

ALLERGIES IN THE WORKPLACE

AN APPROACH TO WORK-RELATED ASTHMA IN THE SOUTH AFRICAN SETTING

Mohamed F Jeebhay, MB ChB, DOH, MPhil (Epi), MPH (Occ Med), PhD

Centre for Occupational and Environmental Health Research, School of Public Health and Family Medicine, and Occupational Medicine Unit, Department of Medicine, University of Cape Town, South Africa

ABSTRACT

Work-related factors, depending on the industry, can be responsible for up to 25% of all adult asthma cases. Despite this, work-related asthma (WRA) remains under-recognised, poorly diagnosed and managed, and inadequately compensated in South Africa. WRA should be considered in all adult patients with new-onset or worsening asthma through taking an appropriate history. The condition can be broadly classified into occupational asthma (OA) due to an allergic or irritant cause, and another entity called work-exacerbated asthma (WEA). OA in South Africa is commonly caused by isocyanates, cereal flour/grain dust, natural rubber latex gloves and cleaning agents. RADS (reactive airways dysfunction syndrome) is caused by exposure to high levels of irritant compounds (e.g. chlorine, ammonia, sulphur dioxide, fire smoke). WEA is due to exacerbation of pre-existing asthma to workplace exposures (e.g. dust, smoke, fumes, cleaning agents) and physical factors (e.g. cold air, strenuous work). Early diagnosis and avoidance of further exposure are the cornerstones of patient management for WRA. All three major phenotypes of WRA are compensable in South Africa. Assisting the patient with a workers' compensation claim will ensure preservation of income and improved quality of life of affected workers.

INTRODUCTION

Work-related factors, depending on the industry, can be responsible for up to 25% of all adult asthma cases. International reviews suggest that the median proportion of adult cases of asthma attributable to occupational exposure is 16% and for work-exacerbated asthma (WEA) around 10%.^{1,2} However, work-related asthma (WRA) remains under-recognised, poorly managed and inadequately compensated.^{3,4} Global estimates (2005) indicate that 38 000 occupational asthma deaths and nearly 1.6 million DALYS (disability adjusted life years) are due to exposure to occupational airborne particulates.⁵

DEFINITION

WRA can be broadly categorised as (i) occupational asthma (OA) or asthma caused by specific agents in the workplace, and (ii) work-exacerbated asthma (WEA) or pre-existing asthma worsened by moderate-to-low level workplace exposures (e.g. dust, smoke, fumes, sprays, cleaning agents) and physical factors (e.g. cold air, humidity, strenuous work) (Fig. 1).²

There are two major forms of occupational asthma: (i) allergic (sensitiser-induced) asthma characterised by a latency period required for developing allergic sensitisation to high-molecular-weight (HMW) proteins prior to the development of symptoms; and (ii) irritant-induced (non-immunological) asthma characterised by rapid onset of asthma following single or multiple exposures to high levels of irritant low-molecular-weight (LMW) chemicals (e.g. chlorine and glutaraldehyde spills, smoke from fires, bleach and ammonia mixtures, sulphur dioxide and chlorine gas). Among these two major patterns, much higher proportions (70-90%) of allergic compared to irritant-induced OA are reported.³

EPIDEMIOLOGY

The reported mean annual incidence of occupational asthma in developing countries is less than 2 per 100 000 compared with very high rates of up to 18 per 100 000 in Scandinavian countries.³ This low figure is attributed to uneven industrial development, under-recognition and the relatively under-developed or non-existent surveillance systems in most countries resulting in a large proportion of cases being unreported. Despite the relatively lower incidence (1.8/100 000) in South Africa, provincial differences exist with a much higher incidence reported in the Western Cape (2.5/100 000), comparable with the USA and other European countries.⁶

High-risk industries and occupations

The patterns of WRA are quite diverse in various countries, demonstrating an array of economic activities associated with the disease. However, there are some common industries and occupations that appear to be consistently associated with a higher incidence of OA.⁷ These include bakers and pastry makers, spray painters especially in the car manufacturing industry, healthcare workers and platinum-exposed workers (Table I, Fig. 2).⁶

