

Obstructive Respiratory Diseases of Occupational Origin: Asthma and Chronic Obstructive Pulmonary Disease



PREVENTION. CARE. RECOVERY.

Te Kaporeihana Āwhina Hunga Whara

»» *A distillation of best practice reflecting ACC's current position*

»» MAY 2007

- It is estimated that 15% of both chronic obstructive pulmonary disease (COPD) and adult asthma can be attributed to occupational exposures.
- Within certain occupational subgroups of the population the incidence of occupational disease is much higher.
- More than 250 agents have been identified as causes of occupational asthma, while exposures to vapours, gases, dusts and fumes in the workplace contribute to the development of COPD.
- Potential occupational causes should be considered in all presenting cases of obstructive respiratory disease and in all new adult asthma cases.
- Occupational diseases are preventable and can be avoided by controlling exposure to causal agents.
- The recognition and control of exposure are the most important means of managing the patient's condition.

Introduction

It is estimated that 15% of both chronic obstructive pulmonary disease (COPD) and adult asthma can be attributed to occupational exposures.¹ Within the New Zealand population, the Asthma and Respiratory Foundation estimates that 15% have asthma, and 10% have COPD. This has social and financial implications for society, with work absenteeism, increased use of medical care, and reduced quality of life. As with all occupational diseases, controlling exposure to causal agents can prevent these conditions.

Definitions

Asthma is a heterogeneous chronic inflammatory disorder of the airways involving airflow limitation that is often reversible, which results in recurrent episodes of symptoms such as wheezing, breathlessness, chest tightness, and cough.² There are two distinct asthma phenotypes, with about one half involving allergic mechanisms and the other half involving non-allergic.³

Occupational asthma is asthma induced by exposure to airborne dusts, vapours or fumes in the work environment.⁴ This includes new-onset adult asthma and workplace exacerbation of pre-existing asthma, which was experienced by 23% of asthmatic adults in one US health maintenance organisation.⁵ New-onset asthma results from either an immunological response to sensitising agents (after a latency period), or an inflammatory response to irritants or inflammatory agents after single or multiple exposures.¹ Reactive airways dysfunction syndrome (RADS) is an acute form of asthma that may arise following a single high-level exposure to inhaled irritants.⁶

COPD is characterised by airflow obstruction and inflammation of the peripheral airways and lung parenchyma, in which the airflow limitation is not fully reversible. It encompasses clinical entities such as emphysema and chronic bronchitis. Patients typically present with a chronic productive cough, dyspnoea, and wheezing.⁷ Diagnoses of asthma and COPD may overlap where there is both a fixed and a reversible component of airflow obstruction.

Epidemiology

Asthma: There is strong evidence of associations between occupation and asthma, although it is not always clear whether these exposures are initiating or exacerbating pre-existing asthma.

A recent review of New Zealand studies of occupational asthma⁸ found elevated risk for farm workers; bakers, food processors and mussel openers; laboratory technicians; wood, sawmill and plywood mill workers; and welders. Cases notified to the Department of Labour during the 1990s identified causal exposures including isocyanates, avian proteins, milk protein, mussel protein, flour, western red cedar/ unspecified wood dusts, formalin/formaldehyde, glutaraldehyde, epoxy resins, chlorine-based cleaners, organophosphates, ozone, aluminium smelting, welding fume, colophony solder, detergent enzymes, irritant metal dusts, acrylates, dibutyl phthalate, polyvinyl chloride fume, and polyurethane foam and fume.^{9,10} More than 250 agents have been identified as causes of occupational asthma, see: www.remcomp.com/asmanet/asmapro/asmawork.htm www.state.nj.us/health/eoh/surweb/wra/agents.shtml

COPD: Cigarette smoking is the predominant cause, although it does occur in people who have never smoked, and there is strong evidence of risks associated with occupational exposures to vapours, gases, dusts, and fumes.^{11,1} At-risk occupations include:

- construction and demolition (asbestos/other fibrous materials, solvents, pigments, ceramics cements, wood dust, welding fume, metal and polymer fume, vehicle exhaust, and asphalt)
- mining, smelting and mineral processing (similar to construction, plus ore-containing materials)
- manufacturing of complex materials (rubber, furniture, clay, or ceramic products)
- food processing and animal confinement (complex mixtures of both organic and inorganic substances)
- maintenance workers (solvents, metal fume and dust, mineral oil products, organic mixtures and temperature extremes)
- firefighters and emergency response workers (complex mixtures including combustion products)
- general labourers.

