OCCUPATIONAL RESPIRATORY DISEASES IN AUSTRALIA

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Australian Government
Australian Safety and Compensation Council

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FOREWORD

The Australian Safety and Compensation Council (ASCC) leads and coordinates national efforts to prevent workplace death, injury and disease in Australia and aims to improve national workers' compensation arrangements and return to work of injured employees.

Through the quality and relevance of the information it provides, the ASCC seeks to influence the awareness and activities of every person and organisation with a role in improving Australia's occupational health and safety (OHS) performance.

The *National OHS Strategy 2002-2012, (*the National Strategy) which was endorsed by the Workplace Relations Ministers' Council on 24 May 2002, records a commitment by all Australian, State and Territory governments, the Australian Chamber of Commerce and Industry and the Australian Council of Trade Unions, to share the responsibility of ensuring that Australia's performance in work-related health and safety is continuously improved.

The National Strategy sets out five 'national priorities' to achieve short-term and long-term improvements.

The priorities are to:

- reduce high incidence and high severity risks
- improve the capacity of business operators and worker to manage OHS effectively
- prevent occupational disease more effectively
- eliminate hazards a the design stage, and
- strengthen the capacity of government to influence OHS outcomes.

In March 2004 it was agreed by the then National Occupational Health and Safety Commission (NOHSC) that, under the national priority to prevent occupational disease more effectively, eight disease categories would be considered for particular focus under any national action plan. These are work-related musculoskeletal disorders; mental disorders, noise-induced hearing loss; respiratory diseases; occupational cancers; contact dermatitis; infectious and parasitic diseases, and cardiovascular disease.

To assist the setting of national action priorities to prevent these diseases, reports were prepared for members on each disease category. The following report is an extract of the information provided to members on the causes and risk factors for cardiovascular disease, the available data on the magnitude and severity for the disease category within Australia, approaches to prevention and evidence for their effectiveness.

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ACKNOWLEDGMENTS

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EXECUTIVE SUMMARY

Background

The project is a literature-based review of epidemiological studies on work-related respiratory diseases and review of existing data sources, including coverage of the magnitude and severity, causes, affected occupational groups and possible prevention approaches, with specific focus on Australia. This information was designed to assist NOHSC's overall OHS strategy and more specifically to inform National Priority Action Plan 3, to prevent occupational disease more effectively.

Definitions and a consideration of relevant concepts and issues

Occupational respiratory disease is the name for a collection of conditions of the respiratory system which can have occupational factors as risk factors for the particular disease. This includes acute, sub-acute and chronic diseases, which may be malignant, non-malignant or in infectious origin. However, many of these conditions can be caused by non-occupational factors, so deciding whether a particular type of respiratory disease should be considered as work-related is not always clear. In particular, this is often a problem in recognizing the work association for those conditions which are common in the community, e.g. asthma, or have a long latent period between work exposure and the development of the disease. In such situations, the relationship to work is often not recognised.

As there are separate occupational disease profiles being written for occupational cancers and occupational infectious diseases, this report focuses on those occupational respiratory diseases in Australia which are not malignant, so for example mesothelioma and lung cancer are not included, and those which don't have an infectious cause, so for example respiratory tuberculosis is not included.

Methods

Most of the information presented in this report is based on published, peer-reviewed literature, identified through literature searches of the major occupational health literature databases. No new investigations were undertaken to obtain information on exposure or disease risk, specifically for the purposes of this report.

Numeric information on the extent of work-related respiratory disease in Australia was obtained from several sources, including national workers' compensation accepted claims in the NOSI database, publicly available data relating to accepted claims to the Dust Diseases Board of NSW, physician notifications in Victoria and Tasmania to the SABRE database held by Monash University and physician notifications in NSW to the SABRE database held by the Dust Diseases Board (DDB) of NSW. Other

possible sources of Australian data, such as the BEACH data and the Hospital inpatient data were not found to provide useful data relating to occupational respiratory diseases.

Main occupational risk factors for occupational respiratory disease

There are many types of occupational respiratory disease and several, such as the pneumoconioses, were first identified more than 100 years ago, and for which well established causes have been identified long ago. Others, such as occupational asthma, have been first recognised in more recent times and causes are still being identified. The spectrum of such occupational respiratory diseases is changing, particularly in industrialised countries, with a stabilisation or reduction in the incidence of those diseases of longer latency and an increase in those conditions of shorter latency, such as occupational asthma. For some occupational respiratory diseases, such as the pneumoconioses, there are only a few work-related risk factors in specific industries but for occupational asthma there are several hundred proven or highly likely work causes spread across a very broad spectrum of jobs and industries. In addition, the pneumoconioses tend to occur after a medium to long period of time of moderately high exposure, whereas occupational asthma tends to occur after a fairly short period of moderate to low exposure.

Estimates of occupational respiratory disease in Australia

There is limited information on the extent of work-related respiratory disease in Australia. A proportionate attributable risk approach showed that the estimated age-adjusted mortality rates (expressed in number of deaths per million per year) were estimated to be 5 and 2 for asthma, and 8 and 0 for dust diseases, respectively in men and women. However, these estimates didn't address morbidity and so underestimate the impact of these diseases, as most people who develop these diseases don't die from them. Overseas estimates suggest that 15% of asthma is occupational in origin, but there is considerable variability in published studies.

There are no comprehensive population-based estimates of work-related respiratory disease in Australia. Workers' compensation data systems are unlikely to be a comprehensive source of information on cases of occupational respiratory disease, but can provide some relevant information. Nearly 1300 respiratory disease claims were reported in Australia for the period 2001-03, which is likely to be a considerable underestimate. Claims are nearly twice as high in men as in women. In men, the commonest categories are 'asbestos-related disease' and 'other diseases'. In women, claims are mainly in the category 'other diseases' followed by 'asthma'. Many of these asthma cases are likely to be aggravation of pre-existing asthma, rather than true occupational asthma.

Review of the most recent annual report of the NSW DDB for the period 2002-2003 shows some interesting comparisons with the NOSI data. A

total of 186 NSW workers received compensation payments from the Dust Disease Board for silicosis in the financial year 2002-03. However, for the three-year period 2001-2003, nationally across all states and territories, only 37 silicosis claims were recorded in the NOSI data. This indicates considerable under-ascertainment of the true number of silicosis cases in official workers' compensation data.

A similar problem exists for occupational asthma cases, based on SABRE data, comprising physician notified cases in Victoria and NSW. According to the SABRE notification scheme, the most common condition reported by physicians is asthma (33 % of the occupational respiratory events reported in Victoria and Tasmania). The asthma incidence rate is 30.9 (95 % confidence interval = 26.8-35.5) per million workers per year with a 2.4 times higher incidence rate in men compared to women. However, this incidence rate is likely to be an underestimate. The two most commonly reported causative agents for asthma in the SABRE notification scheme are wood dust and isocyanates (13.5 % and 5.8 % respectively).

