

# Occupational lung diseases in Australia 2006–2019

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## Glossary

|                  |  |
|------------------|--|
| ABS              | Australian Bureau of Statistics                                    |
| AFOEM            | Australasian Faculty of Occupational and Environmental Medicine    |
| AIHW             | Australian Institute of Health and Welfare                         |
| ANZCOTR          | Australia and New Zealand Cardiothoracic Organ Transplant Registry |
| ANZSOM           | Australian and New Zealand Society of Occupational Medicine        |
| CMDLD            | Coal mine dust lung disease  |
| COPD             | Chronic obstructive pulmonary disease                              |
| CWP              | Coal worker's pneumoconiosis                                       |
| DALY             | Disability adjusted life year                                      |
| DNRME            | Department of Natural Resources, Mines and Energy                  |
| ECRHS            | European Community Respiratory Health Survey                       |
| FEV <sub>1</sub> | Forced expiratory volume in one second                             |
| FVC              | Forced vital capacity  |
| HP               | Hypersensitivity pneumonitis                                       |
| ICD-10           | International classification of disease, 10 <sup>th</sup> edition  |
| ILD              | Interstitial lung disease  |
| IPF              | Idiopathic pulmonary fibrosis                                      |
| JEM              | Job exposure matrix  |
| MAQOHSC          | Mining and Quarrying Occupational Health and Safety Committee      |
| MDLD             | Mine dust lung disease   |
| MeSH             | Medical subject heading  |
| NDI              | National Death Index   |
| NDS              | National Data Set for Compensation-based Statistics                |
| NIOSH            | National Institute for Occupational Safety and Health              |
| NSW              | New South Wales  |
| OIR              | Office of Industrial Relations                                     |
| OLDs             | Occupational lung disease  |
| PAR              | Population attributable risk                                       |
| PMF              | Progressive massive fibrosis                                       |
| PRISMA           | Preferred Reporting Items for Systematic Reviews and Meta-Analysis |
| RCS              | Respirable crystalline silica                                      |
| SA               | South Australia  |
| SABRE            | Surveillance of Australian Workplace Based Respiratory Events      |
| SWA              | Safe Work Australia  |
| SWORD            | Surveillance of Work-related and Occupational Respiratory Disease  |
| TAHS             | Tasmanian Longitudinal Health Study                                |
| TSANZ            | Thoracic Society of Australia and New Zealand                      |
| UK               | United Kingdom   |
| USA              | United States of America   |
| WA               | Western Australia  |
| WHS              | Work, health and safety  |
| WRA              | Work-related asthma  |
| YLD              | Years lived with disability  |

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## Executive summary

Occupational lung diseases (OLDs) from exposures at work comprise a wide range of work-related diseases which historically have resulted in a considerable health burden amongst workers in many countries around the world, including Australia. This review of OLDs in Australia updates a previous Safe Work Australia (SWA) report from 2006 (the 2006 Review).

This review aims to:

1. provide an overview of the extent of OLDs in Australia, commenting on changes in extent of OLDs and relevant data sources since 2006
2. review and comment upon existing data sources related to the reporting of OLDs in Australia
3. estimate the incidence of OLDs highlighting key 'at risk' areas, and
4. analyse any significant trends in OLDs between 2006 and 2019.

The OLDs included in this review are work-related asthma (WRA), chronic obstructive pulmonary disease (COPD), coal workers' pneumoconiosis (CWP), silicosis, asbestosis, mixed pneumoconiosis, hypersensitivity pneumonitis (HP), toxic pneumonitis and alveolar proteinosis. This report does not cover lung cancer, mesothelioma or work-related lung infections in Australian workers.

The information presented in this report comes from three sources:

1. published peer-reviewed literature since 2006 that included Australian OLDs data (56 peer-reviewed publications)
2. 'grey' literature on OLDs made available since 2006, including conference proceedings, and/or abstracts, technical briefs, theses and dissertations, government documents, and other online reports (23 grey literature items), and
3. Australian de-identified, aggregated OLD data from health data custodians, coordinators, jurisdictions and professional societies across Australia including data from the National Data Set for Compensation-based Statistics (NDS).

The main findings of this review are that:

- Identified cases of pneumoconiosis, especially CWP from coal mine work and accelerated silicosis from working with engineered stone, have increased substantially in Australia since the 2006 Review, when they were thought to be declining. The current health screening of engineered stone workers in many states and the establishment of a National Dust Disease Taskforce demonstrate the increasing national importance of this OLD.
- Claims for asbestos-related OLDs, such as asbestosis, continue to decline in Australia in line with reductions in asbestos use after it was banned in 2003. However, deaths from asbestosis continue to increase which could be a function of the long latent period.
- WRA appears to be declining in Australia, largely based on declining compensation claims. However, between nine and 15 per cent of adult asthma cases are likely to be work-related.

Estimates using a population attributable risk (PAR<sup>1</sup>) approach suggest that the number of compensation claims are an under-estimate of the extent of this OLD.

- COPD is becoming an increasingly important type of OLD as more associations with work-related exposure are being identified, but Australian data sources are yet to reflect this increasing importance. Work-related COPD is likely to become more widely recognised in Australia as smoking rates decline.
- There are few available Australian data on other OLDs, such as HP. Overseas data and the spectrum of Australian work with potential exposure suggest they are more common than current data indicates.

Overall trends over time in prevalence, incidence and annual mortality from OLDs are difficult to assess for a number of reasons, including the incompleteness of data sources. The estimates of the extent of OLDs using epidemiological information and health screening data are not adequately precise to allow the accurate assessment of year-on-year-changes. Despite these limitations, Australia has experienced fewer compensated cases of WRA and asbestosis, suggesting a downward trend. However, there has been an increase in cases of CWP in coal miners and of silicosis, especially in engineered stone workers.

The key message that arises from the summary of findings presented above is that OLDs in Australia continue to be a major health concern that requires continuing research, education and awareness of the ongoing presence of these diseases and their causative exposures in workplaces. In addition, the available data demonstrate that OLDs in Australia are a greater health concern than they were at the time of the 2006 Review.

There is also a need for improvement in, and increasing coverage of, OLD data sources. There are some short-term epidemiological studies of OLDs in Australia, but only for a limited number of industries. This review identified a range of other data sources on OLDs, including mortality and hospitalisation data, but these often do not provide relevant work exposure information. Therefore, there is a need for more longitudinal studies of high-risk workers, involving disease and exposure information, to better identify high risk industries and occupations and assist in more effective targeting of prevention activities.

There is also a need for more comprehensive routine respiratory health monitoring<sup>2</sup> where workers are exposed to hazardous substances and targeted health screening in emerging high-risk jobs. The establishment of a national dust disease registry for accelerated silicosis, which the National Dust Disease Taskforce has recommended in its interim report. This registry has been recommended to be

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<sup>1</sup> Population attributable risk is the proportion of the incidence of a particular disease in the population resulting from that exposure. The incidence of the disease in the population would be eliminated if the exposure were eradicated. The PAR is calculated by subtracting the incidence in the unexposed from the incidence in the total population (exposed and unexposed).

<sup>2</sup> Health monitoring means the collection of demographic data, a work history, medical history and a physical examination at baseline and then at appropriate intervals during a person's employment. Changes in health outcomes such as lung function can then be identified, and action taken. For example, reduction in or removal from exposure and, where necessary, early treatment or management of the disease.

developed with the potential to gather data for other occupational lung diseases and will be a major step forward in improving estimates of OLD incidence in Australia in the future.

In the absence of comprehensive national health monitoring data on OLDs, Australia is very reliant on compensation statistics to monitor trends in these important diseases. Compensation data have well-known limitations, especially for OLDs and other occupational diseases, and this highlights the importance of establishing a more comprehensive dataset.

Work-related exposures causing OLDs are an important and preventable cause of work-related mortality and morbidity and this review suggests they continue to make a substantial contribution to the burden of lung diseases in Australia. There is a pressing need to improve our data sources on OLDs to more effectively monitor trends over time and provide regulatory agencies, industry, workers and occupational health professionals with the necessary information to detect emerging respiratory threats and better monitor the effectiveness of prevention programs.

# **Chapter 1**

## **Introduction**

This chapter provides an overview of the aims and scope of the report, brief descriptions of the various OLDs included in this report and considerations relevant to decisions about when a lung disease can be identified as an OLD.

This report contains findings from a review of the peer reviewed and grey literature and data sources relevant to OLDs in Australia and updates the 2006 Review [1]. It has been undertaken to assist SWA, as the agency responsible for national work health and safety (WHS) policy.

## 1.1. Aims of the review

This review aims to:

1. provide an overview of the extent of OLDs in Australia, commenting on changes in extent of OLDs and relevant data sources since 2006
2. review and comment upon existing data sources related to the reporting of OLDs in Australia
3. estimate the incidence of OLDs highlighting key 'at risk' areas, and
4. analyse any significant trends for OLDs between 2006 and 2019.

The scope of this report is limited to non-malignant and non-infectious OLDs in Australia. As such, conditions including mesothelioma, lung cancer, tuberculosis and legionnaires' disease, are outside the scope of this report.

## 1.2. Occupational lung diseases

Exposures at work can be an important determinant of lung health and can affect any part of the respiratory tract, from the nose to the alveoli [2].

The four main groups of OLDs considered in this report are:

- WRA, which includes occupational asthma and work exacerbated asthma
- COPD, which includes chronic bronchitis and emphysema
- pneumoconioses (fibrotic lung diseases):
  - CWP (black lung)
  - silicosis, which includes acute, accelerated and chronic forms
  - asbestosis
  - other, usually mixed, pneumoconiosis, and
- other OLDs
  - HP (or extrinsic allergic alveolitis)
  - toxic pneumonitis
  - alveolar proteinosis.

The exposures responsible for the above OLDs can be found in a variety of different industries and jobs within those industries. A 2017 paper by Hoy and Brims reviewed some of the more common exposures and jobs at risk for developing OLDs in Australia and these are considered in the descriptions of OLDs below [2].

### 1.2.1 Work-related asthma

Asthma is one of the leading causes of preventable mortality and morbidity and has a significant impact on Australia's health and productivity [2]. WRA is known to be an increasingly common form of OLD in many industrialised countries, where the burden of other OLDs is reducing [3]. WRA comprises two forms: occupational asthma and work-exacerbated asthma [4, 5]. Occupational asthma is typically defined as asthma developing *de novo* in someone who has no pre-existing asthma symptoms, as a result of a particular work exposure [6]. Work-exacerbated asthma is the worsening of asthma control because of work-related exposure factors [4, 5].

Most cases of occupational asthma are allergic in nature and involve sensitisation. There is often a latent period between first exposure to a respiratory sensitiser and the onset of respiratory symptoms [1]. Once sensitisation has occurred, the affected worker may then react to much lower levels of exposure to the sensitiser or to other, nonspecific triggers (such as cold air or exercise). Sensitiser-induced occupational asthma develops because of immune-mediated sensitisation to an occupational agent (i.e. to an asthmagen) [2].

Some cases of occupational asthma are irritant-induced rather than sensitiser-induced. Irritant-induced asthma is less common than sensitiser-induced occupational asthma [2]. Irritant-induced asthma is a non-immunological condition that develops following exposure to certain irritant substances at the workplace. These include high-level exposure to fumes, gases, sprays, dusts with irritating properties, spills of volatile compounds, and accidental release of irritants under pressure [2, 7]. It may also develop after repeated low-level exposure to irritants, such as is experienced by professional cleaners, who are exposed to a large variety of industrial cleaning agents [2, 8].

Work-exacerbated asthma typically refers to pre-existing asthma that is worsened, but not caused by, a work-related exposure [2]. Work-exacerbated asthma has received less attention in terms of research and prevention efforts compared to occupational asthma [9]. A wide range of work-related exposures can exacerbate pre-existing asthma symptoms, including chemicals, dusts or allergens. Asthma may also be exacerbated by other types of workplace conditions, such as temperature, emotional stress, and physical exertion [4, 10].

Over 300 known or suspected workplace agents can cause WRA [11]. Exposure to over 250 astmagens was investigated in an Australian study that identified likely asthmagen exposure from the work reported by general population participants in 2016 [8]. The most frequently encountered exposures included:

- wheat flour
- bioaerosols
- latex
- industrial cleaning agents
- sterilising agents, and
- wood dusts.

