat spirometry 15 minutes after administration of bronchodilator (such as four separate 100 μg doses of a short acting β₂ agonist (salbutamol) administered by metered dose inhaler with a spacer)

A video showing correct technique is available at www.nationalasthma.org.au/health-professionals/spirometry-resources/spirometry-technique-video

Box 4 Important factors that affect acceptability of spirometry results

- Efforts must be free from cough, glottal closure, tongue occlusion, or mouth leak
- Ensure maximal inhalation and exhalation (exhalation should be ≥6 seconds (≥3 seconds in children aged <10 years) or until <25 mL is exhaled in 1 second)
- Eliminate hesitation at the start of expiration (back extrapolation volume must be <150 mL or <5% of FVC)
- The device must be regularly cleaned and calibrated to the manufacturer’s specifications

Ref: Jenkins C. BMJ 2012
Standard Spirometric Indices

- **FEV\(_1\)** - *Forced expiratory volume in one second:*
  The volume of air expired in the first second of the blow

- **FVC** - *Forced vital capacity:*
  The total volume of air that can be forcibly exhaled in one breath

- **FEV\(_1\)/FVC ratio:**
  The fraction of air exhaled in the first second relative to the total volume exhaled

Dr Ryan Hoy
## Classification Of Ventilatory Abnormalities by Spirometry

<table>
<thead>
<tr>
<th></th>
<th>OBSTRUCTIVE</th>
<th>RESTRICTIVE</th>
<th>MIXED</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FEV₁</strong></td>
<td>↓</td>
<td>↓ or Normal</td>
<td>↓</td>
</tr>
<tr>
<td><strong>FVC</strong></td>
<td>↓</td>
<td>↓ or Normal</td>
<td>↓</td>
</tr>
<tr>
<td><strong>FEV₁/FVC</strong></td>
<td>↓</td>
<td>Normal or</td>
<td>↓</td>
</tr>
</tbody>
</table>

---

Reversibility assessment

- Reversible airflow obstruction refers to acute response to bronchodilator

- **Preparation**
  - Patients should be clinically stable and free of respiratory infection.
  - Withhold inhaled short-acting bronchodilators in the previous six hours, long-acting beta-agonists in the previous 12 hours, or sustained-release theophyllines in the previous 24 hours.
Bronchodilator response

- Measure baseline spirometry (pre-bronchodilator). An FEV₁ <80% predicted and FEV₁/FVC ratio <0.70 shows airflow limitation (obstruction).
- Give the bronchodilator by metered dose inhaler (MDI) through a spacer device or by nebuliser.
- Give short-acting beta-agonist, at a dose selected to be high on the dose response curve (eg, 200 - 400mcg salbutamol from MDI and spacer – ie 2 to 4 puffs).
- Repeat spirometry 15 - 30 minutes after bronchodilator is given and calculate reversibility.
Bronchodilator response

- An increase in FEV$_1$ of more than 12% and 200mL is a significant bronchodilator response but is not diagnostic of asthma without clinical correlation.

- Complete reversibility of airflow obstruction or a large bronchodilator response is very suggestive of active asthma.

- Both COPD and asthma are not diagnosed by spirometry alone, need to be interpreted with clinical history.
Flow Volume Curves

<table>
<thead>
<tr>
<th></th>
<th>FEV1 (L)</th>
<th>FVC (L)</th>
<th>FER (%)</th>
<th>PEFR (L/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>1.52</td>
<td>4.08</td>
<td>37</td>
<td>4.4</td>
</tr>
<tr>
<td>Post Salb.</td>
<td>2.27</td>
<td>4.54</td>
<td>50</td>
<td>5.2</td>
</tr>
<tr>
<td><strong>Change</strong></td>
<td><strong>+49%</strong></td>
<td><strong>+11%</strong></td>
<td><strong>+13</strong></td>
<td><strong>+18%</strong></td>
</tr>
</tbody>
</table>

**Dr Ryan Hoy**
Asthma - diagnosis

- Normal spirometry does not exclude the presence of asthma.

- **Bronchial provocation testing:** assessment of airway hyperresponsiveness – the ability of the airways to narrow too easily and by too much in response to a range of direct and indirect stimuli.