Approaches to prevention of occupational respiratory disease

Prevention of occupational respiratory disease at the workplace requires the identification of exposure and assessment of risk from airborne substances known to cause such diseases. Elimination or minimization of workers' exposure can be achieved by substitution with a less hazardous substance whenever possible, or exposure reduction, based on the hierarchy of controls. The use of personal protective equipment can be effective, as long as it is used in conjunction with other recognised control measures.

As occupational respiratory diseases can be difficult to identify and control, their prevention is likely to be more effective by implementing complementary actions such as education towards employers, workers, and physicians, research in order to identify new hazards and their impact on workers' health and review of workplace safety and standards on an ongoing basis. This process is best informed by a comprehensive national surveillance scheme to assist in effective targeting of preventive measures.

All OHS jurisdictions are undertaking some initiatives aimed at preventing the incidence of these diseases. For information on these readers are directed to the NOHSC members' websites listed in the last section of the main body of the report.

Conclusions

Work-related respiratory disease is almost certainly an important cause of work-related morbidity and probably mortality in Australia, but our current data sources related to these diseases do not provide reliable or complete national data on the occurrence of these diseases to help target prevention activities.

From the incomplete Australia data which are available, considerations of the situation in similar overseas countries and the likely PARs in Australia, we can cautiously conclude that the pneumoconioses have probably stabilised and are likely to reduce in future years, while occupational asthma is probably increasing and is now the most important type of occupational respiratory disease. COPD is the other major type of occupational respiratory disease and is likely to become of increasing importance in the future, particularly with an aging population.

The prevention of occupational asthma is likely to be particularly important, but has some specific challenges, due to the very large number of workplace agents which can cause this condition, which are found in many occupations and industries in Australia, many of which are likely to be in small to medium size industry.

1. INTRODUCTION

This report is a literature and data source review of occupational respiratory diseases, including coverage of the likely magnitude, causes, affected occupational groups and possible prevention approaches, with specific focus on the situation in Australia. It has been written to assist the National Occupational Health and Safety Commission and State and Territory jurisdictions in developing future programs and activities to reduce the impact of these diseases, as part of the National Occupational Disease Prevention Strategy.

The focus of this report is non-malignant (i.e. non-cancerous) and non-infectious respiratory conditions. Respiratory lung conditions, such as mesothelioma and lung cancer, and infectious respiratory conditions, such as tuberculosis and legionnaires' disease, are considered in the Occupational Respiratory Disease Profiles on Occupational Cancer and Occupational Infectious Diseases respectively.

1.1 Aims of the report

The main aims of this report are to:

1. Provide an overview of the main types of occupational respiratory disease in Australia.

2. Review existing data sources related to the occurrence of occupational respiratory disease in Australia and estimate the extent of these diseases.

3. Identify major 'at risk' areas which could be the target of future prevention activities.

1.2 Structure of the report

The report has several main sections. These present, in order:

- the scope of the report and a consideration of relevant concepts and issues;

- an outline of the methods used in obtaining information for the report;

- information on the main occupational respiratory diseases;

- estimates of the extent of occupational respiratory disease in Australia; and

- a summary of what is known about approaches to preventing work-related respiratory disease.

2. SCOPE AND RELEVANT ISSUES AND CONCEPTS

2.1 Scope

Occupational respiratory disease

Respiratory diseases are common in the Australian community. For some, such as asthma, Australia has one of the highest rates in the world, with about 15% of the community developing this disease at some stage in their lives. The major risk factor for respiratory disease is cigarette smoking, but other causative factors include outdoor air pollution and contaminants in the indoor environment, such as oxides of nitrogen from unflued gas heaters, mould and house dust mites (Newman Taylor 1980). These non-work risk factors are important in their own right, but they may also interact with work factors to exacerbate a particular respiratory disease.

Occupational respiratory disease is respiratory disease that is caused or exacerbated by work factors. In cases of non-malignant and non-infectious occupational respiratory disease which is caused by work, the causative work factor becomes airborne in the work environment and is breathed in by the worker, usually over a prolonged period of time. Such airborne contaminants may exist in several forms, such as dust, mist, fibres, fume, vapour or gas. To be breathed in and cause disease, such airborne contaminants in the workplace need to be what is known as 'respirable', i.e. small enough in size to gain access to the deeper, pulmonary areas of the lung.

Work factors may also aggravate non-occupational respiratory conditions. The major condition in this category is asthma, where it is known that factors such as exercise, stress, cold air, dust and other non-specific irritants can trigger attacks of asthma. Such trigger factors can be encountered in workplaces and, even though they are not the direct cause of the underlying respiratory disease, they can cause significant morbidity and, in severe cases, mortality.

This report focuses on the more common and/or more important occupational respiratory diseases in Australia. For each disease, the basic epidemiology and patho-physiology are briefly considered, along with any specific Australian-based information on occurrence. High-risk occupations, industries and/or tasks are identified and attempts are made to estimate the number of workers at risk in Australia, where reliable data are available.

2.2 Relevant issues

Latency

Latency is defined as the time from first exposure to a workplace airborne contaminant and the development of clinical disease. Latent periods vary quite widely for different types of occupational respiratory disease and this

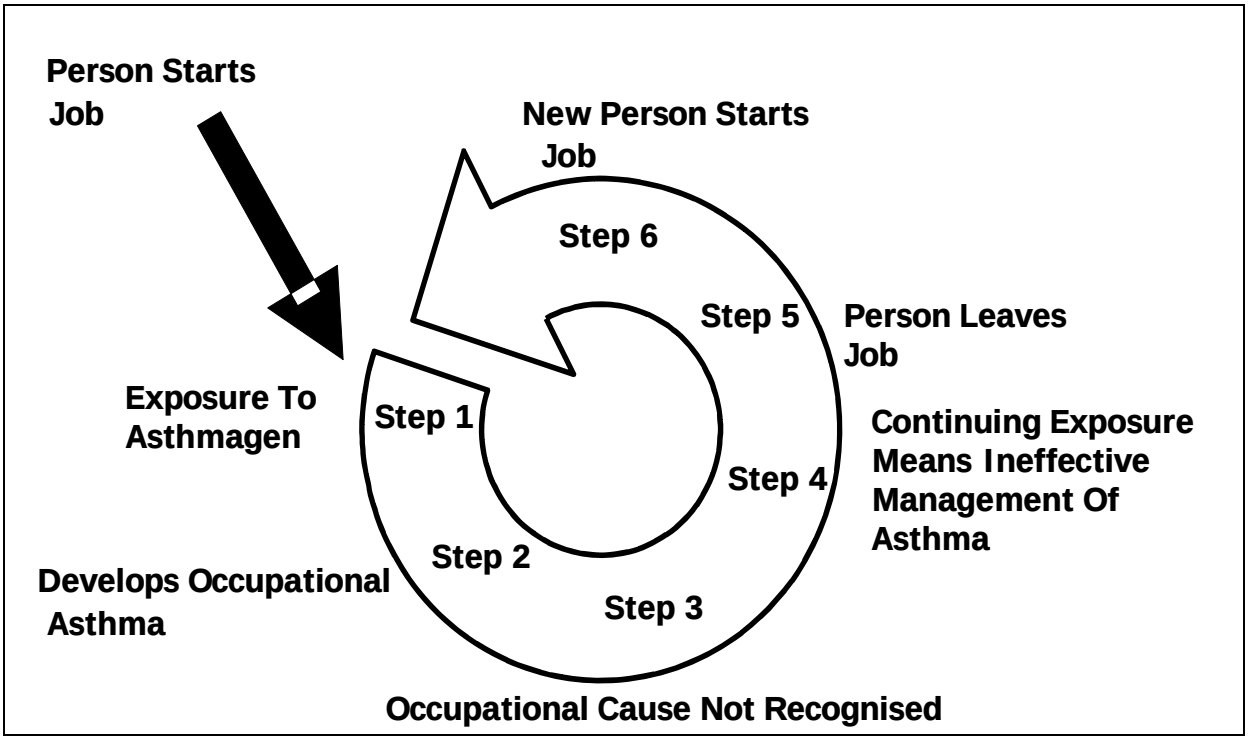
can have important implications for the likelihood of the workplace factor being identified. For inhalational accidents, the respiratory effect is seen almost immediately or within a few hours following exposure, so it is usually fairly easy to identify the role of work factors. For occupational asthma, the symptoms usually don't start until some weeks to months after first exposure and such symptoms often occur outside work hours, usually at night. For some occupational respiratory diseases, such as the pneumoconiosis, the latent period is usually very long, often many years or decades after first exposure and in many cases after the worker has retired from work. Therefore the relationship with work may not be identified for these diseases with moderate to long latencies, unless work factors are specifically looked for.