It is valuable to consider the more common exposures found in overseas research. Data from the Surveillance of Work-related and Occupational Respiratory Disease (SWORD) scheme in the United

Kingdom (UK) found isocyanates, flour and grain and wood dusts were responsible for the highest number of new cases of occupational asthma [12]. Workers in the agriculture, forestry, fishing, health services and social work industries were found to be at highest risk of WRA [4]. The surveillance data from the United States of America (USA) found the highest prevalence of WRA is in the manufacturing industry [13], followed by health care [13, 14] and cleaning [15].

A recently published large data linkage study in Ontario, Canada involving 575,379 participants from 2002 to 2013 identified increased risks of WRA among bakers, painters and decorators. From the job exposure matrix (JEM) analysis, they found increased risks of WRA for those exposed to flour dust and isocyanates [16]. Concrete finishers, some woodworker groups, and shipping and receiving clerks also showed elevated risk of developing asthma [16].

### **1.2.2 Chronic obstructive pulmonary disease**

COPD is a collective term describing conditions affecting the airways of the lungs and more severe forms of the disease can affect the lung alveoli, where oxygen transfer occurs. It is characterised by fixed airflow obstruction and defined by a reduction in the ratio between the forced expiratory volume in one second (FEV<sub>1</sub>) and forced vital capacity (FVC) [17].

COPD includes two main types: chronic bronchitis and emphysema. Chronic bronchitis is defined as a productive cough that persists for at least three months over a period of at least two years. Emphysema is defined as an increase in the size of alveolar air spaces within the lung without any fibrosis of the lung tissue [18, 19]. Changes in lung function over time is a significant predictor of COPD and therefore of interest for the early identification of this disease [20].

COPD is a long-latency disease, meaning that cases tend to develop many years after first exposure and after a prolonged period of exposure to the causative agent. Therefore, respiratory symptoms usually manifest during mid-life or later. Tobacco smoking is the predominant risk factor for COPD [21, 22], with work-related exposures estimated to account for 16 to 20 per cent of COPD [23]. The very strong association between smoking and COPD means that establishing occupational causation in a worker with a smoking history is often difficult. Inhalation of dust particles or gases at the workplace can initiate airway inflammatory processes by activating alveolar macrophages and leukocytes and by releasing reactive oxygen species in the airways, which can eventually result in lung function decline and COPD [24].

Several workplace agents and occupations have been implicated as being associated with an increased risk of COPD. Epidemiological studies have identified several jobs that are 'at risk occupations' for developing COPD, including:

- coal mine workers [10]
- farmers [25]
- timber workers [26]
- cotton textile workers [27]
- manufacturing workers [28, 29]
- industrial workers [30], and

- construction workers [31].

Several work-related exposures [32] have also been associated with increased risk of COPD including:

- grain dust [33]
- cotton dust
- endotoxins
- welding fumes
- flour dust
- biological dust [34, 35]
- mineral dust [36]
- pesticides [35, 37]
- solvents [38, 39], and
- wood dust.

Some studies have investigated longitudinal lung function decline typically in particular occupations, for example firefighters [40]. These changes may indicate the possible development of COPD [39], but lung function decline can also occur in the absence of a specific OLD, as a result of ageing or tobacco smoking.

### 1.2.3 Pneumoconioses

The pneumoconioses comprise a group of non-malignant parenchymal (interstitial) lung diseases caused by inhaling different types of dust particles. Several fibrotic lung diseases are classified as pneumoconioses and they are discussed below.

#### 1.2.3.1 Coal workers' pneumoconiosis (black lung)

Pneumoconiosis resulting from inhalation of coal dust is known as CWP or 'black lung' [41]. CWP is an untreatable but preventable OLD, caused by the cumulative inhalation of respirable<sup>3</sup> coal dust and the level of risk of developing the disease varies according to the composition of the coal dust [42].

CWP is a long-latency disease that gradually develops over a prolonged period of exposure to respirable coal dust. In general, a history of at least 10 or more years of exposure to respirable coal dust is required to develop CWP [43]. Dust particles usually less than 5 µm in diameter can pass through the lung airways (bronchioles and alveoli) into the lung parenchyma and cause the formation of inflammatory lesions containing coal dust, macrophages and fibroblasts [41, 43]. The initial stages of CWP are usually not associated with respiratory symptoms, with the later stages usually presenting with breathlessness and cough [44].

The level of risk of CWP in coal mine workers is dependent on the mine's ventilation and dust suppression strategies. Mine workers who are protected by working in enclosed machine cabins or those who work in open cut mines usually have less exposure to coal mine dust compared to those who

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<sup>3</sup> Respirable dust is the fine airborne particles (less than 10 µm), which can penetrate the respiratory tract, reaching beyond the terminal bronchioles and into the gas-exchange region of the lungs.

work in underground mines [41]. Exposure to residual dust from burned coal, also known as 'fly ash', can also occur in underground mines [45].

### **1.2.3.2 Silicosis**

Silicosis is a preventable but irreversible, progressive, chronic and diffuse OLD that can also lead to progressive massive fibrosis (PMF) [46]. Silicosis is caused by inhalation of respirable crystalline silica (RCS), a major constituent of rocks, sand, granite and more recently in engineered stone products (also known as 'artificial stone', 'composite stone' or 'reconstituted stone'). Stonemasons, bricklayers and workers involved in benchtop fabrication, manufacturing and installation who use this material are at high risk of exposure to RCS and are at risk of developing silicosis [47].

Three forms of silicosis have been described, depending on the intensity and duration of exposure to RCS and radiological features of the lungs. These are acute, accelerated and chronic silicosis.

- Acute silicosis develops after only a few weeks to five years of high exposure to RCS and rapidly progresses to respiratory failure [46]. It has occurred in sand blasters and silica flour workers [46].
- Accelerated silicosis develops within five to 10 years after the initial exposure to RCS and is also associated with high exposure for example in those working with engineered stone [46]. In accelerated silicosis, the progression to PMF can occur within a few years [46-48].
- Chronic silicosis has been the most common form of silicosis historically. It develops more slowly. At low exposure intensity, individuals may become symptomatic more than 10 to 20 years after first exposure [48].

The rate of development of all forms of silicosis depends on the concentration and the surface characteristics of the RCS particles [48].

Exposure to RCS is also associated with other diseases, such as lung cancer, tuberculosis and autoimmune diseases, usually in combination with silicosis [46, 49-54].

The following types of work can involve RCS exposure and one or more types of silicosis [47, 55, 56]:

- various forms of mining, such as coal and hard rock mining
- construction work
- tunnel work
- masonry
- sandblasting
- glass manufacturing
- ceramics work
- steel industry work
- quarrying
- natural and engineered stonework, and
- stone cutting.

### **1.2.3.3 Asbestosis**

Asbestosis is a form of pneumoconiosis resulting from inhalation of asbestos fibres [2]. It is a chronic and irreversible condition, in which symptoms typically start to develop after at least two decades of exposure to asbestos, depending on the level of exposure [57]. The symptoms usually progress over time to affect daily activities and can lead to fatal complications [58]. The rate of progression of asbestosis is related to the level of cumulative asbestos exposure. It is generally recognised that heavy exposure to asbestos fibres is required to develop asbestosis [59].

Asbestos-associated pleural plaques are benign thickened patches of the lining of the lungs which usually become radiologically visible within the first 10 years of exposure to asbestos. They calcify over time, but do not subsequently progress in size or extent. They are indicative of asbestos exposure but do not cause physical impairment [2].

Asbestos is a naturally occurring mineral found in rock, sediment or soil. In Australia, asbestos was commonly used from the mid-1940s until the late 1980s in different products including 'fibro' (asbestos cement sheets), flue pipes, drains, roofs, gutters, brakes, clutches and gaskets [2]. Asbestos usage in Australia was reduced after legislation was introduced in the early 1980s. Some types of work that used asbestos included cement production, ship-fitting, dockyard work and railway carriage maintenance. Historically, construction workers, heating engineers, boilermakers, railway workers and members of the defence force were exposed to asbestos. Construction and demolition workers and asbestos removalists are some of the jobs that could potentially have a significant risk of asbestos exposure [2, 60]. It is possible that some cases of asbestosis or other pneumoconioses could be labelled as idiopathic pulmonary fibrosis (IPF), when there has been insufficient history of the relevant workplace exposure collected [61].

### **1.2.3.4 Other pneumoconioses**

Inhalation exposure to other mineral or metallic dusts are associated with less common pneumoconioses. These dusts include, but are not limited to minerals such as graphite, talc, mica and kaolin, and metals, such as aluminium, antimony, barium, iron, beryllium, cadmium, chromium and cobalt [1, 62]. The clinical features are similar to the pneumoconioses described above. Mixed-dust pneumoconiosis has also been described. This results from inhalation of two or more of the agents known to cause pneumoconiosis, such as coal dust and silica [62].

Another uncommon type of pneumoconiosis is byssinosis, or 'Monday fever', which is associated with exposure to cotton, hemp or flax dust and has some differing clinical features [62]. Symptoms of byssinosis include dyspnea, cough, wheezing, and fever. Symptoms can improve once the worker is removed from the workplace, but typically get worse upon return to work as a result of re-exposure. Prolonged exposure may result in irreversible lung damage [63].

## **1.2.4 Other occupational lung diseases**

### **1.2.4.1 Hypersensitivity pneumonitis**

HP or extrinsic allergic alveolitis is an interstitial lung disease (ILD) caused by an immune response to

inhaled antigens [64, 65]. Several substances can trigger an individual's immune system, causing short- or long-term inflammation that makes it difficult for the lungs to work properly and can cause permanent damage [66]. Types of HP include bird fancier's lung, farmer's lung, hot tub lung, humidifier lung and mushroom workers' lung. It is characterised by acute flu-like effects and in some cases can also lead to serious longer-term effects on lung function [67]. Another type of HP, bagassosis, can develop in people working in sugar cane production, exposed to bagasse (sugar cane dust) [64].

#### **1.2.4.2 Toxic pneumonitis**

Toxic pneumonitis is an acute or sub-acute inflammation of the lungs resulting from exposure by inhalation of metal fumes or chemical agents, such as gases, vapours or aerosols [3]. Inhalation of metal fumes, such as zinc and less commonly of polymer fumes (for example after heating fluoride-containing polymers) [68] can result in influenza-like symptoms, for example metal fume fever. Exposure to a variety of other substances can cause acute chemical pneumonitis. Water-soluble irritants may affect the upper respiratory tract, causing acute laryngitis, tracheitis or bronchitis [66]. The clinical consequences of toxic pneumonitis include diffuse bronchiolar inflammation and obstruction, as well as alveolae filling with fluid (pulmonary oedema).

Work-related exposures to organic dusts and aerosols with microorganisms and endotoxins may lead to organic dust toxic syndrome and febrile reactions, respectively [69]. Typical jobs at risk are in the textile and grain industries and livestock farming, but anyone associated with the handling of organic material can be at risk.

#### **1.2.4.3 Alveolar proteinosis**

Alveolar proteinosis is a rare lung disease characterised by the alveolar accumulation of surfactant. The symptoms are non-specific; primarily dyspnoea and cough, and a restrictive pattern on lung function are typical [70]. Some work-related exposures such as RCS dust have been associated with higher risk of this disease [71].

### **1.3. Factors which can affect the identification of occupational lung diseases**

For several reasons, it can be difficult to link a specific work-related exposure to the development of an OLD in an individual, so a comprehensive work and exposure history is needed. Not identifying a contributing work exposure can lead to underestimates of OLDs. Important factors to consider when identifying a case of OLD include latency, multiple risk factors and presence of specific work-related features.

As shown above, work-related exposures may directly cause an OLD (for example CWP) or aggravate a pre-existing condition (for example work-exacerbated asthma) [4]. Some OLDs may develop after a few months of work or exposure (such as sensitiser-induced occupational asthma), but some OLDs, for example CWP, chronic silicosis and asbestosis, may only be diagnosed many years after first exposure, including after the worker has left the workplace [9]. These time delays in developing an OLD after the

start of exposure are known as latent periods. The wide variability in latent periods for different OLDs reduces the likelihood of workplace exposure(s) being recognised [72].