In South Africa, the patterns of WRA reported in epidemiological studies are quite diverse, demonstrating an array of economic activities associated with the disease (Table II).^{9,10,11,12} A large proportion of epidemiological studies have been conducted in various food processing workplaces, reporting a much higher prevalence of work-related allergic asthma in grain (wheat) milling plants (17%) and bakeries (13%) than in animal processing plants (fish 2%, poultry 12%) and table grape farmworkers (6%). These farm workers were found to be four times more likely to be pesticide crop sprayers. A much higher prevalence of WRA (10-30%) has also been reported among workers handling isocyanates in automotive repair shops and in chemical processing activities. Among the chemical processing and refining plants, platinum refineries appear to cause a much higher disease burden (cumulative index (CI) = 41%) than vanadium plants (CI = 11%). The prevalence of WRA in wood-working operations such as sawmills and furniture plants appears to be much lower (3-7%). Notably absent are epidemiological studies on irritant-induced asthma, despite the presence of large industries where high-risk exposures may be more

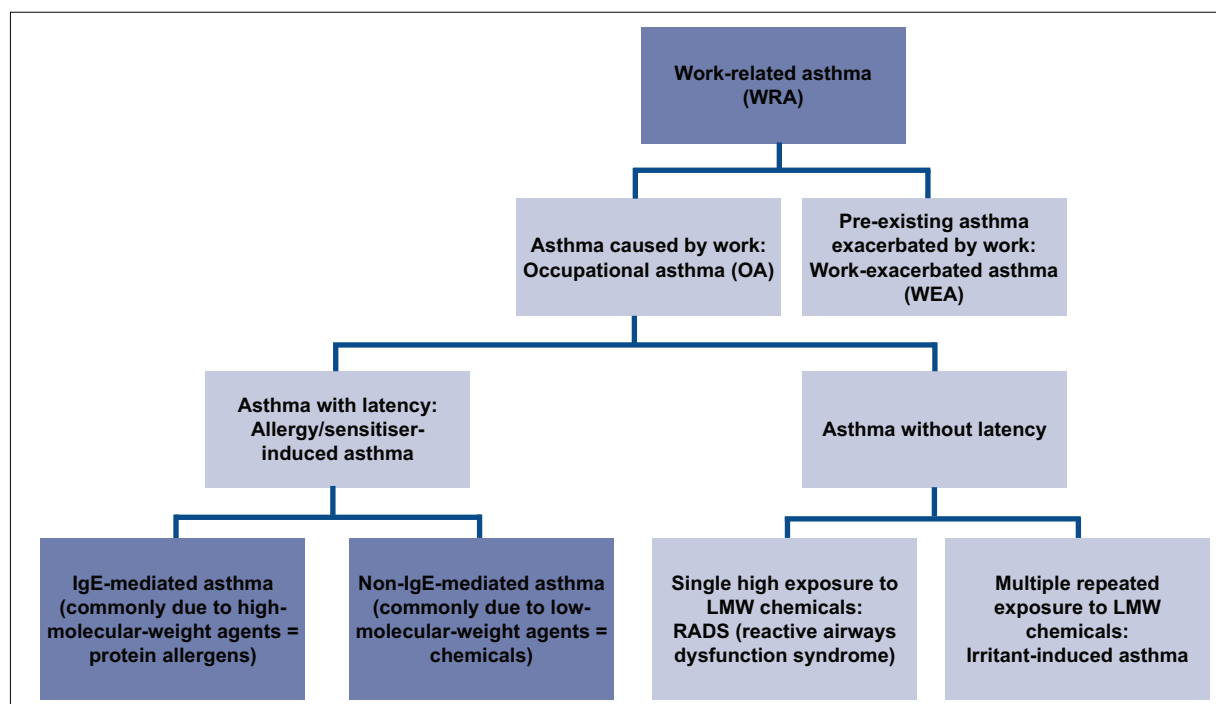


Fig. 1. Classification of work-related asthma.

prevalent. As for studies on WEA, recently published studies of supermarket bakery workers and dental healthcare workers reported a prevalence of 3% and 4% respectively.^{11,12}