Lack of specific work-related features

For some of the occupational respiratory diseases, such as asthma, there are no pathological or clinical differences between disease arising from work exposures and disease arising from non-work exposures. In these cases, the critical distinction is in the history, particularly asking about work exposure to known causes of the disease and the pattern of the onset of respiratory symptoms, which for occupational asthma can have some characteristic features.

However, medical personnel often don't have sufficient knowledge or expertise in occupational medicine to identify these relationships with work and the connection between work and the disease can often be missed. This can lead to a continuous cycle of exposure and disease, as workers begin work with these agents, develop occupational asthma, eventually need to leave the job and, if the cause is not identified, are replaced by another worker who is then at risk of developing the disease (Figure 1 next page). (Sim 2003)

Figure 1. Cycle of exposure and disease in occupational asthma



Population attributable risk

The difficulty establishing the connection between a work exposure and a particular disease case and the lack of reliable occupational disease data have led many studies of occupational disease to use a Population Attributable Risk (PAR) approach to estimate the proportion of all cases of a particular condition that are due to a particular work exposure (or group of exposures). The PAR is dependent on the relative risk of developing the condition due to the exposure, and the proportion of the population that has that exposure.

Few studies have used PAR when considering respiratory disease because the required information on the risk associated with exposure, and the prevalence of exposure, is not commonly available.

3. METHODS

3.1 Introduction

Most of the information presented in this report is based on two sources:

- a review of published, peer-reviewed literature from Australia and elsewhere; and
- a review of available Australian data sources containing information on the occurrence and extent of occupational respiratory diseases.

No new data collection or other investigations were undertaken to obtain general or specific information on exposure to respiratory hazards or risk of occupational respiratory disease for the purposes of this report.

3.2 Identifying and reviewing relevant literature

English language literature published up to January 2005 was searched for relevant articles.

The searches were conducted through Medline (via Ovid) and OSHROM, which incorporates HSELINE, RILOSH, CISDOC, NIOSHTIC, and MEDLINE. Secondary follow-up of sources cited in reference lists was also undertaken. The searches used three groups of keywords.

(1) A range of keywords describing non-malignant, non-infectious respiratory outcomes was used, such as “lung”, “pulmonary”, “asthma”, “pneumoconiosis”, “COPD”, “COAD”, “emphysema”, “chronic bronchitis”, “inhalational accident”, “pneumonitis”, “berylliosis”, “byssinosis”, “RADS”, “silicosis” and “asbestosis”.

(2) Searches were limited to occupational or job-related literature using keywords such as “occupational”, “job” and “industrial”.

(3) keywords “Australia”, “Australian” and “Australians” were used in some searches to ensure literature directly relevant to Australian workers was identified.

The Ovid search was written in such a way that articles were retrieved containing at least one keyword from each of keyword groups (1) and (2), and (3) for those searches where Australian studies were to be identified.

The resultant set of articles was reviewed and relevance of papers to the purpose of this report was determined by considering the abstracts or the full text of each article.

3.3 Occupational Respiratory Disease Data sources

Numeric information on the extent of work-related respiratory disease in Australia was obtained from the following main sources:

- National workers’ compensation information for new claims registered from 2001 to 2003 inclusive came from the NOHSC Online Statistics Interactive National Workers’ Compensation Statistics Databases (NOSI) accessed at the NOHSC website (http://www.nohsc.gov.au/ohsinformation/nosi/default.asp, accessed January 2004).

- Additional NSW compensation claims information was sourced from the Dust Diseases Board of New South Wales, 2002/2003 Annual

Report (http://www.ddb.nsw.gov.au/home.asp, accessed January 2005).

- Information on physician notified cases of occupational respiratory disease in Victoria and NSW were obtained from the Surveillance of Australian workplace-Based Respiratory Events (SABRE) databases in each state. Information on the SABRE programs can be found at: http://www.med.monash.edu.au/epidemiology/oeh/sabre.html for SABRE Victoria http://www.ddb.nsw.gov.au/home.asp for SABRE New South Wales.

4. MAIN OCCUPATIONAL RESPIRATORY DISEASES

4.1 Introduction

There are many respiratory diseases that have been documented in the literature as being related to work, either as a direct causal agent or as being aggravated by work exposures. This section describes those occupational respiratory diseases that in the Australian context are thought to be common, serious and/or have a high public profile. These are listed in Table 1 and a brief description of each follows. The main source of information for these conditions and their definitions is the recently published textbook; *Occupational Disorders of the Lung: Recognition, Management and Prevention*, edited by Hendrick et al (Hendrick et al 2002).

Table 1. Occupational respiratory disease

Disease
1. Asbestos related conditions
2. Silicosis
3. Coal workers' pneumoconiosis
4. Other pneumoconioses
5. Occupational asthma
6. Chronic Obstructive Pulmonary Disease (including emphysema and/or chronic bronchitis)
7. Toxic pneumonitis
8. Hypersensitivity pneumonitis

4.2 Asbestos related conditions

Asbestos is one of the better known workplace respiratory hazards and Australia in the past has had a large asbestos industry, including asbestos

mining, milling and manufacture of asbestos products. While it is best known as a cause of mesothelioma (a malignant condition not considered in this report), asbestos is also known to cause other respiratory conditions as follows:

4.2.1. Asbestosis

Asbestosis is defined as diffuse interstitial fibrosis of the lung resulting from the inhalation and retention of considerable numbers of respirable asbestos fibres, usually after prolonged exposure. Therefore, asbestosis only occurs from work exposures, so when it is diagnosed, the link to occupation should be clear. It is one of the pneumoconioses, with the others members of this group described in Section 4.3 of this report. While the incidence of other pneumoconioses has generally been declining in developed countries such as the UK and the USA over the latter part of the 20th century, the incidence of asbestosis has been increasing or remaining stable over that time. (HSE 1998, NIOSH 1996) Asbestosis is characterised by a progressive reduction in respiratory function and related shortness of breath and other respiratory symptoms and can lead to death through respiratory failure. The rate of progression of asbestosis is usually related to the degree of cumulative asbestos exposure at work.