A further consideration is that respiratory symptoms may occur outside work hours making workplace attribution difficult. Of the listed OLDs, many have specific work exposures which cause them and are not contributed to by other factors outside the workplace, for example the pneumoconioses. For other OLDs, factors outside the workplace can be an important risk factor, such as cigarette smoking and COPD and the pre-existing asthma and work-exacerbated asthma [21]. A case of pneumoconiosis can be diagnosed as a different non-work lung condition, such as sarcoidosis or IPF, if the relevant work-related exposure is not identified. For many diseases, such as asthma and COPD, there are no pathophysiological differences between lung disease arising from work-related exposures and lung disease arising from non-work-related exposures [37].

Genetic variation is likely to account for some of the inter-individual differences in susceptibility to OLDs. This has been demonstrated for pneumoconioses [44] and for asthma [73]. In addition, epigenetic changes may also affect susceptibility, for example to asthma [74].

# **Chapter 2**

## **Methods**

This chapter outlines the methods and approaches used to acquire information on OLDs in Australia for this report. This review was conducted using a similar, but expanded, approach that was used for the 2006 Review [1]. The findings presented in this report are based on the following three sources:

1. a review of published, peer-reviewed literature from Australia containing Australian data on OLDs
2. a review of grey literature, including conference proceedings, and/or abstracts, technical briefs, theses and dissertations, government documents, and other online reports, and
3. a review of Australian data sources potentially containing information on OLDs or related findings.

## **2.1 Review of published peer-reviewed literature from a systematic electronic database search**

A comprehensive search strategy was developed using relevant Medical Subject Headings (MeSH) and keywords to identify published literature on OLDs in Australia. The search was written in such a way that articles were retrieved containing at least one keyword from all the groups (a), (b) and (c) (below) for those searches where Australian studies were to be identified. The search was conducted for articles published from 1 January 2000 to 30 June 2019 and was limited to English language studies. The year 2000 was chosen as the earliest year, to provide a short overlap in the years covered in the 2006 Review to ensure no relevant paper was missed [1]. The final search strategy was developed in conjunction with an experienced librarian at the Monash University Library and adapted as necessary to query the following nine electronic databases. Apart from the databases, follow-up of sources cited in reference lists were also considered.

- Ovid Medline
- Embase via Ovid
- PubMed
- Scopus
- Web of science core collection
- National Institute for Occupational Safety and Health (NIOSH)
- Occupational Health and Safety (OSH) reference database developed by the Canadian Centre for Occupational Health and Safety (CCOHS); the OSHLINE bibliographic database includes- OSHLINE, HSELINE, NIOSHTIC, NIOSHTIC-2, CISILO (Canadian ILO) and PubMed subset
- Ryerson International Labour Occupational Safety and Health, and
- International occupational safety and health information centre.

The searches used three groups of keywords.

- a) 'Pulmonary Disease' (MeSH) OR 'Chronic Obstructive' (MeSH) OR 'Lung Diseases' (MeSH) OR 'Obstructive' (MeSH) OR 'Asthma' (MeSH) OR 'Asthma, Occupational' (MeSH) OR 'Occupational Asthma' (MeSH) OR 'Bronchitis' (MeSH) OR 'Bronchiolitis' (MeSH) OR 'Pulmonary Disease' (MeSH) OR 'Pulmonary Emphysema' (MeSH) OR 'Cough' (MeSH) OR

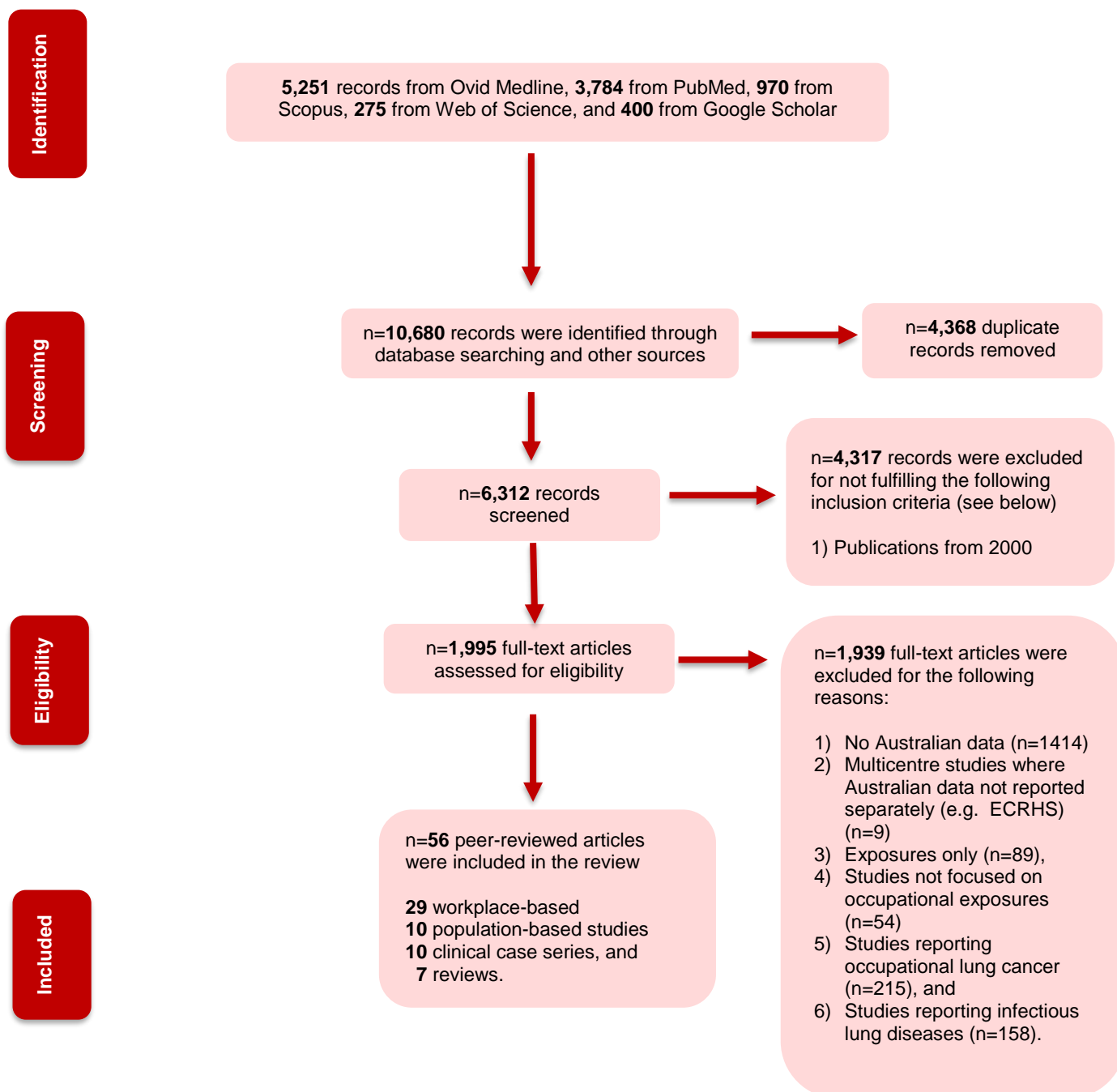
'Dyspnea' (MeSH) OR 'Pneumoconiosis' (MeSH) OR 'Bronchial Disease' (MeSH) OR 'Cystic Fibrosis' (MeSH) OR 'Chronic Airflow Obstruction' (MeSH) OR 'Obstructive Airway Disease' (MeSH) OR 'COPD' (MeSH) OR 'Occupational Lung Disease' (MeSH) OR 'Asbestosis' (MeSH) OR 'Berylliosis' (MeSH) OR 'Farmers Lung' (MeSH) OR 'Silicosis' (MeSH) OR 'Lung Fibrosis' (MeSH) OR 'Chronic Nonspecific Respiratory Disease' (MeSH) OR 'Occupational' (MeSH) OR 'Occupational Diseases' (MeSH) OR 'Inhalation Exposures' (MeSH) OR 'Occupational Exposures' (MeSH) OR 'Occupations' (MeSH) OR 'Pesticides' (MeSH) OR 'Solvents' (MeSH) OR 'Dust' (MeSH) OR 'Dust Exposure' (MeSH) OR 'Agriculture Workers Diseases' (MeSH).

AND

- b) 'Job' (text word) OR 'Jobs' (text word) OR 'Occupations' (text word) OR 'Industrial' (text word) OR 'Work' (text word).
- c) Keywords 'Australia', 'Australian' and 'Australians' were used in searches to ensure literature were directly related to Australian workplaces.

### **2.1.1 Selection of published peer-reviewed papers**

Records were imported into the bibliographic software Endnote™ X8.1 after the literature search of each database. A screening process was adopted for titles and abstracts and papers eligible for a full-text assessment were identified. The 10,767 articles identified through the searches were reviewed in two stages. Firstly, the title and abstract were screened to remove out of scope articles. Following the removal of 4,397 duplicates, the titles and abstracts of 6,370 records were screened against the predetermined inclusion and exclusion criteria (Table 1). This left 1,995 articles where the full text was obtained and reviewed in full, after which further articles were rejected as out of scope. Studies were also included for full-text assessment where there was uncertainty. For example, if it was not clear from the title and/or abstract whether the article included data on OLD. The process was repeated after obtaining full-text versions of the selected titles and abstracts. The remaining 56 relevant publications were retrieved and are included in this report. The Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) flowchart of article selection is given in Figure 1.



**Figure 1** PRISMA flowchart of peer-reviewed article selection process [75]

## 2.2 Review of grey literature

Secondary searches were undertaken to include information beyond that found in formally published, peer-reviewed literature sources. Such information, collectively known as 'grey' literature, includes conference proceedings, and/or abstracts, technical briefs, theses and dissertations, government documents and other online reports. Some of the grey literature could be identified from searching the above electronic databases. We additionally searched in internet search engines such as Google (using the advanced search platform), Google Scholar, Grey Matters, Open Grey, Informit and Trove (National Library of Australia).

We further explored several reports including chronic respiratory conditions identified on the Australian Institute of Health and Welfare (AIHW) website [76]. These reports were:

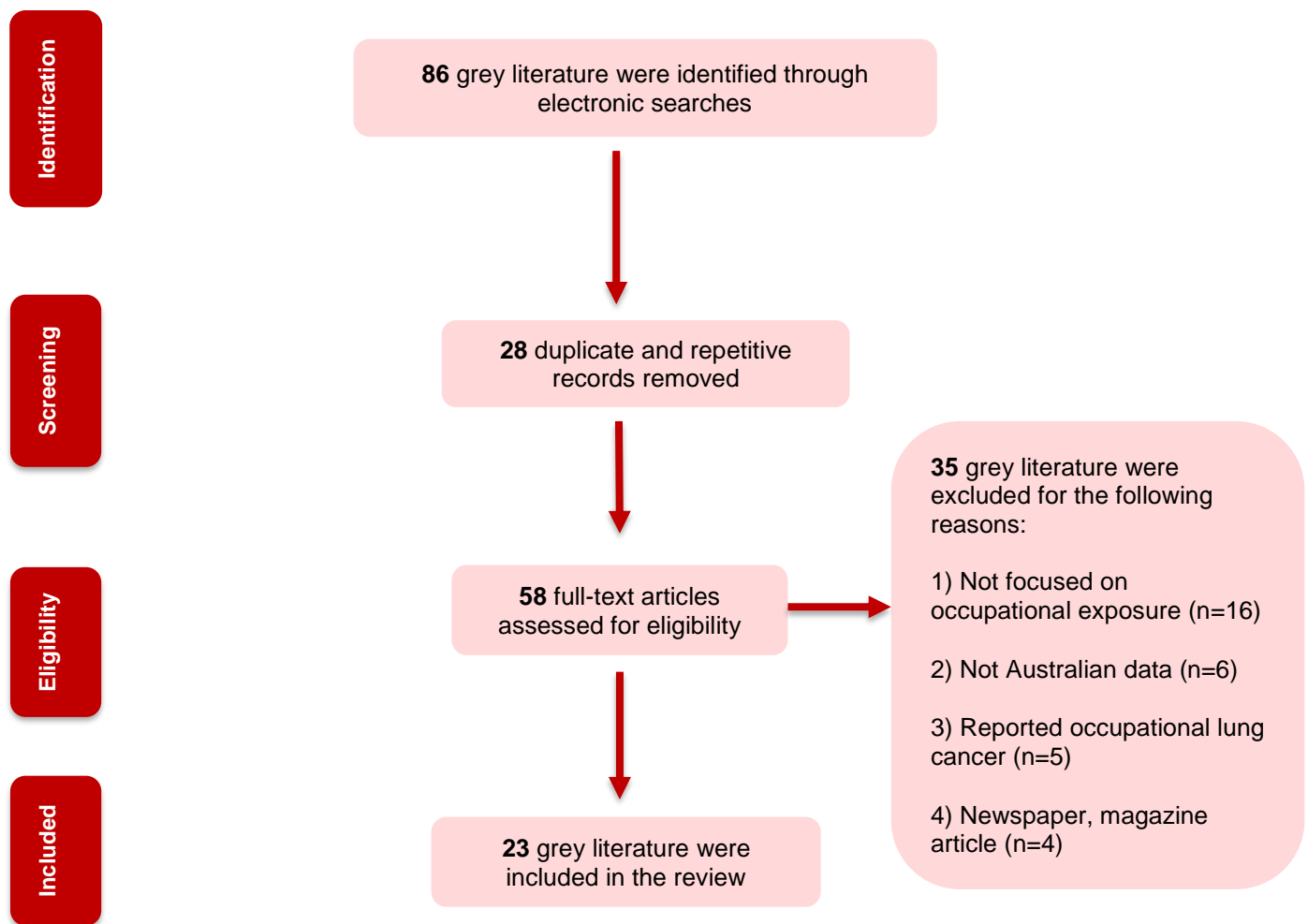
- the Pharmaceutical Benefits Scheme
- Medicare Benefits Schedule data on the treatment of chronic respiratory conditions [77]
- the National Health Survey report conducted by the Australian Bureau of Statistics (ABS) [78]
- NPS MedicineWise MedicineInsight data [79]
- The Bettering the Evaluation and Care of Health Survey [80], and
- AIHW recommended data sources to assess the impact of chronic respiratory conditions and workplace productivity [81].