CAUSATIVE AGENTS

More than 350 agents are known to cause OA, broadly classified into HMW substances (proteins) and LMW substances (chemicals).² In most industrialised countries the main causes of asthma include isocyanates, cereal flour/grain dust, welding fumes and wood dust (Table I).³ Exposure to animal epithelia, hairs and secretions is commonly reported among laboratory animal workers and agricultural workers. Latex-allergy-related asthma appears to be less common since the introduction of latex-free gloves in most healthcare settings. However, the introduction of new agents (hair dyes, hair fixing,

hair waving solutions) containing persulphates in the hairdressing industry and domestic household cleaning sprays are emergent causes. In developing countries the spectrum of agents causing OA is more diverse and less consistent.³ Between 1997 and 1999 SORDSA, the surveillance programme in South Africa, reported *isocyanates* (a component of automotive spray paints and polyurethane foam products), *latex proteins*, *flour and grain*, and *platinum salts* as the most common agents.⁶ In a study of patients presenting with acute asthma in emergency casualty units at two large public hospitals the most commonly cited exposures were cleaning agents, dyes and paints.¹³

DIAGNOSIS

While approaches to the diagnosis of WRA may vary between various countries, a stepwise approach

Table I. Common causative agents for occupational asthma and occupations at risk (adapted from Cullinan⁸)

High-molecular-weight agents		Low-molecular-weight agents	
Occupation	Agent	Occupation	Agent
Baking, milling, pastry making	Flour(s) (e.g. wheat, rye), fungal alpha amylase, other enzymes, egg white	Spray painters	Diisocyanates
Laboratory animal researchers and technicians	Rat, mouse, guinea pig, ferret, proteins, egg	Electronic solderers	Colophony fume
Healthcare workers	Latex	Healthcare workers	Glutaraldehyde, ortho-phthalaldehyde, methyl/butyl methacrylate
Detergent enzyme manufactures	Detergent protease, amylase, lipase, cellulase	Plastic and foam manufacturers	Diisocyanates, acid anhydrides, epoxy resins
Tea packers, coffee processors	Herbal teas, green coffee bean	Woodworkers, lumberjack	Red cedar, iroko, other tropical sawdusts
Seafood processors	Prawn, crab, other shellfish, fish proteins, <i>Anisakis</i> parasite	Textile workers	Reactive dyes
Other food processors	Garlic, egg, enzymes	Hairdressers	Persulphates
Flower and vegetable farmers	Pollens, mites	Pharmaceutical manufacturers	Penicillins, morphine, cimetidine

Table II. Epidemiological studies of work-related asthma in South Africa reported in the literature (1985-2012)

Type of workplace (author, yr published)	N	Outcome measure	Agent/s implicated	Prevalence/ incidence (%)
Biological agents				
Grain mill (Jeebhay 2000, 2005)	111	Work-related grain dust allergic asthma*	Wheat, storage pests (mealworm, cockroach, storage mites)	17%
(Yach 1985)	582	Asthma symptoms		23 - 25%
Grain mill (Bartie 2004)	84	Work-related asthma symptoms	Maize, storage pests (weevils)	7%
Soybean processing (Mansoor 2004)	115	Work-related soybean allergic asthma*	Soybean	IR: 2 per 1 000 person months
Supermarket bakeries (Baatjies 2009)	517	Occupational asthma and fungal alpha-amylase	Cereal flour (wheat, rye)	13%
Poultry processing (Rees 1998)	134	Asthma symptoms (feathers, droppings, serum)	Feed, poultry matter	12%
Seafood processing (Jeebhay 2008)	594	Occupational asthma	Fish products, fish parasite (<i>Anisakis</i>)	2%
Table grape vineyards (Jeebhay 2007)	207	Work-related spider mite allergic asthma*	Spider mite	6%
Experimental laboratory (Lopata 2004)	10	Work-related locust allergic asthma*	Locust matter (wings, faeces, body)	30%
Hospitals (high risk) (Potter 2001)	717	Work-related latex allergic asthma*	Natural rubber latex protein	7%
Dental institutions (Singh 2012)	421	Work-related latex allergic asthma*	Natural rubber latex protein	1%
Furniture plant (Pitt 1985)	27	Work-related asthma symptoms	Wood dust (yellow wood, stinkwood, blackwood, imbuia)	7%
Sawmill plant (Fox 2004)	392	Asthma	Wood dust (pine wood)	3%
Chemical agents				
Chemical processing/ packaging (Soderlund 1993)	20	Work-related asthma	Toluene diisocyanate	30%
Automotive spraypainting (Randolph 1997)	40	Asthma symptoms	Hexamethylene diisocyanate	10 - 15%
Platinum refinery (Calverley 1995)	78	Platinum salt sensitivity [†]	Platinum salts	24 month CI: 41%
Vanadium plant (Irsigler 1999)	1440	Asthma symptoms	Vanadium pentoxide	24 month CI: 11%
IR – incidence rate, CI – cumulative incidence. * Work-related asthma symptoms + antigen specific allergic sensitisation with or without spirometry changes † Work-related upper and/or lower airway symptoms + antigen allergic sensitisation Adapted from Jeebhay and Quirce ³ and updated from Jeebhay <i>et al.</i> , ^{9,10} Singh <i>et al.</i> ¹¹ and Baatjies <i>et al.</i> ¹²				