4.2.2. Benign pleural conditions

Occupational exposure to asbestos is also known to cause several chest conditions, which are collectively known as benign pleural disease. They are called 'benign' because they usually do not cause any adverse effect on the health of the affected person, nor do they shorten life. They are usually found incidentally during x-rays of the chest for other reasons and can cause a great deal of anxiety in people who are told they have these conditions. They are also not thought, in themselves, to increase the risk of other more serious asbestos-related conditions, but are considered a marker of previous cumulative asbestos exposure which may increase the risk of such conditions. While asbestos exposure is almost always the cause of occupational benign pleural conditions, there can be other causes, such as injury, intrathoracic infections such as tuberculosis or inhaling other types of fibres. The most common examples of benign pleural conditions are pleural plaques and pleural thickening.

Pleural plaques are defined as circumscribed areas of thickening of the parietal pleura of the chest wall and diaphragm. They are the most common respiratory effect of asbestos exposure. (Craighead et al 1982) They have also been found in workers exposed to other dusts or fibres, such as talc, kaolin or ceramic fibres.

4.3 Pneumoconiosis due to other causes

One of the main and best known types of pneumoconiosis is asbestosis, which was considered in Section 4.2. However, there are several other

types of pneumoconioses, the two most important of which are silicosis and coal workers' pneumoconiosis.

4.3.1. Silicosis

Silica is a naturally occurring widely abundant mineral that forms the major component of most rocks and soil. Free silica occurs in several crystalline forms, and in amorphous non-crystalline forms. Among the crystalline forms, quartz is the most abundant, and is the form usually associated with human disease. Cristobalite and tridymite are less common but more biologically active, and can cause disease with appropriate exposure. Amorphous non-crystalline forms of silica occur in nature, mainly as diatomaceous earth (the skeleton of marine organisms). The amorphous forms of silica are classified as nuisance dusts and don't induce pneumoconiosis. However, high-temperature processes may convert non-crystalline silica into crystalline forms, including cristobalite.

Silicosis is a chronic diffuse lung disease caused by long-term inhalation of dust containing free crystalline silica. Concomitant inhalation of other dusts like coal or iron may lead to mixed pneumoconioses. Silicosis requires high and usually prolonged free crystalline silica inhalation. Lung function abnormalities are uncommon in early silicosis, but reduction in lung function appears in workers with more advanced disease. The inhalation of silica is also associated with a variety of other non-malignant non-infectious adverse health effects that occur at lower doses than may be needed to produce silicosis, including chronic bronchitis and chronic obstructive pulmonary disease (Hnizdo and Vallyathan 2003).

The mineral form (quartz, cristobalite, tridymite) of silica, particles size, the concentration in the air, and the duration of exposure all impact on the prevalence, latency, and progression of silicosis. Prevention in the workplace should primarily be focused on silica exposure control and reduction. Exposure to airborne particles of free crystalline silica can occur in numerous industries and environments. Occupations with exposure to harmful silica are those where:

- Rock is drilled or removed from the earth: rock mining, tunnel drilling
- Stones are processed: stone cutting or sculpting, granite monuments carving
- Silica or sand are used as abrasives: abrasive blasting, foundry casting, tool grinding
- Silica or sand powder is produced: production of silica flour and of diatomaceous earth
- Silica or sand powder is used: glass, crystal, and ceramic manufacture, pottery.

Silicosis is becoming less common in industrialised nations through aggressive measures to control airborne dust in the workplace, but is still a major problem in many developing nations.

4.3.2. Coal workers' pneumoconiosis

Pneumoconiosis due to coal dust (and coal mine dust) is known as 'coal workers' pneumoconiosis' (CWP). Coal rank (percentage carbon) is an important predictor of CWP development. Coal composition is not uniform and its carbon content can vary. The higher the carbon content the greater the risk of pneumoconiosis. Coal can be separated by silica-containing rock. Silica contamination increases the pneumoconiosis risk and consequently in some coal miners, pneumoconiosis may represent a mixed picture of coal pneumoconiosis and silicosis. Nevertheless, pneumoconiosis occurs when coal dust exposure is encountered without any silica contamination. First stages of CWP are not associated with respiratory symptoms. By contrast, CWP at later stages may present with breathlessness and cough.

Miners have the most obvious risk of inhaling hazardous amounts of dust, depending on how effective is the mine's ventilation and its method of suppression of dust. Prevalence of progressive massive fibrosis after a working lifetime of exposure to coal dust in underground miners was estimated between 1.3 and 2.9 % in Australia, based on data from the UK and the USA, respectively (Kizil and Donoghue 2002). In open-cast mines, dust levels rarely approach those in the confined environment of underground mines. Workers who are not protected by working in enclosed machine cabins may nevertheless be exposed to high levels. Another source of exposure is residual dust from burned coal known as 'fly ash'. Its composition is variable and it is suggested that its toxicity with respect to fibrogenicity is likely to be lower than that of coal dust.

4.3.3. Other pneumoconioses

Although the three work exposures which most commonly cause pneumoconiosis are asbestos, silica, and coal, inhalation of other mineral or metallic particles of dust can also lead to pneumoconiosis. Examples are minerals, such as talc, mica, or kaolin, and various metals, such as beryllium, cadmium, chromium and cobalt. The development of pneumoconiosis typically requires 5 or more years of exposure to the causative agent(s) with latency between exposure and diagnosis of 10 or more years. Therefore, making a diagnosis and establishing the type and severity of mineral or metal exposure can be problematic for these less well known causes of pneumoconiosis.

Symptoms in cases of pneumoconiosis from any cause are highly variable and can range from no respiratory complaints at all to severe dyspnoea in the case of advanced pneumoconiosis with massive fibrosis. Most of these pneumoconioses are associated with ventilatory impairment. Chest

radiographic abnormalities using the International Labour Office (ILO) standardised method (ILO 2000 revised classification) provide the principal features for diagnosing and staging pneumoconiosis.

4.4 Occupational asthma

Occupational asthma is defined as asthma caused by exposure to agents encountered in the working environment in workers without pre-existing asthma. Airways responsiveness is obstruction in nature and results in wheeze, chest tightness, cough and shortness of breath. The symptoms appear and tend to increase during the working week and often remit during absence from work.