The following websites were also screened to identify any publicly available relevant government agency or independent medical or scientific advisory committee reports, or annual reports (hereafter referred to as reports).

- SWA
- AIHW
- ABS
- icare
- Coal Services New South Wales (NSW)
- Australian open government data (data.gov.au)
- Business Queensland
- Comcare
- WorkSafe Victoria
- SafeWork NSW
- WorkSafe ACT
- WorkCover Queensland
- WorkCover WA
- WorkSafe Tasmania
- Return to Work SA
- NT WorkSafe
- Australian and New Zealand Society of Occupational Medicine (ANZSOM)
- Thoracic Society of Australia and New Zealand (TSANZ)

- Lung Foundation Australia
- Australasian Faculty of Occupational and Environmental Medicine (AFOEM), and
- Australian and New Zealand Cardiothoracic Organ Transplant Registry (ANZCOTR).

Reports from the grey literature were identified through the website search and were also screened according to the inclusion and exclusion criteria. The initial search in the database and relevant websites identified 86 grey literature sources. Of them, 28 duplicates and repetitive records were removed. The full text of the remaining 58 records were reviewed against the predetermined inclusion and exclusion criteria (Table 1). Finally, 23 grey literature sources were included in this report (Figure 2).



**Figure 2** Grey literature selection process

**Table 1 Inclusion and exclusion criteria for peer-reviewed and grey literature**

| Inclusion criteria   |
|--|
| <ul style="list-style-type: none"><li>○ published, peer-reviewed articles</li><li>○ published grey literature</li><li>○ reports that were underpinned by a systematic review of relevant studies</li><li>○ literature published since 1 January 2000 to 15 October 2019</li><li>○ studies based on human adults (i.e. 18 years of age or older), and</li><li>○ English language.</li></ul>                                     |
| Exclusion criteria   |
| <ul style="list-style-type: none"><li>○ published before 1 January 2000</li><li>○ did not report Australian data</li><li>○ did not report Australian data separately</li><li>○ reported only exposures but not any OLD</li><li>○ not work-related exposures</li><li>○ reported only malignancy or infectious lung diseases, and</li><li>○ papers superseded by more recent findings that incorporated the same data.</li></ul> |

## 2.3 Review of Australian data sources

We requested de-identified and aggregated information and any detailed descriptions of any health screening programs for OLDs or any as yet unpublished research findings from data custodians, research coordinators, the Australian Government, states and territories jurisdictions and professional societies across Australia. Those contacted are listed in Table 2. We received responses from all the organisations and data custodians contacted. However, most contacts have published available data in their annual report and do not hold any other unpublished data. SWA supplied additional data which overlapped with the data from the NDS.

Ethics committee approval was not necessary for this report, as all of the literature used was publicly available. The workers' compensation data that were obtained had been de-identified and grouped (no individual level data were obtained) and therefore was examined under the terms of the confidentiality agreements with the original data providers.

**Table 2 List of data resources contacted, information received and reported included**

| Information received from contact with agency or research group                                  |  | Grey literature                    |
|--|--|------------------------------------|
| <b>Surveys</b>   |  |                                    |
| ABS National Health Survey data  | Data not available   | -                                  |
| Delphi Survey  | Data not available   | See reference [82]                 |
| Migrant workers in Australia   | Data not available   | -                                  |
| Australian Work Exposure Study   | Data not available   | -                                  |
| The Burden of Disease Study-<br>Australian data  | See section 3.5.1.2  | -                                  |
| Adelaide Respiratory Health<br>Project   | Data not available   | -                                  |
| AIHW   | Death data available (see Figure 10)   | -                                  |
| <b>Government source</b>   |  |                                    |
| Victorian State Government,<br>Victoria's open data directory                                    | Data not available   | Not relevant                       |
| Coal mine lung disease data<br>Queensland  | Data not available   | See Figure 7 and<br>reference [83] |
| Queensland Health  | Data not available   | See reference [43]                 |
| Office of Industrial Relations,<br>Queensland  | Data not available   | See reference [83]                 |
| icare  | Data not available   | See reference [84]                 |
| Commonwealth Government  | Data not available   | -                                  |
| SWA  | NDS Workers' compensation claims for<br>specific lung diseases 2007–08 & 2016–17   | Available and included             |
| Mining and Quarrying<br>Occupational Health and Safety<br>Committee (MAQOHSC) and<br>SafeWork SA | Health screening program for engineered<br>stone benchtop industry workers supported<br>by MAQOHSC (See Table 5)                       | -                                  |
| WorkSafe ACT, Access Canberra  | Territory based workers' compensation<br>claims were available, but health screening<br>data was unavailable                           | -                                  |
| NT WorkSafe  | Territory based workers' compensation<br>claims were available   | -                                  |
| WorkSafe Tasmania  | Data not available   | -                                  |
| WorkSafe Victoria  | Health screening program and silicosis<br>exposure registry (See Table 5)  | -                                  |
| WorkCover NSW  | Asbestos and silica dust health screening  | -                                  |
| WorkCover QLD  | Health screening program and compensation<br>data (See Table 5)  | -                                  |
| WorkSafe WA  | Health screening program and compensation<br>data  | -                                  |
| <b>Professional organisations</b>  |  |                                    |
| ANZCOTR  | Research reporting number of lung<br>transplants as a result of work-related<br>disease according to ANZCOTR database<br>(See Table 6) | Available, not relevant            |
| ANZSOM   | Data not available   | Available, not relevant            |
| Coal Services NSW  | Data not available   | See reference [85]                 |
| TSANZ  | Data not available   | Available, not relevant            |
| Lung Foundation Australia  | Data not available   | See reference [82]                 |
| AFOEM  | Data not available   | Available, not relevant            |

# **Chapter 3 Main findings**

This chapter presents information about the types of data sources found in the searches and information about OLDs in Australia found in those data sources. Data are drawn from published peer-reviewed studies, grey literature, health screening data sources, workers' compensation statistics and other types of OLD findings identified in the searches. There is a specific focus on prevalence, incidence and trend data over time, where available.

## **3.1 Introduction**

The search identified 56 peer-reviewed publications and 23 grey literature sources. The designs of the studies in the 56 peer-reviewed publications were:

- four workplace-based cohort studies
- 25 workplace-based cross-sectional studies
- three population-based cohort studies
- seven population-based cross-sectional studies
- 10 clinical case series, and
- seven reviews.

The main findings from these peer-reviewed publications and grey literature sources are summarised in Table 7 to Table 15 in the Appendix. The summary information in these tables includes:

- the exposures assessed
- the methods used in the exposure assessment
- the OLD outcomes investigated, and
- the key findings.

The cohort and cross sectional studies were grouped by the disease outcome and are presented in Table 7 to Table 10, the studies based on workers' compensation data are presented in Table 11, the clinical case reports in Table 12 and reviews from the peer reviewed literature are in Table 13. Grey literature data are presented in Table 14 and national workers' compensation claims in Table 15.

## **3.2 Types of data sources identified**

### **3.2.1 Epidemiological studies**

Epidemiological studies were conducted either in workplace-based or in population-based settings. Studies in workplace settings usually focused on specific hazards and the degree of risk of developing an OLD associated with exposure levels [86]. They also usually presented some exposure information for the period of the study or across the working life of the participants. The main focus of these workplace-based studies was usually to provide scientific evidence that some work-related exposures are associated with OLDs, rather than investigating incidence or trends over time for OLDs in that workforce [3, 21].

Population-based studies were either cross-sectional or included a longitudinal follow-up of members of the general population to investigate relationships between work-related exposure and OLDs [87]. General population studies include individuals who have experienced a wide variety of occupations

over their lifetime so that an association between a specific exposure and disease may be difficult to identify [88]. Exposure information is less likely to be available, and where it is available, is usually of lower quality than for workplace-based studies [88].

### **3.2.2 Other data sources**

Seven papers were identified that used other data sources, such as disease notification data, workers' compensation data, and the Coal Services NSW database (Table 11). This included information on voluntary and anonymous notified cases of OLDs by respiratory and occupational physicians in Victoria, Tasmania and NSW obtained from the Surveillance of Australian workplace Based Respiratory Events (SABRE) scheme<sup>4</sup> [89-91].

### **3.2.3 National Data Set for Compensation-based Statistics**

The NDS is compiled by SWA. It includes accepted workers' compensation claims from Commonwealth, state and territory agencies. It includes accepted compensation cases that result in death, permanent disability or temporary disability involving five or more days off work. NDS data are used as WHS indicators at a national level [92-94].

SWA extracted and provided national OLD-specific claims data from 2007–08 to 2016–17. Because of the small numbers in each year, yearly breakdowns were not provided to preserve confidentiality. Instead, a summary of the findings over time periods of more than one year are presented.

## **3.3 Work-related asthma**

### **3.3.1 Prevalence of work-related asthma**

#### **3.3.1.1 Epidemiological studies**

This review identified 13 epidemiological studies with Australian data relevant to occupation and prevalence of asthma. Of these, 10 studies were workplace-based, and three studies were population-based (Table 7). Brief descriptions of the most recent papers with the most relevant findings regarding rates are:

- A large cross-sectional survey conducted in 2016–17 in a large tertiary public healthcare facility reported 13.7 per cent (51/371) asthma among nursing staff, 12.7 per cent (26/205) in allied health workers and 11.9 per cent (35/294) in doctors [95].
- Another large population-based study conducted among 5,331 adults in NSW that reported 18 per cent (910/5331) prevalence cases of WRA [96].

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<sup>4</sup> The SABRE scheme was a voluntary surveillance scheme for occupational respiratory diseases in Victoria and Tasmania and later in NSW based at Monash University between 1997 and 2008. SABRE ceased because of lack of funding, despite a Senate report in 2006 recommending that SABRE be expanded nationally to provide Australia-wide data on OLDs (91).

- A cross-sectional study among 454 male West Australian (WA) aluminium industry workers reported 2.1 to 5.9 per cent prevalence of asthma symptoms [97].
- A study of 570 South Australian (SA) firefighters found four per cent (24/570) of metropolitan firefighters reported they have been previously diagnosed with asthma [98].
- A cross-sectional study among 1,424 male Australian Gulf War veterans found that 13.7 per cent (190/1424) reported having had asthma and 11.5 per cent (165/1424) reported doctor-diagnosed asthma [99].
- A small hospital-based cross-sectional survey among hospital staff in NSW of 66 participants found 17 per cent (11/66) with severe uncontrolled asthma had occupations with high-risk exposures to latex, cleaning products and dust, while 14 per cent (9/66) with severe uncontrolled asthma had low-risk exposures such as exhaust fumes [100].
- Exposure to a wide range of other asthmagens has been identified in other included studies, including exposure to bauxite, organic and inorganic dust, flour, wood dust, formaldehyde and cleaning agents [95-98, 101].