is often used, the extent of the investigation being largely dependent on the definition subscribed to, the purpose of the investigation and the level of diagnostic capabilities available. The American College of Chest Physicians (ACCP) Consensus Statement outlines the approach recommended for the diagnosis and management of WRA.² Recently, a taskforce of the European Academy for Allergy and Clinical Immunology also released a consensus statement for investigation of WRA in non-specialised centres.¹⁴ Thereafter, the diagnosis of asthma should be confirmed and the patient investigated to determine the presence of WRA, and

then performing these tests, whenever possible, *prior* to advising the patient to change jobs. The following approach can be used:

Step 1. Assess the probability of WRA based on clinical history

WRA (OA and WEA) should be considered in all adult patients with new-onset or worsening asthma by taking an appropriate history. In all individuals, document the onset and timing of symptoms, medication use and their temporal relationship to periods at and away from work. The following questions can be asked of

HIGH-RISK OCCUPATIONS FOR WRA IN SOUTH AFRICA



a. healthcare workers



b. spray painters



c. bakery workers

Fig. 2. High-risk occupations for work-related asthma in South Africa: a. healthcare workers; b. spray painters; c. bakers.

any patient with asthma that starts or becomes worse during work:

- Were there any symptoms prior to working in this current job?
- Were there any changes in work processes in the period prior to the onset of symptoms?
- Was there an unusual work exposure within 24 hours before onset of initial asthma symptoms?
- Do asthma symptoms differ during times away from work such as weekends, holidays or other extended times away from work?
- Are there symptoms of allergic rhinitis and/or conjunctivitis that worsen with work?

An occupational history is crucial in assessing the probability of being exposed to a respiratory sensitiser or irritant. The important questions to ask of the individual are:

- A history of the job duties and duration
- A list of the hazardous exposures (obtain material safety data sheets (MSDs) for products used by the individual), and cross-check against a list of known agents causing OA from reliable sources such as:
 - <http://www.asmanet.com/asmapro/agents.htm>
 - <http://eaaci.net/sections-a-igs/ig-on-occupational-allergy/allergen-list/235-occupational-allergens-list>
 - http://www.asthme.csst.qc.ca/document/Info_Gen/AgenProf/Bernstein/BernsteinAng.htm
 - <http://www.oem.msu.edu/userfiles/file/Resources/AsthmaCausingAgents.pdf>
 - <http://hazmap.nlm.nih.gov/>
- If an agent is not found on the list, it is possible to predict the likelihood of the agent to cause asthma based on its molecular structure¹⁵ <http://www.medicine.manchester.ac.uk/oeh/research/>
- Assess the level of airborne exposures of the job (degree of dustiness)
- Use of protective devices/equipment (respiratory, goggles, gloves)
- Presence of respiratory disease in coworkers.