Two main mechanisms are recognised, depending on the existence or not of a latent period preceding the onset of symptoms. This period of latency can range from weeks to years. In the majority of cases, immunological hypersensitivity mechanisms appear responsible. Occupational asthma with latency is often due to an immunological mechanism, particularly where asthma develops in response to exposure to a high molecular weight agent, such as an animal or vegetable protein. This immunological mechanism, known as sensitisation, is also likely to be involved in asthma induced by low molecular weight agents, such as isocyanates, although specific scientific evidence of this is often lacking.

Occupational asthma without latency may occur in response to extremely high levels of exposure to irritant gases or vapours, which usually have a low molecular weight. Acute toxicity due to accidental respiratory exposure to toxic chemicals is the initiating event and inflammatory mechanisms appear to be important in this irritant-induced asthma, which can also be known as Reactive Airways Dysfunction Syndrome (RADS). It is not clear whether or not a new low level exposure to the irritant agent may lead to asthma symptoms. Chlorine and ammonia are two of the main etiological agents of such irritant-induced asthma.

In addition to the two mechanisms described above, another type of work-related asthma is work aggravation of pre-existing asthma. In this situation, an individual with pre-existing asthma who is exposed to factors in the workplace such as non-specific gases or fumes, smoke or cold dry air may have an asthmatic attack precipitated by these factors. This condition is known as work-aggravated asthma. In this case, the exposure is not likely to cause asthma by itself or to worsen the airways responsiveness, but it is considered work-related in that it is exacerbating a pre-existing condition. Therefore, it is distinguished from asthma that is specifically occupational, i.e. which is caused by a sensitising agent encountered at work.

Occupational asthma is the most common occupational lung disease in developed countries, as shown by the results of physician notification schemes for occupational respiratory disease, which have been

established in several western countries, including the UK, Finland, Canada, the USA, South Africa and Australia. While reliable figures are difficult to find, it has been estimated that up to 15% of new asthma in adults are directly attributable to occupational exposures (http://www.nationalasthma.org.au/publications/amh/st_occupational.htm) (Blanc and Toren 1999).

The number of workplace chemical agents known to cause occupational asthma has been increasing, with over 450 known agents (Hendrick and Burge in Hendricks et al 2002). Unlike the agents which cause pneumoconiosis, which tend to occur in a limited number of industries, asthmagenic agents are found in a large variety of different industries and occupations within those industries. The following table shows some of the more common causes of occupational asthma and some of the more common occupations at risk.

Table 2. Common causes of occupational asthma

	Causes	Occupations at risk
High molecular weight	Animal excreta	Farmers, animal laboratories staff
	Latex	Health care workers
	Flour	Bakers, millers
	Enzymes	Food and detergents processing
	Vegetable gums	Pharmaceutical industry
Low molecular weight	Wood dust	Sawmill workers, joiners, cabinetmakers
	Isocyanates	Spray painters, insulation workers, polyurethanes work
	Formaldehyde, glutaraldehyde	Healthcare workers
	Alkaline persulfates	Hairdressers
	Colophony	Welders
	Metal fumes (nickel, chromium, platinum)	Metal work, galvanizing

Removal from exposure to the sensitizing agent may lead to remission of occupational asthma, although sensitization is usually permanent. If diagnosis is delayed due to the causative agent not being identified and exposure continuing, then the affected worker can develop respiratory symptoms from non-specific exposures unrelated to the initial sensitiser. (Smith et al 1999)

4.5 Chronic Obstructive Pulmonary Disease (including emphysema and/or chronic bronchitis)

Chronic Obstructive Pulmonary Disease (COPD) is defined as a disease state characterised by the presence of airway obstruction due to chronic bronchitis, emphysema or chronic asthma (asthma has been considered separately). This definition emphasizes the presence of chronic airflow obstruction, rather than any specific pathological process or aetiology. Chronic bronchitis is defined clinically as a productive cough which persists for several months over a period of at least 2 years. Emphysema is defined pathologically as an increase in the size of alveolar air spaces. The associated loss of elasticity causes small airways to collapse, which increases resistance to airflow.

Surveys of adults in the general population show that COPD is more prevalent in men than in women. Usually between 10 % and 20 % of adult men meet the criteria of chronic bronchitis. In Australia, a recent survey in Melbourne showed that chronic bronchitis was reported by 12.0 % and emphysema by 1.2 % of adults aged 45-69 years (Abramson et al 2002).

Although initial research on COPD focused on the role of respiratory infections and pollution as etiological factors, the importance of smoking on COPD was soon established. These findings have diverted attention away from other possible etiological agents. Occupational COPD has no characteristic clinical features which allow it to be distinguished from COPD caused by cigarette smoking or other non-work factors.

More recently epidemiological investigations have reported excess rates of COPD in a variety of industrial settings. The potential contribution of the occupational environment to the development of COPD has been established with a reasonable degree of certainty for a few exposures, such as mineral particles (coal, silica, and cobalt), organic dusts (cotton, grain, and wood dust), gases and fumes (isocyanates, welding fumes, ammonia, and chlorine). Chronic bronchitis is common among coal miners. Coal dust seems to have an adverse effect on ventilatory function independently of simple pneumoconiosis. In the same way, the association between cumulative silica dust exposure and airflow obstruction is independent of silicosis (Hnizdo and Vallyathan 2003). Cotton dust exposure is a cause of COPD known as byssinosis. Grain and wood dusts have been shown to cause a similar range of respiratory symptoms.

4.6 Other occupational respiratory conditions

There are several other, less common, forms of occupational respiratory disease, the more important of which are briefly described here.

4.6.1. Toxic pneumonitis

Toxic pneumonitis refers to acute or sub-acute disorders generally resulting from exposure by inhalation. The inhaled agents capable of causing toxic pneumonitis can be divided into chemical and organic agents.

Chemical agents include gases, vapours, and aerosols. Various clinical presentations can occur, however three main categories can be described. Inhalation fevers cover a group of 'flu-like syndromes' caused by metal fumes (most frequently after zinc heating) and less commonly by polymer fumes (after heating fluorine-containing polymers). The nature and the course of these fume fevers are benign and self-limiting. Acute chemical pneumonitis can be caused by a great variety of substances. Water-soluble irritants can affect the upper respiratory tract, causing acute laryngitis, tracheitis, or bronchitis. They can also cause eye irritation (formaldehyde, ammonia, hydrogen chloride). In contrast, poorly water-soluble gases are potentially the most hazardous because of the absence of an immediate irritant effect. As they are not absorbed by the upper tract, they reach the deep pulmonary region of the lung more easily and can lead to a delayed pulmonary oedema (nitrogen dioxide in silos, ozone, and phosgene).

Exposure to organic dust or aerosols can cause febrile reactions. Monday morning fever in cotton mills, grain fever, swine confinement fever, farmers fever have been known for decades. The clinical picture is of an influenza-like reaction. Typical occupations at risk are in the textile and grain industries and livestock farming, but every occupation associated with the handling of organic material can be at risk.