### **3.3.2 Incidence of work-related asthma**

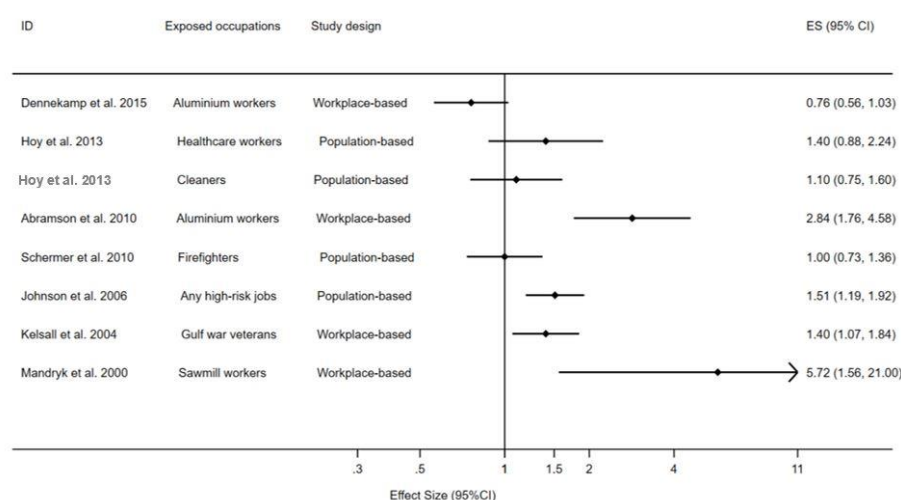
#### **3.3.2.1 Epidemiological data**

Only two studies have directly estimated the incidence of WRA (Table 7). One study used longitudinal population-based cohort data from a Tasmanian study to investigate asthma incidence among 792 people aged 13 to 44 years and reported 37 per cent had cumulative incidence of asthma. Of those subjects, 12 per cent were exposed to latex, 10 per cent to bioaerosols, and 15 per cent to industrial cleaning agents [102]. The authors concluded that the incidence of asthma increased significantly with cumulative latex exposure, especially over six years of exposure [102]. Among aluminium smelter workers, the incidence of asthma symptoms ranged from 10/100 to 28/100 person-years<sup>5</sup> in men, and 9.8/100 to 22/100 person-years in women [103]. A survey using data from the Australian Aluminium Council and aluminium workers' health monitoring data found that the incidence of WRA among aluminium smelters has declined from 9.46/1000/year in 1992 to 0.36/1000/year in 2006, an overall 96 per cent reduction [104].

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<sup>5</sup> Person-years are measurements that consider both the number of people and the amount of time each person spends in an epidemiological study. For example, a study that followed 1000 people for one year would be 1000 person-years of data.

A forest plot of results from some of the studies which investigated WRA yielded an increased risk resulting from work-related exposure (Figure 3).



**Figure 3** Forest plot of studies relevant to estimating the work-related contribution to asthma in Australia

### 3.3.2.2 Other data sources

The Global Burden of Disease Study in 2003 estimated that in Australia, there were 78,493 new cases of asthma in 2003 and of them, more than 20,000 were working-age adults (15 to 64 years) [105, 106]. Applying a PAR of nine to 15 per cent to this estimate, they concluded that between 1,850 and 3,090 new cases of asthma in 2003 were associated with work-related exposures, giving an annual incidence of 195 to 325 cases per million working adults per year [105, 106] (Table 3).

**Table 3** Estimated incidence of WRA in Australia in 2003

| Population attributable risk* | Estimated number of cases (2003) | Incidence rate* (cases per million workers per year) |
|-------------------------------|----------------------------------|--|
| 5                             | 1030                             | 110  |
| 9                             | 1850                             | 195  |
| 15                            | 3090                             | 325  |

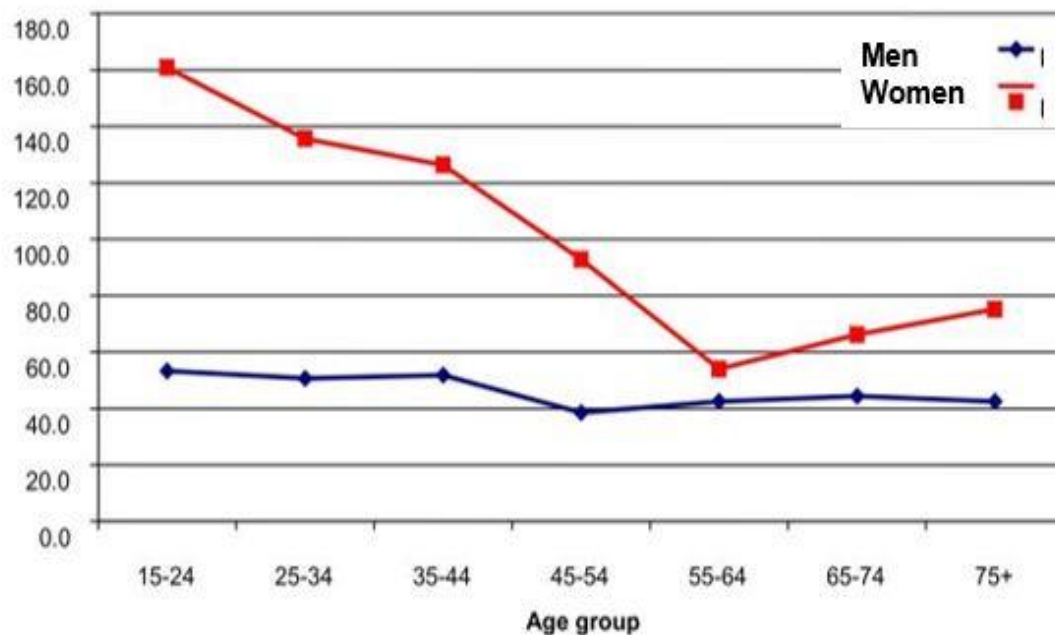
\* Estimates based on an estimated number of new asthma cases in those aged 15 to 64 years old in 2003 and in the labour force as of June 2003.

In 2005, the Victorian Department of Health and Human Services investigated WRA detection and surveillance. The report identified a PAR of 15 per cent among Victorian adults (Table 14) [6]. The analysis found 1,034 Disability Adjusted Life Years (DALYs) were associated with WRA, two-thirds of which occurred in women, and the majority of cases occurred after retirement (<65 years) (**Error! Reference source not found.**) [6].

In Victoria and Tasmania, the SABRE scheme in 2004 identified that 33 per cent of the notified OLDs were WRA as reported by respiratory and occupational physicians, which was then the most commonly diagnosed OLD notified to SABRE (Table 11) [89].

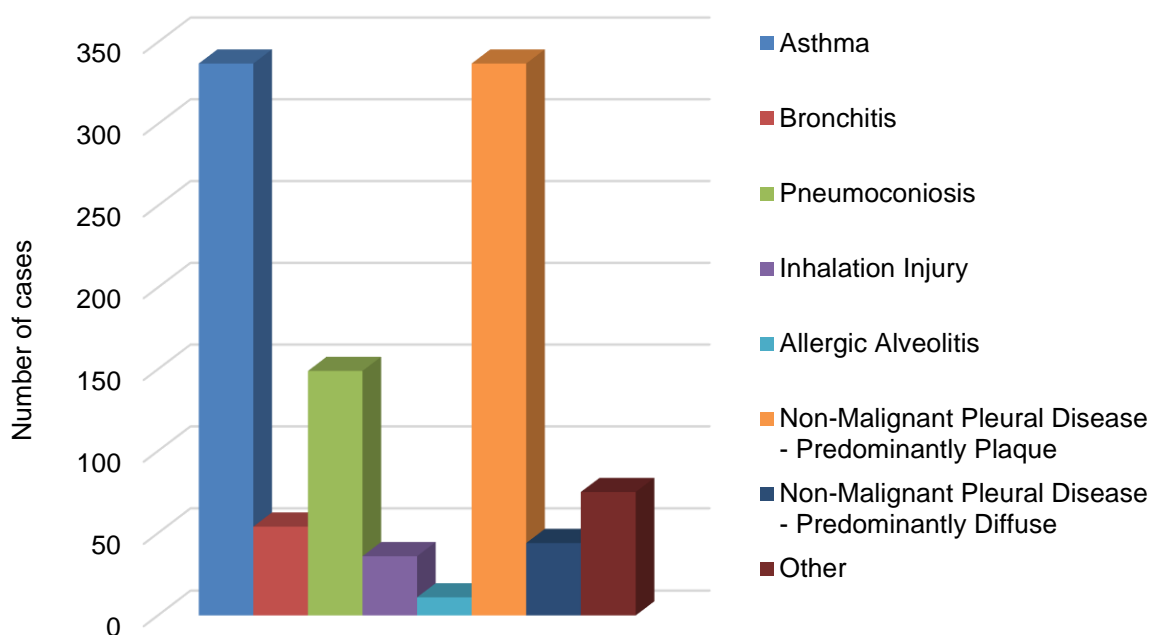
In later unpublished data from SABRE, between 1999 and 2008, there were approximately 1,043 non-

malignant, non-infectious OLDs reported to SABRE. Of these diagnoses, approximately 337 (32.3 per cent) were WRA (Figure 5). The asthma incidence was 30.9 (95% CI 26.8 - 35.5) per million workers per year with 2.4 times higher incidence in men compared to women [89].



**Figure 4** DALYs for WRA in Victoria [6]

In contrast, SABRE NSW identified approximately 3,190 diagnoses of OLDs reported between January 2001 and November 2007. Of these diagnoses, only three per cent (80 cases) were WRA [90]. SABRE NSW identified that 13.5 per cent of these cases were associated with wood dust exposure and 5.8 per cent with isocyanate exposure [89].



**Figure 5** SABRE notifications for Victoria from 1999 to 2008 (unpublished data)

### 3.3.2.3 National Data Set for Compensation-based Statistics

In the ten-year period from 2007–08 to 2016–17, there were 7,185 accepted compensation claims in the NDS, for all respiratory system diseases; 1,100 (about 15 per cent) were for asthma (Table 4). In 2004–05, 51 per cent of the accepted asthma claims were from female workers, and about 33 per cent were for workers aged 45 to 54 years [106]. There was a downward trend in the rate of workers' compensation claims for WRA from 2000–01 to 2010–11. The rate of claims reduced to 15 claims per million workers over that seven-year period (Figure 6) [107]. It is important to note that a very large proportion (69 per cent) of the claims over this period were not classified, so some asthma claims may be also be included there. Such a large proportion of unclassified respiratory claims makes it more difficult to interpret trends over time for specific OLDs.

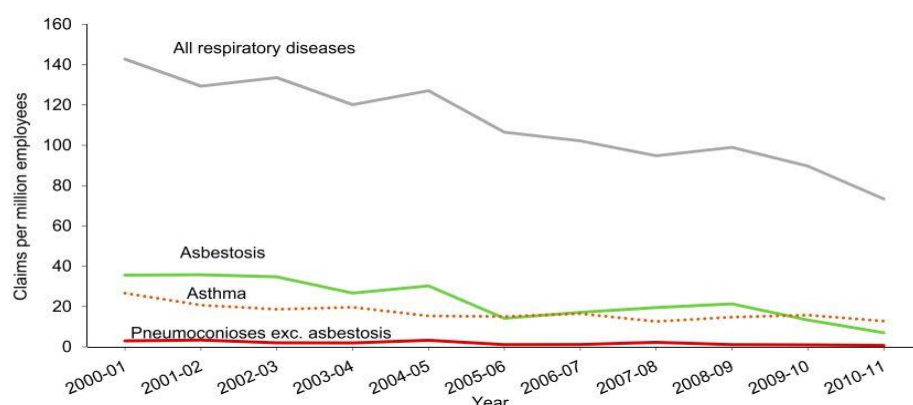
**Table 4 Number of workers' compensation claims for respiratory system diseases and claims from 2007–08 to 2016–17**

| Nature of respiratory diseases                       | Accepted claims | Total claims |
|--|-----------------|--------------|
| Asthma   | 1100 (15%)      | 1290         |
| COPD/chronic bronchitis, emphysema                   | 70 (1%)         | 115          |
| Asbestosis   | 890 (12%)       | 1150         |
| CWP  | 10 (0.1%)       | 10           |
| Silicosis  | 70 (1%)         | 105          |
| Pleural plaques and other pneumoconiosis             | 45 (0.6%)       | 50           |
| Other respiratory diseases, not elsewhere classified | 5000 (69%)      | 5945         |

Explanatory notes for the tables above

1. The claim number in the above table represent all workers compensation claims excluding journey claims.
2. The number of claims shown in this table have been rounded to the nearest 5 to maintain confidentiality.
3. All workers compensation was lodged between 1 July and 30 June.