Step 2. Confirmation of asthma diagnosis

Confirmation of asthma requires the demonstration of reversible airways obstruction. Using spirometry, significant reversibility is defined as a postbronchodilator increase of $\geq 12\%$ accompanied by a 200 ml increase in forced expiratory volume in 1 second (FEV₁). Serial peak expiratory flow rate (PEFR) monitoring over a 2-week period demonstrating a diurnal variation of $\geq 20\%$

is also considered to be diagnostic for asthma. An improvement of the FEV₁ after a steroid trial (30 mg/daily for 2 weeks) is another method to make the diagnosis. Furthermore, methacholine/histamine challenge tests have also been used to detect nonspecific bronchial hyperresponsiveness (NSBH) in individuals who display 'normal' spirometry findings. Fractional exhaled nitric oxide (FeNO) tests are increasingly being used to determine the presence of allergic airway inflammation when the diagnosis of asthma is equivocal. A FeNO greater than 50 ppb is indicative of the presence of eosinophilic airway inflammation suggestive of asthma.¹⁶ The differential diagnosis for WRA includes upper respiratory tract irritation and odour triggers, vocal cord dysfunction, eosinophilic bronchitis and asthma-like syndromes (e.g. byssinosis, grain dust chronic obstructive pulmonary disease (COPD)).

Step 3. Establish the work-relatedness of asthma

Serial PEFR testing using a portable peak flow meter is currently the most practicable test that can be used to assess work-relatedness in patients who are still working. Serial PEF monitoring may be particularly useful when tests against LMW chemicals that commonly cause asthma through non-IgE-mediated reactions are not available. Its main drawbacks are requirements for daily recording of PEFR and dependence on the individual's compliance. For PEFR to yield meaningful results, recordings need to be done 4 times daily over a 4-week period, with 2 weeks usual work and 2 weeks away from work. On each occasion, 3 efforts are conducted with the highest of the 3 efforts recorded. A specially designed chart is most appropriate. Record daily as (yes/no) adverse responses to workplace exposure, tight chest, flu symptoms, and any use of inhaled bronchodilator (indicate frequency of use). It is critical that exposure days are clearly recorded.

A computer-assisted system called Occupational Asthma System (OASYS) (<http://www.occupationalasthma.com/oasys.aspx>) has been used to provide a simple and validated method for interpretation of serial measurements of PEF and to assess work-relatedness of the recordings. A work-effect index (WEI) score is computed, with values >2.5 being highly suggestive of work-relatedness of asthma. The score has a 75% sensitivity and a 94% specificity for OA diagnosis.

Should the PEFR results be equivocal, the patient should be referred to a specialised centre for a nonspecific challenge test (methacholine or histamine) to document nonspecific airway responsiveness while the individual is still working in the job in question. The test should also be repeated during a period (optimally, at least 2

Table III. Diagnostic criteria for work-related asthma compensable under the Worker's Compensation system in South Africa under the Compensation for Occupational Injuries and Diseases Act – COIDA (1993)