- Administration of increasing doses of agonist or ‘stimulant’

- Measured by PD$_{20}$FEV$_1$
  - Provoking dose causing a 20% fall in FEV$_1$
Bronchial provocation tests

- Indirect challenges
  - Mannitol (Aridol®)
  - Hypertonic saline
  - Distilled water
  - Eucapnic voluntary hyperpnoea (EVH)
  - Exercise
  - Adenosine monophosphate (AMP)

- Direct challenges
  - Histamine
  - Methacholine

- Specific challenges
  - Occupational agent
Measurement of bronchial hyperresponsiveness

% fall in FEV1

Methacholine dose (μmol)

0 0.1 1 10

Severe

Moderate

Mild

Normal

Dr Ryan Hoy
These groupings are not mutually exclusive; e.g. OA can be followed by WEA
Work-related asthma

- **Occupational asthma** is a disease characterised by variable airflow limitation and/or hyperresponsiveness and/or inflammation due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace. (Asthma in the Workplace 3rd Ed.)

- OA is a preventable occupational disease caused by an exposure at the workplace.

- **Work-exacerbated asthma** (WEA) is pre-existing asthma (not caused by work) worsened by exposures at work such as fumes, dusts, temperature.
Work-related asthma (WRA)

Occupational asthma, caused by work (OA)

- Sensitizer-induced OA
- Irritant-induced OA (Including reactive airways dysfunction syndrome, RADS)

Work-exacerbated asthma (WEA)

These groupings are not mutually exclusive; e.g. OA can be followed by WEA
Work-exacerbated asthma

- WEA may range from a single transient exacerbation after an unusual exposure to daily work-related worsening of asthma that can mimic occupational asthma — may occur in up to 25% of working persons with asthma.

- WEA associated with more severe asthma, higher smoking rates, lower atopy, higher medication usage.

- Occupational exposures:
  1. inhaled agents: dusts, smoke, fumes, sprays, common environmental allergen encountered at work and mixtures of allergen and irritants;
  2. physical factors (such as temperature, humidity, physical exercise) and
  3. other factors, such as respiratory infections related to work, workplace stress.

Dr Ryan Hoy
Work-exacerbated asthma - diagnosis

Criterion 1: Pre-existing or concurrent asthma:

Criterion 2: Asthma–work temporal relationship: self reports of symptoms and medication use, or more objective serial peak flow recording

Criterion 3: Conditions exist at work that can exacerbate asthma

Criterion 4: Asthma caused by work (i.e., occupational asthma) is unlikely

Work-exacerbated asthma

Management:

- Aim to reduce/avoid exposure to exacerbating factor as far as practicable
- Assess risk of undertaking job, based on inherent requirements of the position
- If job does pose risk if asthma exacerbation, should develop plan for periodic review.
- Standard asthma management as per guidelines
- Asthma action plan – copy with OH&S, or safety officer
Case study 1

- John, 18 years old, has applied to Swell Furniture to be a furniture maker. He will work in the sawmill cutting and sanding wood. On his pre-employment form, he declares he has a history of asthma. The company refers him to you for assessment being mindful of their duty of care [OHS] and their compliance with disability discrimination legislation.

- What is the concern? How would you assess him?

- What work exposures are of concern? Is his risk of developing OA increased because of his history of asthma?

- Should he wear a dust mask? Should he have regular lung function tests?
Case study: comments

- Wood dust at work is a common exposure
- Asthma is a common disease: 21% of people aged 18 years and over have ever been diagnosed with asthma
- Wood dust is a respiratory irritant and a potential sensitiser (eg western red cedar)
- Assess risk based on asthma characterisation: active, well-control, typical exacerbating factors, high risk features, presence/severity of airflow obstruction or BHR.
- An exacerbation is unlikely to cause significant acute risk to John or co-workers. Unlikely firefighters of SCUBA divers.
Case study: comments

- Wood dust exposure is a factor for OA development. 5% in woodworkers.
- Typically sensitiser is low molecular weight, therefore atopy not a risk factor.
- Other exposures: formaldehyde, endotoxins, specific woods more asthmagenic.
- Dust mask lowest level of control in hierarchy. Dust control measures of primary importance.
- Regular clinical review would be advisable especially first 2 years. Not mandated.
Work-related asthma (WRA)

Occupational asthma, caused by work (OA)

Sensitizer-induced OA

Irritant-induced OA (Including reactive airways dysfunction syndrome, RADS)

Work-exacerbated asthma (WEA)

These groupings are not mutually exclusive; e.g. OA can be followed by WEA
Occupational asthma

- **Occupational asthma** is a disease characterised by variable airflow limitation and/or hyperresponsiveness and/or inflammation due to causes and conditions attributable to a particular occupational environment and not to stimuli encountered outside the workplace. (Asthma in the Workplace 3rd Ed.)
Epidemiology

- The ECRHS-II involving almost 7000 participants in 13 countries showed that the population attributable risk of occupational asthma was between 10 and 25%, equivalent to an incidence of 250 to 300 cases per 1 million people per year. (Kogevinas, 2007).