Data source: NDS



**Figure 6** The claims of respiratory diseases per million employees from 2000 to 2011 based on workers' compensation-based statistics [107]

#### **3.3.2.4 Estimates of exposure to asthmagens**

In the absence of reliable data on the incidence of WRA, researchers have investigated the extent of causative exposures at the workplace, rather than lung disease as an outcome. Identification of the industries where workers are exposed to workplace agents such as asthmagens (defined as workplace exposures known to cause occupational asthma) provides information about where OLD prevalence and incidence studies could be conducted.

Fritschi et al estimated the prevalence of exposure to asthmagens at work in Australia, using a telephone survey, as part of the Australian Work Exposure Study. The overall results were published in 2016 [8]. The survey identified that exposure to at least one asthmagen was more common among men (47 per cent) than women (40 per cent) and when the findings were extrapolated to the Australian population using labour force survey data, and it was estimated that, approximately 2.8 million men and 1.7 million women were estimated to be exposed to at least one occupational asthmagen. Among men, the most common exposures were to bioaerosols (29 per cent), and metals (27 per cent), whilst the most common exposures among women were latex (25 per cent) and industrial cleaning and sterilising agents (20 per cent).

The findings were explored in later Australian Work Exposure Study papers focused on specific exposures (for example isocyanates) where three per cent of the Australian working population are thought to be exposed mainly in construction, woodworking, painting and printing [108]. Other papers have focused on specific workgroups. For example, latex glove uses in healthcare workers with almost two-thirds (63 per cent) reported wearing latex gloves, and 26 per cent using powdered latex gloves [109].

Information on asthmagens was also reported to SABRE Victoria and Tasmania. The more commonly reported asthmagens were:

- paint fumes
- aluminium potroom gas/fumes
- solvents
- latex
- flour
- cleaning agents, and
- products of thermal combustion such as tobacco [89].

### **3.4 Chronic obstructive pulmonary disease**

#### **3.4.1 Prevalence of chronic obstructive pulmonary disease, chronic bronchitis and emphysema**

##### **3.4.1.1 Epidemiological data**

Five epidemiological studies published since 2000 have investigated the association between work-related exposure and COPD (Table 8) [34, 37, 38, 110, 111]. Some of these studies also reported

chronic bronchitis and emphysema as separate outcomes. Estimates of the prevalence of work-related COPD in the Australian population range from eight to 19 per cent [34, 37, 38].

Two recent general population-based cross-sectional studies with 1,335 participants were published from the Tasmanian Longitudinal Health Study (TAHS) group [37, 38]. These studies identified that 12 per cent (28/226) of COPD cases were attributed to work-related exposure to pesticides [37], chlorinated solvents [38] and dust, gases and fumes [37] when assessed using a JEM. A large proportion, 29 per cent (63/223), of self-reported and doctor-diagnosed chronic bronchitis cases were attributed to exposure to pesticides by the TAHS study [37]. The authors identified a number of jobs (these included: gardeners, farm labourers, nursery growers, forestry labourers and animal producers) for which the prevalence of COPD resulting from pesticide exposure was significantly increased compared to all other jobs [37].

Another study from Melbourne, Victoria with a sample size of 1,213 found that biological dust exposure increased the risk of COPD and chronic bronchitis in women [34]. They also estimated that 19 per cent (226/1213) of COPD and four per cent (49/1213) of chronic bronchitis cases were attributed to work-related exposures [34].

We did not identify any Australian longitudinal studies with repeated follow-up of the same participants or occupational group to directly estimate the incidence of COPD related to work-related exposures.

Three longitudinal studies investigated lung function decline, which may indicate the possible future development of COPD or pneumoconiosis (Table 10) [39, 40, 112]. Two of these three longitudinal studies observed excess declines in lung function in association with work-related exposures to aromatic solvents and dust, smoke and other fire-related exposures [39, 40].

#### **3.4.1.2 Other data sources**

A recent case series review from the Coal Mine Workers' Health Scheme in the Queensland coal industry identified 22 confirmed cases of COPD in 2015 (Table 14) [43]. The report from the Queensland Department of Natural Resources, Mines and Energy (DNRME) identified 21 COPD cases in the 2019–2020 financial year and nine cases in 2018 financial year (Figure 7) [83]. Given there are other recognised causes of COPD, including cigarette smoking, it was not clear how much of the COPD in Queensland coal mine workers was related specifically to coal dust exposure.

#### **3.4.1.3 National Data Set for Compensation-based Statistics**

During the 10 years from 2007–08 to 2016–17, there were 70 (<1 per cent) accepted claims for COPD/chronic bronchitis in the NDS (Table 4). The number of cases per year was too small to investigate whether there was a trend of claims for COPD/chronic bronchitis over time.

## 3.5 Pneumoconioses

### 3.5.1 Coal workers' pneumoconiosis

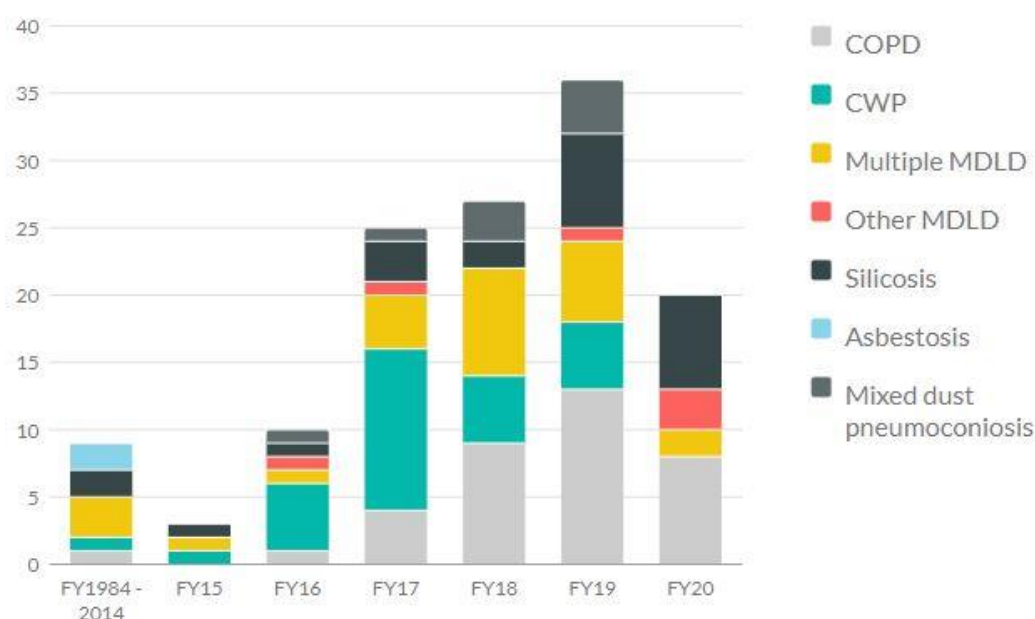
#### 3.5.1.1 Prevalence and incidence of coal workers' pneumoconiosis

There are no nationally representative datasets available that allow for the estimation of the true prevalence and incidence of CWP in Australia [43]. An extensive literature search for this report revealed a lack of evidence from epidemiological studies on CWP. However, there are several other sources of information available from state governments, in particular from Queensland and NSW.

Increasing attention has been given to CWP in Australia since a couple of new cases were identified among Queensland coal mine workers in late 2015 after no cases had been identified since the 1980s. This prompted a review of the respiratory component of the coal mine workers' health assessment scheme, undertaken by Monash University and the University of Illinois, Chicago [113]. This review highlighted several deficiencies in the scheme and since then, the DNRME has put in place many measures to improve the design, operation and quality control of the scheme.

A recent case series from the Queensland coal industry identified 79 cases of coal mine dust lung disease (CMDLD) including 27 confirmed cases of CWP, 18 cases of mixed dust pneumoconiosis and five cases of dust-related diffuse fibrosis in 2015 (Table 14) [43]. The latest report from DNRME identified that 40 CWP cases (including those identified in the case series above) have been diagnosed since 1984 (Figure 7). This report also identified seven cases of silicosis, six cases of other mine dust lung disease (MDLD), and five CWP cases in the 2019 financial year [83]. Despite this recent spike in CWP in Queensland, the long-term data from Coal Services 2010 annual report states that there have been no new cases of CWP in NSW since the 1980s [85].

The number of cases referred to above probably underestimates the burden of CMDLD, as it has not been mandatory to report CWP or other pneumoconioses in Australia until 1 July 2019 when mandatory reporting for OLD came into effect in Queensland. No data on notifications under this scheme are available.



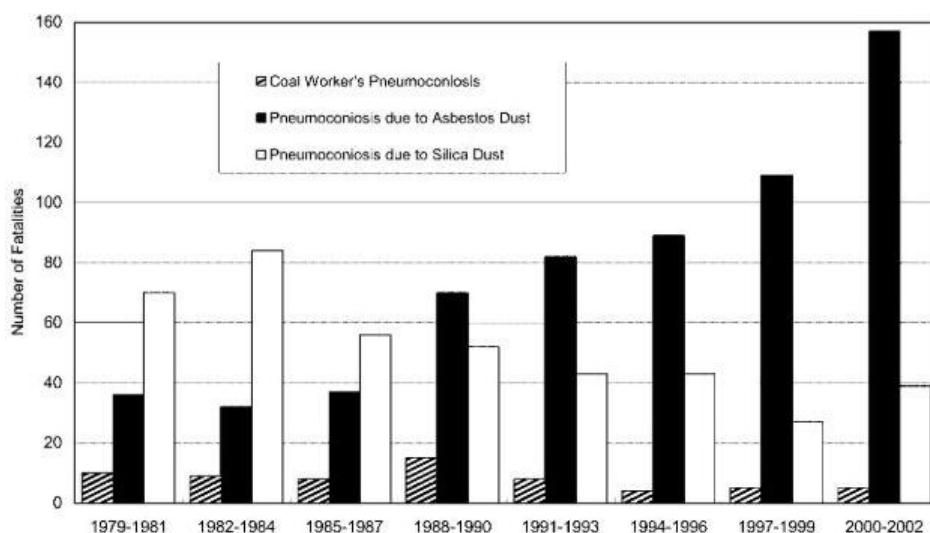
**Figure 7** Cases of MDLD, including COPD, CWP, and pneumoconiosis, reported to DNRME for all mining workers from 1984 to 2020 [updated on 31 January 2020]

\*FY=Financial Year, (FY20 data are incomplete)

**Multiple MDLD:** Individual has more than one MDLD. This figure may include cases of CWP, silicosis and mixed dust pneumoconiosis. **Other MDLD:** Another type of MDLD or the specific type of lung disease is to be confirmed (cases in this category may be re-classified over time as additional information is provided to the DNRME).