Occupational asthma (immunological) as per Circular Instruction 176*	Occupational asthma (irritant-induced) as per Circular Instruction 177*	Work-aggravated asthma as per Circular Instruction 184*
Requires all 4 criteria (A - D): (A) A medical practitioner's diagnosis of asthma and physiological evidence of reversible airways obstruction or airways hyperresponsiveness. (B) An occupational exposure preceding the onset of asthmatic symptoms. (C) An association between symptoms of asthma and work exposure. (D) An exposure and/or physiological evidence of the relationship between asthma and the workplace environment (Requires D1 and preferably one or more of D2 – D5): (1) Workplace exposure to agent reported to give rise to occupational asthma. (2) Work-related changes in FEV₁ or PEFR. (3) Work-related changes in serial testing of nonspecific bronchial hyperresponsiveness (e.g. methacholine challenge test). (4) Positive specific bronchial challenge test. (5) Positive skin-prick test or raised specific IgE antibody level to the suspected agent.	Requires all 5 criteria: (1) Medical history indicating pre-existing asthma or history of asthmatic symptoms, prior to the start of employment or exposure to the known aggravating agent. (2) Presence of work-related exposures preceding and/or associated with the onset of an asthmatic attack or the worsening of symptoms. (3) Presence of work-related factors known to aggravate asthma symptoms (e.g. cold air, dusty work, chemical or biological irritants, indoor air pollutants, physically strenuous work, second-hand smoke). (4) Increase in symptoms or medication requirements, or documentation of work-related changes in PEFR or FEV₁ after start of employment or occupational exposure. (5) Presence of reversible airflow obstruction and/or nonspecific bronchial hyperresponsiveness on pulmonary function testing.	Requires all 5 criteria: (1) Medical history indicating the absence of pre-existing asthma-like complaints. (2) Onset of symptoms after a single or multiple exposure(s), incident(s) or accident(s). (3) An occupational exposure to a gas, smoke, fumes, vapour or dust with irritant properties. (4) Onset of symptoms within 24 hours of exposure with persistence of symptoms for at least 3 months (an association between symptoms of asthma and exposure). (5) Presence of airflow obstruction on pulmonary function tests and/or presence of nonspecific bronchial hyperresponsiveness on tests done at least 3 months after exposure.

* As published in the Government Gazette, Republic of South Africa, Pretoria <http://www.labour.gov.za/programmes/>

weeks) away from the work exposure to identify work-related changes.

Step 4. Determine whether WEA and/or OA is present

Diagnostic criteria for the various entities of work-related asthma used by the worker's compensation dispensation in South Africa are outlined in Table III.

The presence of WEA should be considered highly probable in individuals who have asthma not caused by work but whose pre-existing asthma subsequently worsens while working. They usually have moderate-to-low level workplace exposures to dust, smoke, fumes, sprays or extreme physical factors (e.g. cold air, humidity, strenuous work). There is commonly no documented workplace exposure to agents known to induce OA.

Step 5. Confirmation of sensitisation to occupational allergens

Immunological assessment aims to support the diagnosis of occupational allergic asthma in symptomatic patients exposed to HMW agents (proteins) and some LMW chemicals. In individuals with suspected sensitiser-induced OA, immunological tests such as skin-prick testing or in vitro specific IgE assays can be used to identify sensitisation to specific occupational allergens when these tests are technically reliable and available, e.g. Phadia ImmunoCAP. It is well documented that atopic workers exposed to a number of HMW agents (proteins) are at increased risk of becoming sensitised to these allergens.

Step 6. Confirmation of the causal role of occupational agents using the specific bronchial challenge test – the gold standard

In very rare circumstances, when the diagnosis or causative agent is equivocal, a specific inhalation challenge test (the reference standard for OA) with the agent concerned is performed in South African facilities.

MANAGEMENT

Early diagnosis and early avoidance of further exposure are the cornerstones of patient management for WRA.

Exposure avoidance

Patients with sensitiser-induced OA, whenever possible, should be removed from exposure to the causative agent as it is associated with the best health outcome. If this is not possible, reduction of the exposure through relocation to less exposed jobs is the second option.¹⁷ A recent Cochrane review confirmed that removal of individuals with OA from exposure compared to continued exposure is associated with a beneficial effect on asthma symptoms and lung function including both FEV₁ and NSBH. This was especially apparent for occupational asthma due to LMW agents.¹⁸ The use of respirators is of limited value as they are generally ineffective in totally preventing exposure. They may be associated with improvement, but not a complete suppression, of respiratory symptoms and airway obstruction in the short term.

In individuals with irritant-induced OA or WEA, the main focus should be on optimising asthma treatment and reducing the exposure to the relevant workplace triggers. For WEA individuals may continue working in their current job provided there is close environmental control and asthma treatment is optimised. If this is not successful, changing jobs to a workplace with fewer triggers in order to control asthma is recommended. However, patients with reactive airways dysfunction syndrome (RADS), can continue to work in the same job provided measures are taken to prevent further exposures to high concentrations of irritant agents.²