- Systematic analysis of six longitudinal general population-based studies, three case-control studies and eight cross-sectional analyses from seven general population-based samples noted a median PAR of 17.6%. (Toren K, 2009)

- Significant discrepancy between rates of asthma diagnosed as work related by health-professions (4.7% new asthma cases) and self reported work-related asthma (18.2%). (Mazurek, 2013)
Epidemiology

- Data from surveillance programs has demonstrated that occupational asthma has become the most prevalent lung disease in developed countries.

Dr Ryan Hoy
Work-related asthma (WRA)

Occupational asthma, caused by work (OA)

- Sensitizer-induced OA

Work-exacerbated asthma (WEA)

Irritant-induced OA (Including reactive Airways Dysfunction Syndrome, RADS)

These groupings are not mutually exclusive; e.g. OA can be followed by WEA
Sensitiser-induced OA

- A workplace sensitiser is an agent that induces asthma through a mechanism that is associated with a specific immunologic response.

- High molecular weight agents (HMW >10 kD, usually a protein or glycopeptide) can cause production of specific IgE antibodies and typical allergic responses.

- Following development of sensitisation very low levels of exposure can induce asthma.
<table>
<thead>
<tr>
<th>Agent</th>
<th>Workers at Risk of Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High-molecular-weight agents</strong></td>
<td></td>
</tr>
<tr>
<td>Animal allergens</td>
<td>Farmers, persons who work with laboratory animals, veterinarians</td>
</tr>
<tr>
<td>Plants</td>
<td>Greenhouse workers, farmers</td>
</tr>
<tr>
<td>Plant products (e.g., natural rubber latex)</td>
<td>Latex-glove makers and users, makers of other latex products</td>
</tr>
<tr>
<td>Cereals and grains</td>
<td>Farmers, grain workers, bakery workers</td>
</tr>
<tr>
<td>Other foods (e.g., milk powder and egg powder)</td>
<td>Food-production workers, cooks</td>
</tr>
<tr>
<td>Fungi</td>
<td>Office workers, laboratory workers</td>
</tr>
<tr>
<td>Enzymes</td>
<td>Laboratory workers, pharmaceutical workers, bakery workers</td>
</tr>
<tr>
<td>Insects</td>
<td>Farmers, greenhouse workers</td>
</tr>
<tr>
<td>Fish and crustaceans</td>
<td>Workers handling herring or snow crabs</td>
</tr>
<tr>
<td>Vegetable gums (e.g., guar and acacia)</td>
<td>Printers, including carpet makers</td>
</tr>
</tbody>
</table>
Low molecular weight chemicals (LMW)

- Some LMW have been associated with the production of specific IgE antibodies, such as complex platinum salts (platinum refineries, manufacture of cytotoxic drugs), acid anhydrides (hardeners in epoxy resins in chemical plants and in powder paints) and reactive dyes (used in textiles).

- Most LMW chemical mediated sensitisation however not through IgE mechanism and remains poorly understood.

- Diisocyanates (TDI, MDI, HDI) have been the most common cause of occupational asthma in many industrialised countries.

- Diisocyanates are important sensitisers that are used in the production of rigid or flexible polyurethane foam, hardeners in urethane spray paints and adhesives.
### Low-molecular-weight agents

<table>
<thead>
<tr>
<th>Substance</th>
<th>Occupational Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diisocyanates (e.g., toluene diisocyanate, hexamethylene diisocyanate, and methylene diphenyl diisocyanate)</td>
<td>Makers of rigid or flexible polyurethane foam, installers of polyurethane foam insulation, urethane spray painters, those who work with urethane adhesives or urethane molds in foundries</td>
</tr>
<tr>
<td>Acid anhydrides (e.g., phthalic anhydride, maleic anhydride, and trimellitic anhydride)</td>
<td>Makers of epoxy resins for plastics</td>
</tr>
<tr>
<td>Acrylic monomers</td>
<td>Chemical-industry workers, dental workers, aestheticians applying artificial nails</td>
</tr>
<tr>
<td>Wood dusts (e.g., from red cedar and exotic woods)*</td>
<td>Carpenters, sawmill workers, forestry workers</td>
</tr>
<tr>
<td>Complex platinum salts</td>
<td>Refinery workers, jewelry workers</td>
</tr>
<tr>
<td>Other metal salts (e.g., nickel chromium)</td>
<td>Metal-plating workers, welders of stainless steel</td>
</tr>
<tr>
<td>Biocides (e.g., glutaraldehyde and chlorhexidine)</td>
<td>Health care workers</td>
</tr>
<tr>
<td>Phenol-formaldehyde resin</td>
<td>Makers of wood products, foundry workers</td>
</tr>
<tr>
<td>Persulfates and henna</td>
<td>Hairdressers</td>
</tr>
<tr>
<td>Drugs (e.g., antibiotics)</td>
<td>Pharmaceutical workers, pharmacists</td>
</tr>
<tr>
<td>Aliphatic amines (e.g., ethylenediamines and ethanolamines)</td>
<td>Lacquer handlers, soldering workers, spray painters, professional cleaners</td>
</tr>
</tbody>
</table>