A New Zealand population-based study found increased risk (independent of smoking) in bakers, other food processors, chemical processors and spray painters. Risks were also significantly elevated in workers who reported being “ever-exposed” at work to vapours, gases, dusts and fumes, with an estimated 19.3% of COPD being attributable to these work exposures.¹²

Maori and Pacific peoples are at increasing risk of occupational lung diseases as employment patterns change. This occupational exposure to agents, together with the greater rates of smoking amongst Maori, increases the overall incidence of obstructive lung disease in these subgroups.

Diagnosis

Potential occupational causes should be considered in all presenting cases of obstructive respiratory disease, particularly in cases of new-onset asthma in adults or of COPD in patients with a history of workplace exposure to known risk factors.

Specific guidelines exist for the diagnosis of occupational asthma^{13,6} and COPD,⁷ which rely on confirming the presence of symptoms and establishing a plausible relationship with work exposures. For occupational asthma a clinical history will help establish any temporal pattern between the occurrence of symptoms and work schedules, days off, and any specific jobs or exposures. A detailed work history is vital to establish both occupational history and exposure to specific asthma-related agents. The use of peak flow diaries (while at work and away from work) is a most valuable tool accessible to all practitioners in considering occupational asthma. An occupation-related COPD diagnosis will also depend on a work history implicated as a cause in the epidemiological literature.

Treatment and Management

The treatment of obstructive respiratory diseases of occupational origin should follow the protocols in best practice guidelines for managing asthma and/or COPD. However, the recognition and control of exposure to the causative agent is the most important means of managing the patient's condition. This might be difficult, however, when the causal agent and the concentration that initiates or contributes to the condition is unknown. An early diagnosis of the work-relatedness of asthma in particular, if accompanied with reduction or elimination of exposures, will improve the patient's long-term prognosis. Furthermore, the reduction or elimination of workplace exposures will prevent aggravation of pre-existing asthma, or the development of new-onset asthma, in other workers.

Issues Relevant for ACC

Patients who have COPD or new-onset asthma as a result of their employment will be considered for ACC cover.

References

1. Balmes J, Becklake M, et al. American Thoracic Society Statement: Occupational contribution to the burden of airway disease. *Am J Respir Crit Care Med* 2003;167:787-97.
2. Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention 2006. www.ginasthma.org.
3. Pearce N, Pekkanen J. How much asthma is really attributable to atopy? *Thorax* 1999;54:268-72.
4. Francis H, Prys-Picard C, et al. Defining and investigating occupational asthma: a consensus approach. *Occup Environ Med* 2006; 63:028902.
5. Henneberger P, Derk S, et al. The frequency of workplace exacerbation among health maintenance organisation members with asthma. *Occup Environ Med* 2006; 63:551-7.
6. Chan-Yeung M. Assessment of asthma in the workplace. ACCP consensus statement. *American College of Chest Physicians. Chest* 1995;108:1084-1117.
7. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease 2006. www.goldcopd.org.
8. Driscoll T, Mannelte A, et al. The burden of occupational disease and injury in New Zealand: Technical Report. NOHSAC: Wgn, 2004.
9. Walls C, Crane J, et al. Occupational asthma and other nonasbestos occupational respiratory diseases notified between 1993 and 1996. *N Z Med J* 1997; 110: 246-9.
10. Walls C, Crane J, et al. Occupational asthma cases notified to OSH from 1996 to 1999. *N Z Med J* 2000; 113: 491-2.
11. Kennedy S. Agents causing chronic airflow obstruction. In: Harber P, et al, eds. *Occupational and environmental respiratory disease*, 1st ed. St Louis: Mosby-Year Book; 1996.p. 433-49.
12. Fishwick D, Bradshaw L, et al. Chronic bronchitis, shortness of breath, and airway obstruction by occupation in New Zealand. *Am J Respir Crit Care Med* 1997; 156: 1440-6.
13. Guide to the Management of Occupational Asthma 1995. Occupational Safety and Health Service, Department of Labour, Wellington.