Optimising asthma treatment

Pharmacological treatment of WRA does not differ from therapy of other types of asthma, and should comply with the Global Initiative for Asthma (GINA) guidelines. Efforts should be made to increase the use of inhaled corticosteroids early after the diagnosis as this has been shown to improve the prognosis.² Other comorbid conditions, e.g. rhinitis, should also be treated optimally. General measures such as cessation of smoking and avoidance of exposure to common

Table IVa. Parameter 1: Postbronchodilator FEV₁

Score FEV ₁	% Predicted
0	>Lower limit of normal (80)
1	70 - lower limit of normal
2	60-69
3	50-59
4	<50

Table IVb. Parameter 2: Minimum medication prescribed

Score	Medication
0	No medication
1	Occasional bronchodilator, not daily
2	Occasional or daily bronchodilators and/or daily low-dose inhaled steroid (<800 µg beclomethasone or equivalent)
3	Daily bronchodilator and/or daily high dose inhaled steroid (>800 µg beclomethasone or equivalent and occasional (1-3/year) course oral steroid)
4	Daily bronchodilator and/or daily high dose inhaled steroid (>800 µg beclomethasone or equivalent) and frequent (>3/year) course systemic steroid or daily oral steroid

Table IVc. Summary impairment scores in cases accepted as occupational asthma (OA) (sum of parameters 1 and 2)

Impairment total score	Permanent disablement
0-1	15%
2	20%
3	30%
4	40%
5	50%
6	60%
7	70%
8	80%
Fatal case of OA	100%

aeroallergens to which the patients may also be sensitised, or to environmental irritants, should always be recommended.

Ongoing follow-up and assessment of impairment/disability

The patient should be monitored on an ongoing basis and if asthma is severe or worsens, job change should be considered if the individual is still exposed. Impairment and disability evaluation should be carried out as soon as the asthma is stabilised. For workers' compensation purposes, assessment is done *3 weeks after removal* from exposure and after 2 years when the asthma is stabilised.

The degree of impairment is generally evaluated by considering the presence of symptoms and exacerbations, postbronchodilator FEV₁, degree of reversibility or NSBH, type and dose of medication needed for control, and disability is evaluated in relation to the person's job.²⁰

A recent review of poor prognostic factors for the outcome of OA identified older age, exposure to HMW agents (proteins), longer duration of exposure to the offending agent at time of diagnosis, and impaired lung function at diagnosis as highly predictive of poor outcomes. While the effect of gender was equivocal, other factors such as atopy and smoking appeared to have no influence.

Patients should be counselled regarding optimal work accommodation and placement and their eligibility to apply for workers' compensation during this period.

Assist with workers' compensation claim

WRA has serious socioeconomic consequences and therefore assistance provided by the medical practitioner with the compensation claim will contribute towards preservation of the income and improved quality of life of the affected individual. About one-third of workers with OA are unemployed at 6 years after diagnosis and are known to suffer financially.⁷ Workers' compensation for OA is covered by specific legislation for non-miners (Compensation for Occupational Injuries and Diseases Act – COIDA, 1993) except for asthma in platinum salt refinery workers, which is covered by another piece of legislation (Occupational Diseases in Mines and Works Act, 1973) that compensates miners with occupational lung diseases. The workers' compensation dispensation (COIDA) covers all types of WRA (Table III).

Medical practitioners should support affected workers in the application of a workers' compensation claim by completing the medical report required in support of the compensation claim after each visit. They also have a legal obligation to report an occupational disease to the corresponding workers' compensation system and the Department of Labour. The detailed workers' compensation submission procedures are outlined elsewhere.²² The degree of disability under COIDA legislation is based on the level of bronchial obstruction and the need for medication to control residual asthma (Table IVa-c).²³ In instances where sensitisation persists after removal from exposure, 15% permanent disablement is awarded should the worker have normal lung function and there is no need for medication.

Claim acceptance accords the following benefits to the patient:

- cover for medical expenses (tests and treatment)
- replacement of loss of wages (75% of full wage) for periods absent from work
- permanent disablement benefits (lump sum or pension if disability >30%)

- additional compensation if employer was negligent or if the individual's disability worsens as a result of the asthma.