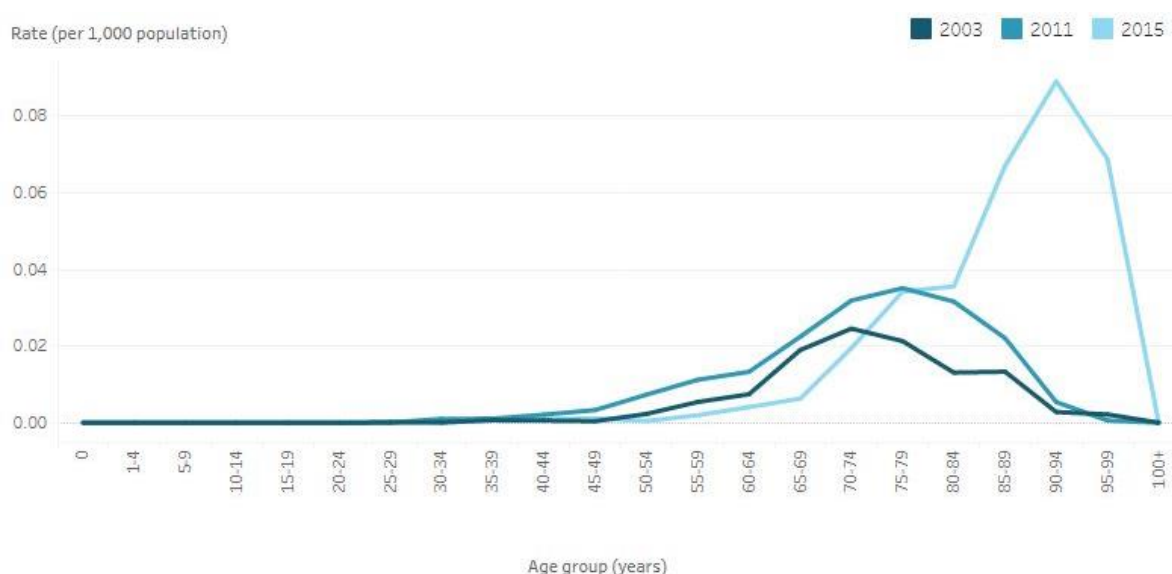
### 3.5.1.2 Epidemiological and other data sources

The only identified retrospective analysis of pneumoconiosis mortality data from Australia was conducted in 2006 [114]. This study found that nationally, more than 1,000 deaths had been attributed to pneumoconioses between 1979 and 2002, with six per cent classified as CWP. The number of asbestosis deaths increased steadily over time from 1982 to 2002. Deaths from silicosis fell over this period and there was little change in deaths from CWP [114] (Figure 8, Table 9).



**Figure 8** Pneumoconiosis-related fatalities in Australia between 1979 and 2002 [114]

The Australian Burden of Disease Study 2015 estimated that years lived with disability (YLD) for pneumoconiosis increased by 18.9 per cent from 2003 to 2015. However, DALY and years of life lost decreased by 12.4 per cent and 14 per cent respectively over this period [76] (Figure 9). There was no breakdown for the different types of pneumoconioses, such as CWP.



**Figure 9** Comparison of age-specific YLD rates from 2003 to 2015 for pneumoconiosis

Figure source: AIHW Australian Burden of Disease Study 2015: Interactive data on disease burden.

[www.aihw.gov.au/reports/burden-of-disease/abds-2015-interactive-data-disease-burden/contents/comparisons-over-time](http://www.aihw.gov.au/reports/burden-of-disease/abds-2015-interactive-data-disease-burden/contents/comparisons-over-time)

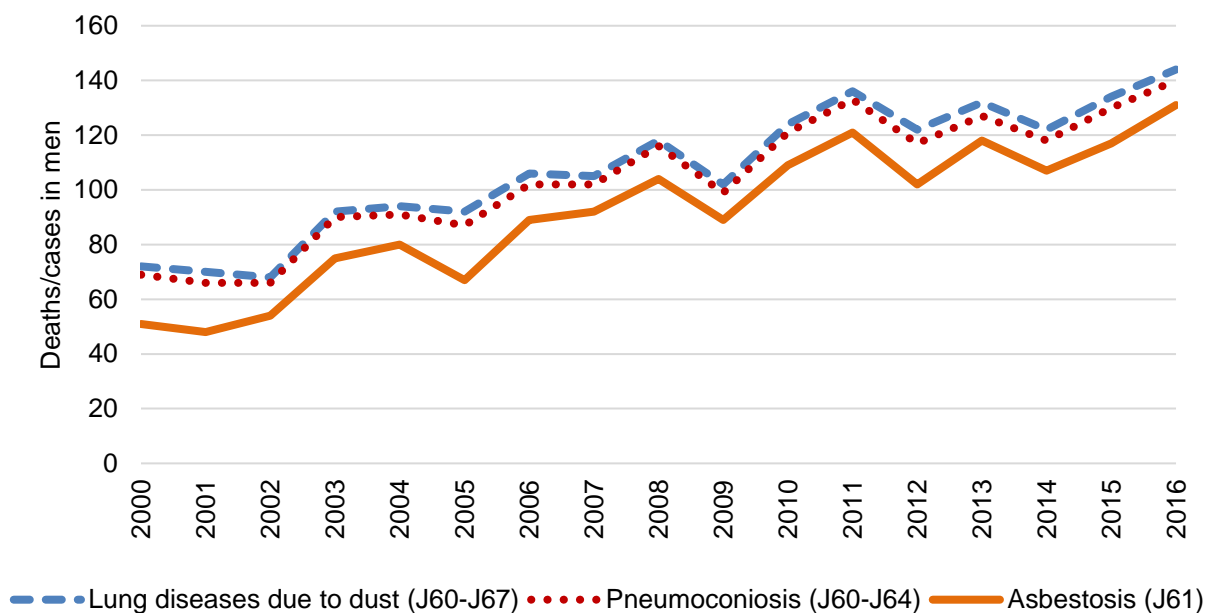
The AIHW maintains the National Death Index (NDI). Deaths are coded to the International Classification of Diseases tenth revision (ICD-10) [115] and entered into the NDI. Registered deaths from pneumoconiosis are coded J60-J64.

In men, there were 99 pneumoconiosis deaths in 2009 and this increased to 140 in 2016. Registered deaths from pneumoconiosis are coded J60-J64. This includes:

- CWP (J60)

- pneumoconiosis from asbestos and other mineral fibres (J61)
- pneumoconiosis from dust containing silica (J62)
- pneumoconiosis from other inorganic dusts (J63), and
- unspecified pneumoconiosis (J64).

Death from lung diseases caused by any dust (ICD codes 60-67) increased from 102 deaths in 2009 to 144 deaths in 2016 (Figure 10). These deaths include J65 pneumoconiosis associated with tuberculosis, J66 airway disease from exposure to specific organic dust and J67 hypersensitivity pneumonitis from organic dust. Deaths from asbestosis are included in each group in the graph and are clearly the major contributor.



**Figure 10** Pneumoconiosis-related mortality from the NDI for men from 2000–16

\*Graph reproduced using national mortality data from AIHW

\*The underlying causes of death are determined by grouping specific causes of death and counting the number of deaths assigned to each cause. These were coded according to ICD-10 and are available in the AIHW General Record of Incidence of Mortality data [116].

The trends of death rates from pneumoconiosis have historically been higher for men than for women. There were four deaths in women from pneumoconiosis (J60-J64) in 2016, whereas there were 10 deaths in 2015 and six deaths in 2014.

### 3.5.1.3 National Data Set for Compensation-based Statistics

Approximately 1,000 workers' compensation claims were lodged by coal mine workers in Queensland in 2017, but less than one per cent were for respiratory conditions which include cold, influenza, asthma and bacterial infections. The Queensland Office of Industrial Relations (OIR) advised that there were 41 claims lodged for CMDLD by coal mine workers in 2017 [117], but the number of claims specifically for CWP was not identified.

During the 2007–08 to 2016–17 period, 10 claims for CWP were accepted in the NDS (Table 4). There were fewer than five cases per million employees for CWP from 2000 to 2008, whereas no claims were lodged between 2008 and 2011. In 2017, two new cases were identified among individuals, who had

worked in NSW and Queensland coal mines [117, 118]. In addition, given the long latency, many cases may be diagnosed after retirement and so will not appear in workers' compensation data.

## 3.5.2 Silicosis

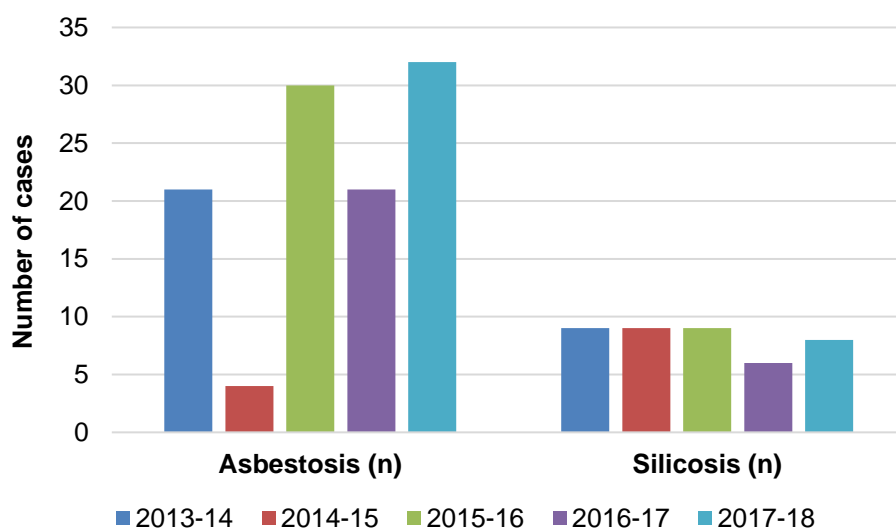
### 3.5.2.1 Prevalence and incidence of silicosis

This section includes data sources on chronic silicosis arising from sources other than from work with engineered stone, which is discussed separately in section 3.5.2.3.

Available epidemiological data on silicosis in Australia are set out in Table 11 [119, 120]. A lung cancer mortality study of 1,467 men compensated for silicosis between 1995 to 2001 used data from icare in NSW. Of the 1,467 compensated silicosis cases, there were 515 mortalities from non-malignant respiratory disease, however smoking rates were higher than expected compared to national rates [119].

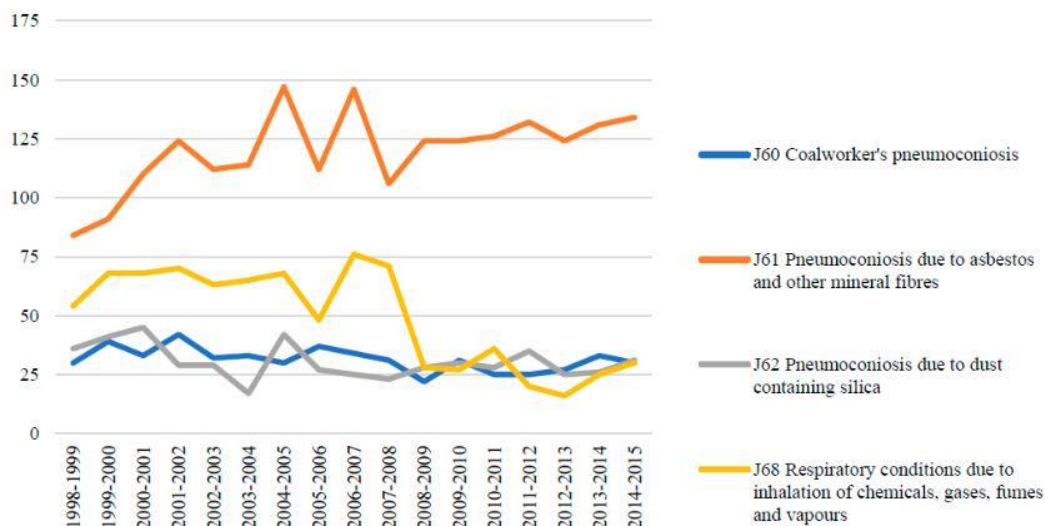
Another study investigated the incidence of compensated silicosis cases among WA gold miners. This study found that there were no compensated claims for silicosis in WA for gold miners after 1974 [120].

The SABRE scheme identified 90 chronic silicosis cases in NSW between 2001 and 2008 [90]. icare also carry out medical screening of workers in NSW exposed to dust. They identified eight silicosis cases during the 2017–18 period and six cases in the previous year (Table 14) (Figure 11) [84]. There was no trend visible in the number of cases per year between 2013 (nine cases) and 2018 (eight cases). The latest data from the DNRME listed 23 silicosis cases since 1984 (current as at 31 January 2020); of which, 14 cases have been reported since July 2019 (Figure 7).



**Figure 11** Number of asbestosis and silicosis cases (not specified the type of silicosis) certified by medical assessment panel, from 2013–14 to 2017–18 in NSW [84]

A 24-year retrospective analysis of national mortality data was performed for the period of 1979 to 2002 [114]. This study identified 1,000 pneumoconiosis cases during this period and found that 38 per cent were from silicosis [114]. According to Australian hospitalisation data between 1998 and 2015, there were 517 hospitalisations for silicosis (Figure 12) [57].