4.6.2. Hypersensitivity pneumonitis

This entity, also known as extrinsic allergic alveolitis, is a disease of the lung caused by an immune response to inhaled antigens in sensitised individuals. Hypersensitivity pneumonitis (HP) can manifest itself in an acute (febrile influenza-like reaction), subacute (progressive shortness of breath), or chronic way (chronic bronchitis-like). Most cases result from microbiological contamination when vegetable produce is stored without being adequately dried or is stored under damp conditions. In Australia, one type of HP, bagassosis, can occur in individuals working on sugar cane production. It is related to exposure to and manipulation of bagasse (sugar cane dust). The other more prominent types of HP are farmers' lung, mushroom workers' lung, and bird fanciers' disease.

5. OVERVIEW OF DISEASE BY EXPOSURE AND OCCUPATION GROUPS

Trends of prevalence and incidence of the three main groups of occupational respiratory disease appear to have been evolving in different

ways in developed countries. Pneumoconioses have generally stabilised or are on the decline, whereas occupational asthma has generally been increasing. COPD has more recently been identified as an emerging occupational respiratory disease and various potential exposures have been identified.

5.1. Pneumoconioses

Severity and prognosis of pneumoconioses are causes are generally well known and prevention measures have been taken in order to reduce their prevalence. However, as the latency period for this group of occupational respiratory disease can be very high, it is expected that new cases will continue to be diagnosed in the future.

Because of its mechanical properties, asbestos has been widely used in construction, mechanics, and insulation. Current and previous exposure to asbestos can mainly be encountered in construction activities, including all occupations related to construction repair, maintenance and renovation, or demolition of structures potentially containing asbestos (builders, electricians, plumbers, carpenters, insulation workers…).

Exposure to free crystalline silica can occur in occupations where rock or stones are processed. However, manipulation or production of silica and sand powder is also harmful, as well as high-temperature process of non-crystalline silica (see section 4.3.1). Lower exposure to silica is also known as a cause of COPD.

In coal miners, the most harmful clinical expression of exposure to coal dust is pneumoconiosis which can be worsening by concomitant exposure to silica. Chronic exposure to coal dust can also lead to COPD.

5.2. Asthma

Unlike pneumoconioses for which a few exposures cause the majority of disease, several hundred workplace agents have been related to occupational asthma. Therefore, asthma can be encountered in many different types of occupations and workplaces (some examples are given in section 4.4). However, wood dust and isocyanates are the most commonly reported causative agents in the SABRE Victoria notification scheme. In woodworkers, occupational exposure to wood dust has been well documented as a cause of asthma, hypersensitivity pneumonitis, and lung function impairment. Previous studies have reported a dose-relationship between respiratory function and dust exposure. Occupations exposed to wood dust include sawmill workers, joiners, carpenters, cabinetmakers. Isocyanates are used mainly in polyurethane foams manufacture and in two-part polyurethane paints. Wheat flour in bakers and millers, and latex in health care workers are two other main aetiological agents for occupational asthma.

In the UK, the SWORD/OPRA surveillance schemes estimated the average annual incidence over the three years 2001-2003 as around 3 cases per 100 000 workers per year. Isocyanates were the most commonly cited agents, with flour/grain being the second most common agent.(McDonald et al 2000) The occupations with the highest incidence rate of occupational asthma as reported to chest physicians were 'vehicle spray painters', 'bakers, flour confectioners' then 'moulders, core makers, die casters'. The industries with the highest incidence rate of occupational asthma were 'manufacture of basic metals' then 'manufacture of motor vehicles, trailers and semi-trailers' (http://www.hse.gov.uk/statistics/causdis/asthma.htm). The British Occupational Health Research Foundation has recently issued guidelines on prevention, identification, and management of occupational asthma (http://www.bohrf.org.uk).

In the USA, the 2002 Work-related Lung Disease (WoRLD) Surveillance Report (http://www.cdc.gov/niosh/docs/2003-111/2003-111.html) provides data on occupations and agents related to occupational asthma. Agriculture production and livestock, and farm machinery and equipment were associated with the highest proportionate mortality ratios (PMR) for the 1990-1999 period (1.51 and 1.51 respectively), followed by child day care services (1.40). Half of the occupational groups associated with significantly elevated PMRs for asthma were related to health care and education. The two most frequently reported types of agents associated with cases of occupational asthma in the USA between 1993 and 1999 were cleaning materials and mineral and inorganic dusts. Isocyanates accounted for nearly 10 % of the agents related to occupational asthma.

Exposure to silica and coal in miners can lead to COPD, as stated previously. Some occupational categories such as textiles workers have a high risk of COPD. Byssinosis due to cotton dust has been described. Many other potential occupational exposures are suspected but further studies are needed in this area. Some recent research at Monash University has shown that workplace biological dust exposure is an important cause of COPD (Matheson et al 2004).

6. ESTIMATES OF THE BURDEN OF OCCUPATIONAL RESPIRATORY DISEASE

6.1. Introduction

A review of available data sources shows that there is limited information on the extent of work-related respiratory disease in Australia. The incomplete information that is available comes from a variety of sources, including published studies; workers' compensation claims data, the Dust Diseases Board of NSW and the two SABRE programs in Victoria and NSW. SABRE stands for Surveillance of Australian workplace-Based Respiratory Events, comprising physician notifications of cases of occupational

respiratory disease cases in those two states. Published general practitioner and hospital presentation data sources don't provide useable information, because respiratory disease cases are included in categories that also contain such diseases not related to work. Available information for specific industrial sectors, where available, is also summarised here.

6.2. Population-based estimates

Overall estimates

Estimation of occupational contribution to respiratory disease is difficult because an occupational disease can easily be attributable to other concomitant factors, unless it is specifically related to a unique workplace causative factor or it can be differentiated by its clinical features.

Internationally, a review of published papers found a range of PARs for occupational asthma of between 2% and 33%, with a median of 9%. (Blanc and Toren 1999) A more recent estimate from the USA, based on data collected as part of the third National Health and Nutrition Examination Survey, estimated that 36% of asthma is occupational in origin, although this is at the high end of generally accepted estimates. (Arif et al 2002) Leigh et al assumed a PAR of 15% for each of occupational asthma and COPD, in estimating the costs in the USA to be $US1.6 billion for asthma and $US5 billion for COPD due to workplace causes. (Leigh et al 2002) Although there is considerable uncertainty in these kinds of figures, they suggest that occupational respiratory diseases are likely to have serious health, economic and social implications in countries like Australia.

In Australia, a proportionate attributable risk approach has previously been used to estimate the magnitude of premature mortality induced by exposure to hazardous substances in the workforce (Morrell et al 1998). The estimated age-adjusted mortality rates (expressed in number of deaths per million per year) were estimated to be 5 and 2 for asthma, and 8 and 0 for dust diseases, respectively in men and in women. However, these estimates only addressed mortality, not morbidity. As most cases of non-malignant occupational respiratory disease do not result in death, mortality estimates underestimate the true extent of these conditions.