CONCLUSION

WRA, especially in developing countries, remains under-recognised, poorly managed and inadequately compensated. There is a need for more widespread surveillance systems, using internationally accepted definitions, to identify trends in the incidence of OA across various industries, including the informal sector. The use of validated instruments (questionnaires, immunological tests, serial PEFr) that are easily accessible, administered and interpreted using uniformly accepted protocols will enhance the recognition and consequent management of the disease. Improved medical practitioner awareness and a high index of suspicion for occupational causes of recent-onset adult asthma will also contribute towards better management of this disease. Further assistance with diagnostic, management and compensation-related issues can also be sought from specialist occupational medicine referral clinics located at the National Institute for Occupational Health (Gauteng), Groote Schuur Hospital (Western Cape) and King Edward Hospital (KwaZulu-Natal).

Acknowledgements

This is an updated version of an article originally published in CME Nov/Dec 2009 Vol.27 No.11 and a talk presented at the ALLSA Congress in Cape Town, July 2012.

Declaration of conflict of interest

The author declares no conflict of interest.

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R25 000 will be made available in 2012. The purpose of the award is to support local research into allergic conditions of Southern Africa. Preference will be given to supporting non-established researchers demonstrating research potential.

Closing date for application
31 October 2012

Application details can be obtained from the ALLSA office
Please visit the ALLSA Website at www.allergysa.org to submit your electronic application

Please note that only electronic submissions from fully paid-up ALLSA members will be processed

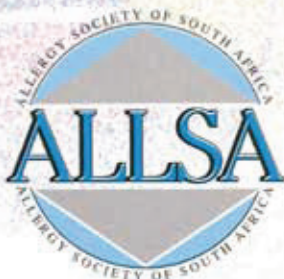
ALLSA NATIONAL OFFICE

P.O. Box 88
Observatory
7935

TELEPHONE & FAX ENQUIRIES

Fax: (021) 448 0846
Tel: (021) 447 9019

E-mail: mail@allergysa.org



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ALLSA MEMBERSHIP 2012



Support your society, it supports allergology in South Africa

ALLSA remains one of the world's most pro-active and innovative allergy societies. Our pioneering website and patient information resources have spurred other national societies to follow suit.

ALLSA relies on an active membership base to continue to provide excellent resources to healthcare workers in Southern Africa. We welcome new members from all over Southern Africa and membership is open to all healthcare workers with an interest in allergology. Our current membership includes medical practitioners (general practitioners, physicians, pulmonologists, ENT specialists, dermatologists, ophthalmologists, paediatricians and anaesthetists), nurses, dieticians, medical technologists, pharmaceutical industry staff and medical students.

Membership currently costs only R300 annually; this is a tax-deductible expense.

Members enjoy a number of privileges which include:

- The highly rated ALLSA journal – Current Allergy & Clinical Immunology, which is edited by Profs Weinberg and Haus, and published quarterly. This journal is available on-line.
- Access to ALLSA's comprehensive allergy website at www.allergysa.org.
- On-line CPD accreditation.
- Patient information guides and leaflets on common allergic disorders.
- Discounted ALLSA congress registration fees for our annual congress.
- Regional allergy courses, meetings and journal clubs.
- Support with examination preparation for the Diploma in Allergology of the Colleges of Medicine of SA.
- Access to allergy research funding and annual ALLSA research awards of up to R50 000 per research study.

For more details on membership and privileges please contact Ruwayda Adams on tel 021-447-9019, fax 021-448-0846, or e-mail enquiries to mail@allergysa.org

Please cut out the membership application form and post together with your payment.

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ALLSA Membership Application

R300 annual subscription

Dr/Sr/Mr/Mrs/Ms:

Address:
.....

E-mail address:

Phone: Fax:

Speciality:

Special Interests:
.....

Comments:
.....

Post to: Allergy Society of South Africa, PO Box 88, Observatory 7935

Direct debit details:

Standard Bank: Rondebosch

Sort Code: 02-50-09

Account Number: 07 149 1821

Cheques payable to **Allergy Society of South Africa**

For direct deposits: Kindly fax your bank deposit slip as proof of payment (Fax No. 021-448-0846)