* Wood dusts can contain low-molecular-weight sensitizers, such as plicatic acid in red-cedar dust, but can also cause sensitization and promote the production of specific IgE antibodies to high-molecular-weight components (e.g., in
Work-related asthma (WRA)

Occupational asthma, caused by work (OA)

Work-exacerbated asthma (WEA)

Sensitizer-induced OA

Irritant-induced OA (Including reactive Airways dysfunction syndrome, RADS)

These groupings are not mutually exclusive; e.g. OA can be followed by WEA
Irritant-induced OA

- Reactive Airways Dysfunction Syndrome (RADS) Brooks, 1985

**Modified Criteria for RADS, Tarlo 2014**

- History of new-onset or recurrence of childhood asthma
- Symptoms onset related to one or more high level exposures
- Symptoms can begin >24 hours after exposure (may be several days)
- Exposures include very high concentrations of gas, fumes or spray, or highly irritating dust.
- Airway hyperresponsiveness or reversible airflow obstruction
- Symptoms persist for ≥ 3 months

Dr Ryan Hoy
World Trade Centre Disaster

- WTC responders – exposed to highly alkaline concrete dust

- At 1 year 16% of people with high exposure were considered to have irritant induced asthma.

- 9 years of follow-up of the WTC cohort 36.4% of those with respiratory symptoms had recovered.
Diagnosis

- Irritant-induced OA

- Appropriate clinical history of exposure and symptoms and demonstration of variable airflow obstruction or non specific BHR.

- Sensitiser-induced OA
  - Should be suspected in everyone with new onset asthma.
  - Refractory, severe or difficult asthma.
  - High risk industry
  - Job duties held when the asthma first developed

- Variable onset respiratory symptoms related to work including beginning of the work shift, toward its end, or even in the evening after working hours. Typically, remission or improvement during weekends and holidays

- Latency period ranging from weeks to years after the first exposure to the sensitiser is observed before the initial onset of work-related symptoms

- Occupational rhinitis prior to asthma symptoms with HMW agents

Dr Ryan Hoy
Diagnosis

- Clinical history alone has poor specificity for diagnosis of OA. (Malo, 1991).

- Investigation as soon as suspected, whilst still working.

3 steps to make the diagnosis of OA:

1. **Confirm presence of asthma!**

2. Identification of the workplace as the cause of the patient’s asthma.

3. Identification of a specific agent causing OA.

- Each investigation has limitations, which may be overcome by combination of tests.

- Protocols published by ACCP, BTS, etc.

Dr Ryan Hoy
Patient with asthmalike symptoms and work and clinical history compatible with occupational asthma

Assessment for asthma (on the basis of reversible airflow limitation, airway hyperresponsiveness, or both and immunologic testing if possible)

No evidence of asthma

Asthma

Patient is working

Patient is not working

Patient is working

No asthma

Investigate alternative conditions (e.g., rhinitis, hyperventilation, and vocal-cord dysfunction)

Specific inhalation challenge in the laboratory not available

Specific inhalation challenge in the laboratory available

Serial monitoring of PEF, with or without methacholine challenge, with or without sputum eosinophil counts at work and away from work, or specific inhalation challenge in the laboratory or at work if available

If occupational asthma is strongly suspected from history, a combination of objective evidence of asthma plus a positive skin test for specific IgE antibodies to the suspected agent has high predictive value for occupational asthma

Negative

Positive

Negative

Sensitizer-induced occupational asthma unlikely

Occupational Asthma

Non-Work-Related Asthma

Possible

Consider return to work

Impossible

Dr Ryan Hoy

Tarlo, 2014
Diagnosis

- Work and clinical history suggestive of occupational asthma

- Demonstrate variable airflow obstruction or non-specific bronchial hyper-responsiveness.