More recently, the burden of occupational disease was estimated in New-Zealand, a country with a similar industry base to Australia (Driscoll et al 2004). An attributable fraction of 1.9 % to 3.1 % was estimated for occupational asthma, with isocyanates considered the largest category of causal agents. A wide range of other agents was found, including proteins, flour, wood dusts, formaldehyde, glutaraldehyde, epoxy resins, colophony, and detergent enzymes. In addition, a cross-sectional population-based study on individuals aged 20-44 years in New Zealand found an attributable fraction of 19.3 % for COPD from work (Fishwick et al 1997).

6.3 Workers' compensation-based estimates

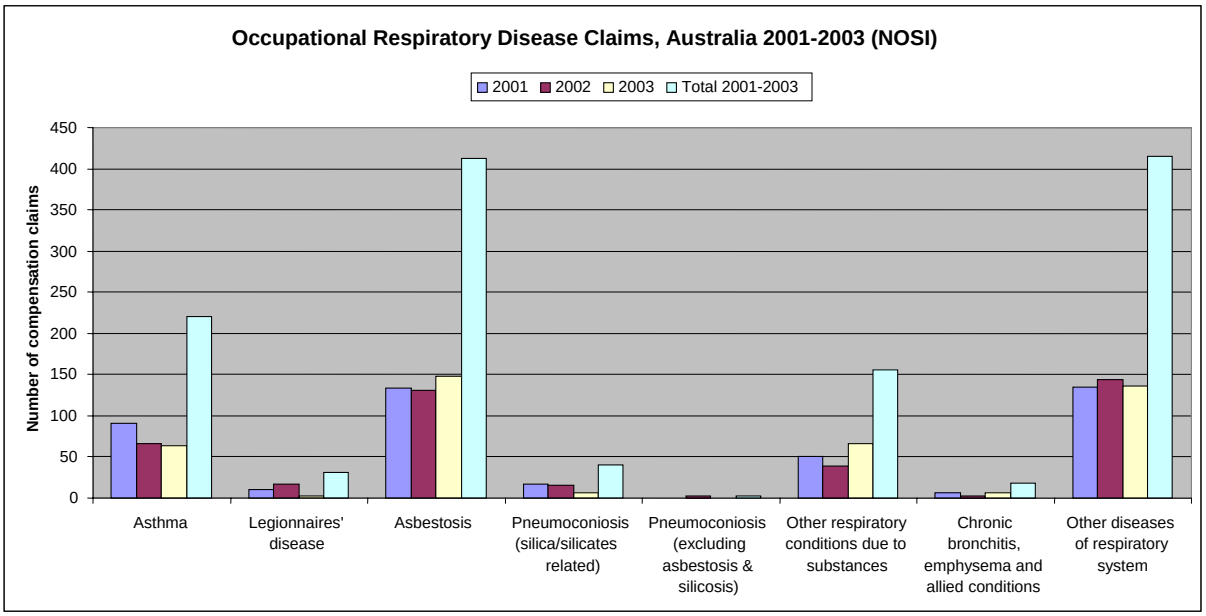
Workers' compensation data systems are widely known to have serious limitations in providing reliable data on rates of work-related respiratory disease. One reason is that published workers' compensation information at a national level in Australia only includes cases that result in five or more days off work. Many serious occupational respiratory disease cases will probably result in this much time off work, but many respiratory disease cases will not be serious enough to do so. A sizeable (but unknown) proportion of respiratory disease cases will not be formally diagnosed or will occur in workers after they leave work, in which case the connection to work is unlikely to be established and a workers' compensation claim is unlikely to be made. Also, a sizeable minority of workers has been shown not to be represented in Australian workers' compensation statistics (Macaskill and Driscoll 1998).

Figure 2 shows the numbers of cases of accepted claims for occupational respiratory diseases in Australia over the three year period of 2001-2003. These figures strongly suggest that the workers' compensation cases are significant underestimates of the number, probably by a factor of at least ten, for at least some of the types of work-related respiratory disease cases, based on information from other sources. The most common occupational respiratory disease in Figure 2 is asbestosis, the numbers of which were fairly constant over each of the three years. The next most common specific condition was asthma, while other specific conditions, such as pneumoconiosis and chronic bronchitis were very uncommon. Two of the more common categories are 'Other respiratory diseases due to substances, and 'Other diseases of respiratory system', but there is insufficient detail available about what conditions are included in these broad categories to provide any meaningful information.

When some specific compensation claim subgroups are reviewed, claims are nearly twice as higher in men than in women. They are in mainly in the categories 'asbestos-related disease' and in 'other diseases' in men. In women, claims are mainly related to 'other diseases' followed by 'asthma'. The three industries with higher disease claims are manufacturing followed by education, and health and community services. In manufacturing, asbestos related-disease is the main disease group (233 claims) whereas asthma is the second group (59 claims). In education and health and community services, claims are mainly in the 'Other diseases of the respiratory system' group. When considering occupation groups, the higher number of claims is in professionals, associate professionals and labourers respectively. Professionals' claims are mainly in 'Other diseases of the respiratory system' while asbestos-related disease is the main disease in associate professionals and labourers. Although there is insufficient available information to investigate this properly, it is likely that many of the claims in the professional and educational sectors relate to work aggravation of respiratory diseases, rather than true occupational respiratory disease.

Therefore, there should be caution in the use of compensation data to inform prevention efforts for occupational respiratory disease

Figure 2. Occupational Respiratory Disease claims, Australia, 2001-2003 (NOSI)