- Assess for the presence of specific IgE (skin prick tests, ImmunoCAP) when available.

- If still working perform serial peak flow monitoring for 4 weeks, include at least 1 week away from work.

- Serial methacholine challenges, performed towards the end of a working week then repeated at the end of a period away from exposure (>10 to 14 days). Threefold or greater change in PC20 between tests supportive of sensitiser-induced OA.
## Socio-economic consequences

<table>
<thead>
<tr>
<th>Country</th>
<th>Reference</th>
<th>No. of subjects</th>
<th>Follow-up (yr)</th>
<th>Work disruption (%)</th>
<th>Loss of income (% of workers)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UK</td>
<td>Gannon, 1993</td>
<td>112</td>
<td>Median: 1.4</td>
<td>35%</td>
<td>Exposed: 44% Unexposed: 74%</td>
</tr>
<tr>
<td>Canada, BC</td>
<td>Marabini, 1993</td>
<td>128</td>
<td>Mean: 4.8</td>
<td>41%</td>
<td>NA</td>
</tr>
<tr>
<td>Canada, Qc</td>
<td>Dewitte, 1994</td>
<td>134</td>
<td>Range: 2-5</td>
<td>25%</td>
<td>NA</td>
</tr>
<tr>
<td>UK</td>
<td>Cannon, 1995</td>
<td>87</td>
<td>5</td>
<td>39%</td>
<td>55%</td>
</tr>
<tr>
<td>France</td>
<td>Ameille, 1997</td>
<td>209</td>
<td>Mean: 3.1</td>
<td>34%</td>
<td>46%</td>
</tr>
<tr>
<td>USA</td>
<td>Gassert, 1998</td>
<td>55</td>
<td>Mean: 2.6</td>
<td>69%</td>
<td>NA</td>
</tr>
<tr>
<td>UK</td>
<td>Ross, 1998</td>
<td>770</td>
<td>1.5-5.5</td>
<td>37%</td>
<td>NA</td>
</tr>
<tr>
<td>Belgium</td>
<td>Labranos, 2002</td>
<td>86</td>
<td>Median: 3.3</td>
<td>38%</td>
<td>62%</td>
</tr>
<tr>
<td>Finland</td>
<td>Piirila, 2005</td>
<td>213</td>
<td>Mean: 10</td>
<td>14%</td>
<td>NA</td>
</tr>
</tbody>
</table>

Adapted from: Vandenplas et al., Eur Respir J 2003;22:689
Figure 1.1  Cycle of exposure and disease in occupational asthma

Person Starts Job

Exposure To Asthmagen

Develops Occupational Asthma

Occupational Cause Not Recognised

New Person Starts Job

Step 1

Step 2

Step 3

Step 4

Step 5

Step 6

Person Leaves Job

Continuing Exposure Means Ineffective Management Of Asthma

Dr Ryan Hoy
**ASTHMA**

**FEV₁ decline in occupational asthma**

W Anees, V C Moore, P S Burge


**Figure 1** Model of change in FEV₁ over time in response to exposure and removal from exposure.

Dr Ryan Hoy
Management

- Evaluate symptomatic workers early and obtain an accurate diagnosis
- Remove workers from further exposure to the causative agent after confirmed diagnosis.
- Control other triggers and pharmacological management as per guidelines
- Assistance with workers compensation claim may be required
- Monitor patients asthma in future work locations to ensure safe work placement.

Dr Ryan Hoy
Prevention is possible
Conclusions

- Ask all patients what they do for work.
- Suspect work may influence asthma either as a cause or aggravating factor.
- Clinical history alone has poor specificity for OA, objective evaluation of lung function is required.
- Early intervention has the best prognosis
- OA is a preventable cause of asthma
Case study 2

Farid, 18 years of age, presents with almost daily symptoms of itchy eyes, stuffy nose, cough, shortness of breath and wheeze. He says he wakes most days with a wheeze and describes frequent use of salbutamol. He has recently been on holiday and felt better during that time. He has a past history of seasonal hay fever.

On further questioning, Farid reveals that he has been working in a bakery for 12 months. One of his roles is to weigh the flour. His nasal and eye symptoms started 3 months ago, followed by the chest symptoms 2 months later. His symptoms improve modestly over the weekend and recur within minutes of starting work.

- How would you proceed to investigate and diagnose him?
- How would you manage him?