6.4. Dust Diseases Board of NSW data

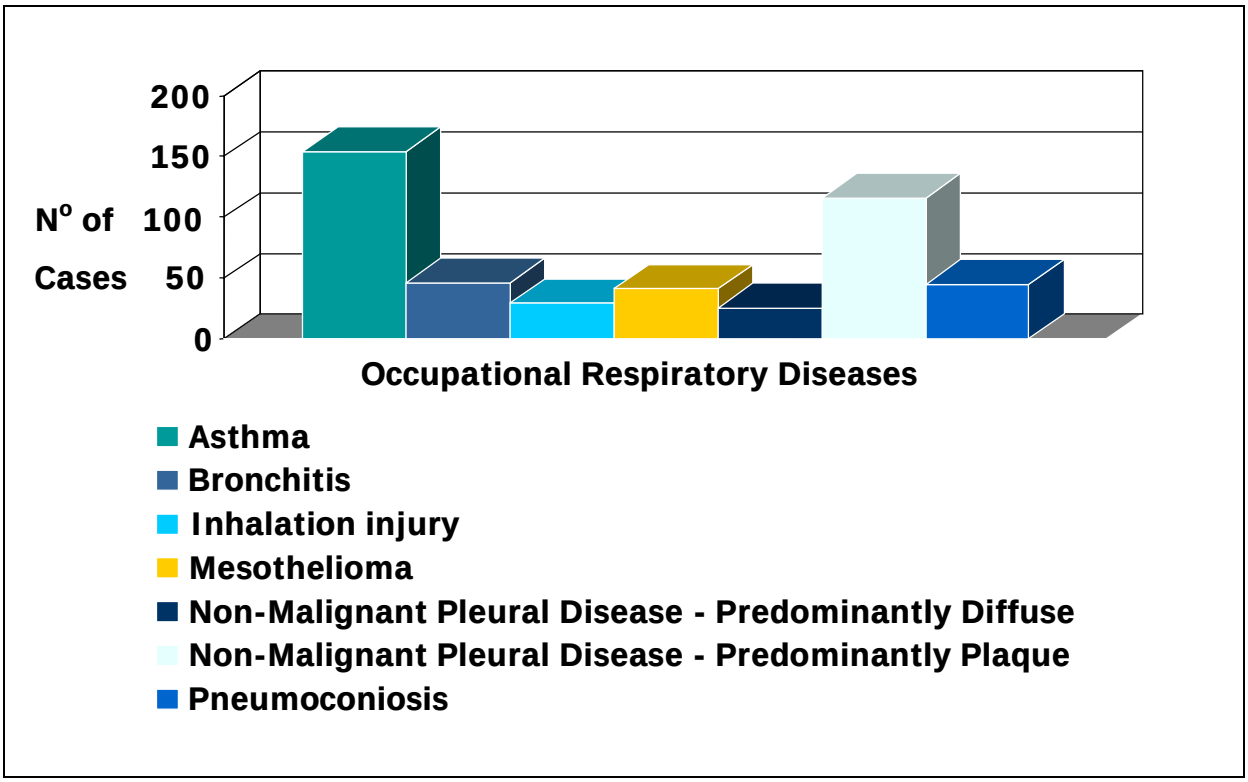
Review of the most recent annual report of the NSW DDB for the period 2002-2003 shows some interesting comparisons with the NOSI data. A total of 186 NSW workers received compensation payments from the Dust Disease Board for silicosis in the financial year 2002-03. However, for the three-year period 2001-2003, nationally across all states and territories, only 37 silicosis claims were recorded in the NOSI data. This indicates considerable under-ascertainment of the true number of silicosis cases. This is one example of a serious trend in workers' compensation data, where there seem to be serious under-reporting in published figures and insufficient linkage to other useful data sources, such as the DDB of NSW.

6.5. Physician notification programs in Australia

According to the SABRE notification scheme, (Figure 3) the most common condition reported by physicians in Victoria and Tasmania is asthma, comprising 33% of the occupational respiratory events reported. (Elder et al 2004). The asthma incidence rate is 30.9 (95 % confidence interval = 26.8-35.5) per million workers per year with a 2.4 times higher incidence rate in men compared to women. However, this incidence rate is likely to be underestimated, as SABRE in Victoria and NSW has incomplete coverage of physicians who see cases. The two most commonly reported causative agents for asthma in the SABRE (Victoria) notification scheme are wood dust and isocyanates (13.5 % and 5.8 % respectively). The finding of asthma as the most commonly reported occupational respiratory

disease is similar to that found in overseas physician notification schemes. The next most commonly reported condition in Victoria and Tasmania is non-malignant pleural disease from asbestos exposure.

Figure 3. SABRE notifications in Victoria 1999-2002



However, figures from the SABRE program in NSW show that the spectrum of reported cases of occupational respiratory diseases is quite different from that in Victoria. (Johnson et al 2004). Occupational asthma is an uncommon disease to be reported, whereas the most common conditions are dust related, especially pneumoconiosis. There may be several reasons for these large regional differences, which are unlikely to reflect true differences in the occurrence of these conditions. One important factor may be doctors who take part in SABRE in the two states having a different spectrum of patients in their practice. Another may be the fact that dust diseases have a higher recognition factor in NSW than Victoria, due to the existence of the Dust Diseases Board. It will be important to identify and address such sources of variability in state figures, if we are to develop a reliable national surveillance system for occupational respiratory diseases in the future.

7. APPROACHES TO PREVENTION OF OCCUPATIONAL RESPIRATORY DISEASE

According to the above figures, which although incomplete and show some inconsistencies between different data sources, suggest that the health, social and economic impact of occupational respiratory disease in Australia is higher than generally recognised. In the compensation figures, diseases of the respiratory system had the next higher average direct cost ($36 200 per new disease case) after neoplasm ($76 200). This can be explained by the fact that a high proportion of these cases either result in a fatality or a permanent incapacity. Therefore, it is important that prevention programs be put in place to reduce this burden.

Some general principles relevant to prevention of occupational respiratory disease, based on the hierarchy op controls, are briefly reviewed in this section:

- Identification of the substances used at the workplace and determination of the degree of exposure by measurement of the work environment. Material Safety Data Sheets (MSDS) may be useful when available, but they are not always reliable.

- Substitution with a less hazardous substance whenever possible; elimination of exposure by improving production processes and work methods; control of exposure (by the use of local exhaust ventilation systems for example). Those strategies are aimed to eliminate or minimize workers' exposure.

- Use of personal protective equipment (PPE) where substitution or elimination is not feasible or where other control measures are insufficient, by themselves to reduce exposure to acceptable levels.

As occupational respiratory diseases are difficult to identify and are therefore underestimated, their prevention is likely to be improved by complementary actions such as:

- Education on occupational disease to develop awareness. Information on specific occupations or industries at risk may be disseminated, focusing particularly on employers, workers, as well as on physicians:

    - Employers and workers, particularly in small and medium-size workplaces where asthmagenic agents are commonly found, may not always have sufficient toxicological information available to them about substances they use and may not have access to OHS expertise to advise them.

    - Physicians are likely to benefit from an increased focus on occupational history taking as part of their medical and/or specialist training. Many medical courses in Australia have

been found to include insufficient occupational medical training (Shanahan et al 2000). An early diagnosis can lead to withdrawal of a patient from harmful exposure and improve the prognosis of the disease, especially for occupational asthma.

- Improvement and increasing coverage of surveillance schemes, involving disease and exposure information, can help us to identify industries and occupations at risk and assist in more effective targeting of prevention activities by jurisdictions and industry.
- Research projects can supplement surveillance activities by identifying new hazards, better evaluating the health risks to workers, helping in identifying acceptable workplace exposure limits and assessing the effectiveness of interventions.
- Review of workplace occupational exposure limits on an ongoing basis, according to reliable surveillance data and that from other reliable sources.

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10. GLOSSARY

COPD Chronic Obstructive Pulmonary Disease

CWP Coal workers' pneumoconiosis

HP Hypersensitivity pneumonitis

MSDS Material Safety Data Sheet

NDS National Data Set for Compensation-based Statistics

NOHSC National Occupational Health and Safety Commission

NOSI NOHSC Online Statistics Interactive

OHS Occupational Health and safety

PAF Population Attributable Fraction

PAR Population Attributable Risk

PMR Proportionate Mortality Ratio

PPE Personal Protective Equipment

RADS Reactive Airways Dysfunction Syndrome

SABRE Surveillance of Australian workplace-Based Respiratory Events