**Figure 12** Number of OLDs hospital admissions 1998 to 2015 [57]

\* The Queensland Health review of Queensland hospital patients coded to ICD-10 code J60, identified that the majority of the patients were allocated to the code because carbon pigment was identified in lymph nodes biopsied as part of a cancer diagnosis process rather than because of a CWP diagnosis [113].

### 3.5.2.2 National Data Set for Compensation-based Statistics

During the 2007–08 to 2016–17 period, 105 claims were lodged nationally for silicosis, of which 70 claims had been accepted, equivalent to one per cent of all accepted claims for respiratory conditions in the NDS (Table 4).

### 3.5.2.3 Silicosis in engineered stone workers

Since 2017 an increasing number of cases of an accelerated and rapidly progressive form of silicosis and associated PMF have been identified among engineered stone workers in Australia [47]. In 2018, a case-series identified seven cases of engineered stone associated silicosis from Queensland, Victoria and NSW, which were identified by respiratory physicians [47].

In response to the emergence of this accelerated form of silicosis among engineered stone workers, three states; Queensland, Victoria and SA, have implemented a respiratory health screening program for workers who have worked or are working with engineered stone.

In Queensland from mid-September 2018, the OIR and WorkCover Queensland conducted health screening of 1,017 workers exposed to RCS through their work with engineered stone products. Of the 1,017 completed screenings, 302 had abnormalities reported in their screening and were referred to respiratory physicians for further testing. One hundred and ninety nine workers have been diagnosed with a work-related respiratory condition [121]. Of these 199 workers, 26 were diagnosed with PMF, 163 have work-related silicosis and 10 have another respiratory condition. A total of 186 claims resulting from this screening program were received by WorkCover Queensland (Table 5). More than 100 workers' compensation claims were lodged in Queensland between August and November 2018 for silicosis [122].

The Victorian Government launched a free respiratory health screening program in 2019 for an estimated 1,500 current and former Victorian workers exposed to RCS through working with engineered stone products. WorkSafe Victoria is funding Monash University to undertake a research program in parallel with the screening program, which includes a screening registry and silica associated disease registry, which have been running since mid-2019. As of 5 September 2019, 115 workers have completed screening, and 78 of them were referred to respiratory physicians for further testing. WorkSafe Victoria has accepted a total of 22 silicosis-associated disease claims as of 5 September 2019 (Table 5).

Free comprehensive respiratory health screening for workers exposed to RCS in engineered stone has been offered to SA workers by MAQOHSC [123, 124]. As of 5 September 2019, 326 workers have completed screening, and 66 of them were referred to respiratory physicians for further testing. The silicosis-associated claims data in SA are not yet available (Table 5).

**Table 5 Health screening for engineered stone associated silicosis diagnosis and referrals**

| Jurisdiction        | Number of workers screened | Number of silicosis diagnoses | Referred to respiratory physician | Compensation claims | Statistics current as at: |
|---------------------|----------------------------|-------------------------------|-----------------------------------|---------------------|---------------------------|
| <b>Queensland *</b> | 1017                       | 163                           | 302                               | 186                 | 31/1/2020                 |
| <b>Victoria **</b>  | 115                        | Data not available            | 78                                | 22                  | 5/9/2019                  |
| <b>SA †</b>         | 326                        | Data not available            | 66                                | Data not available  | 5/9/2019                  |

\* Latest data from: [www.worksafe.qld.gov.au/silicosis/background-to-silicosis](http://www.worksafe.qld.gov.au/silicosis/background-to-silicosis)

\*\* Screening data from financial year 2019–20

† Data received from SWA

icare and SafeWork NSW conduct regular lung health screening of dust exposed workers to identify OLD cases [125]. Seven-hundred and eighty-nine workers from 58 workplaces have been screened so far in the current financial year (2019–20). Of these, 76 workers were identified with abnormalities and referred to respiratory physicians for further testing. In the financial year 2018–19, a total of 65 workers screened were found with abnormalities. A total of 56 claims, including any dust diseases related claims, have been accepted as of 9 October 2019. It is unclear how many of these claims relate to work with engineered stone.

WorkSafe WA is currently undertaking respiratory health monitoring of workers exposed to dust [126]. As of 11 September 2019, 144 workers from 36 workplaces in WA have completed screening, and two silicosis-associated claims have been accepted. It is not clear if these two cases relate to workers working with engineered stone.

Some engineered stone workers have required a lung transplant [47, 122]. According to the voluntary ANZCOTR database, three lung transplants were performed in 2018 because of engineered stone associated silicosis and another two cases were on the waiting list (Table 6). Four cases were reported in a case series analysis conducted in a NSW respiratory clinic and one of them was waiting for a lung transplant at the time of this review (Table 14) [127].

**Table 6 Number of lung transplants resulting from work-related disease according to the ANZCOTR database**

| Diagnosis     | Number of lung transplantations | Start Date | End Date |
|---------------|---------------------------------|------------|----------|
| Silicosis     | 3                               | 1994       | 2018     |
| ILD           | 122                             | 1997       | 2018     |
| ILD/Emphysema | 3                               | 2013       | 2018     |
| Asbestosis    | 2                               | 1991       | 2018     |
| HP            | 16                              | 2010       | 2019     |

\*Data was supplied by ANZCOTR

This recent rapid increase in cases of engineered stone associated silicosis in several states resulting from work with engineered stone prompted the Australian Government to establish a National Dust Disease Taskforce in 2019 [128]. The Taskforce's terms of reference include consideration of the establishment of a national dust disease register, including possible scope and outcomes to be achieved and research required to support understanding, prevention and treatment of preventable OLDs.

### 3.5.3 Asbestosis

#### 3.5.3.1 Prevalence and incidence of asbestosis

We identified only one study in the peer-reviewed literature on asbestosis in Australia within the time frame for this review (Table 9) [129]. No Australian population-based data on asbestosis were identified.

The study examined 6,500 Australian born and Italian migrant workers employed at the Wittenoom asbestos-contaminated site in WA between 1943 and 1966. It reported higher mortality from asbestos-related diseases and pneumoconiosis in Italian migrant workers compared to an age-matched WA population [129]. The Wittenoom cohort has been studied for many years, but the previous reports were outside the time frame of this review [130].

A total 1,218 notified cases of asbestosis out of a total 3,856 cases (32 per cent) from the Victorian SABRE scheme had also been diagnosed as having pleural plaques, indicating either occupational or non-occupational asbestos exposure (Table 11) [90]. Medical screening by icare reported 9.8 per cent (32/324) asbestosis cases during the 2017–18 period, which is more than for 2016–17 (21 cases, 6.7 per cent ) and for 2014–15 (4 cases, 1.3 per cent ) periods (Table 14 and Figure 11) [84]. A retrospective analysis of Australian national mortality data identified 1,000 deaths from pneumoconiosis between 1979 to 2002 identified that 56 per cent of deaths were from asbestosis (when mesothelioma cases were excluded) [114].

Between 1998 and 2015, there were 2,041 hospitalisations for asbestosis (ICD-10 code J61) and 487 (43 per cent ) hospitalisations for pleural plaques according to the latest AIHW report (Figure 12) [131]. This can be compared to 833 hospitalisations during the same period for other respiratory conditions including cases from inhalation of dust, chemicals, gases, fumes and vapours (ICD-10 code J68) [57].

In 2014, SWA reported increased hospitalisation in the 2004–05 period resulting from exposure to asbestos, mineral fibres, organic dust, and coal dust [107].

According to the ANZCOTR, two lung transplants were performed between 1991 and 2018 because of asbestosis. The precise number of Australian lung transplant cases resulting from asbestosis or other OLDs is unclear, as participation in the ANZCOTR database is voluntary and occupational data are incomplete (Table 6).

### **3.5.3.2 Other data sources**

icare and SafeWork NSW conduct lung health monitoring and screening of asbestos workers. Health screening of 114 workers from 17 workplaces were completed in the financial year 2019–20 (ongoing), and of them, 113 workers screened were identified with no abnormalities and one worker was referred to a respiratory physician for further testing. In the financial year 2018–19, a total of 1,722 workers and 81 workplaces participated in the screening and, of them, 1,707 were found to have no respiratory abnormalities, and 15 workers were referred to respiratory physicians for further testing.

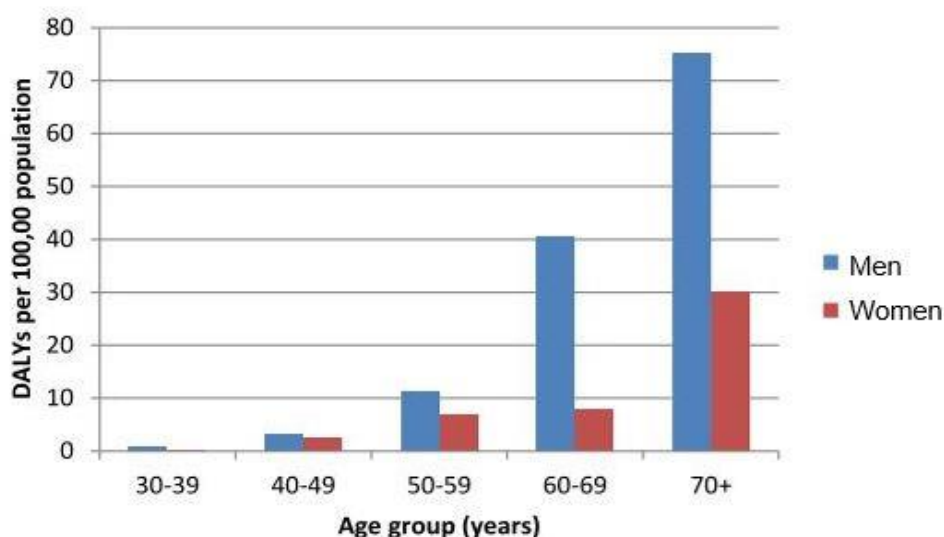
### **3.5.3.3 National Data Set for Compensation-based Statistics**

In the 10-year period from 2007–08 to 2016–17, 1,150 compensation claims were lodged for asbestosis, of which 890 claims were accepted, which is 12 per cent of all accepted OLD claims in the NDS (Table 4). Another 45 accepted claims (0.6 per cent of all accepted claims) were for pleural plaques and pneumoconiosis from asbestos exposure. Workers' compensation claims for asbestosis have decreased over time [107]. During 2000–01, there were 38 claims per million employees for asbestosis, which had declined to fewer than 10 claims per million in 2010–11 [118].

## **3.5.4 Other pneumoconioses**

### **3.5.4.1 Prevalence and incidence of other pneumoconioses**

There were 1,043 non-malignant, non-infectious OLDs reported to SABRE Victoria between 1999 and 2008. Of these, 149 (14.3 per cent) were pneumoconioses (Figure 5). A review of lung diseases in Australia conducted by a research group at the University of Sydney using hospital data identified that during the 2011–12 period, there were 197 hospitalisations for pneumoconiosis among people aged 30 years and over [82]. There were 1,040 patient-days recorded for pneumoconiosis in the same age group, with a clear predominance of men being hospitalised (Table 14). This review estimated that pneumoconiosis accounted for 1,959 DALYs with the burden being more than double in men (1,369 DALYs) compared to women (589 DALYs) (Figure 13) [82]. In 2012, there were 120 deaths (0.14 per 100,000 adults aged 30 or more years) attributed to pneumoconiosis in Australia, with men accounting for 117 (97.5 per cent ) of the deaths [82]. There was no breakdown into specific types of pneumoconiosis.



**Figure 13** Burden (DALYs) per 100,000 adults from pneumoconiosis in Australia 2010–2012, by age and sex

### 3.5.4.2 National Data Set for Compensation-based Statistics

NDS data show that during the 2004–05 period, there was increased hospitalisation for pneumoconiosis resulting from exposure to asbestos and other mineral fibres, HP from organic dust, and pneumoconiosis from coal dust or dust containing silica (Figure 6) [107]. There were 45 accepted claims for other types of pneumoconiosis excluding asbestosis, CWP and silicosis during the period between 2007–08 and 2016–17 (Table 4).

## 3.6 Other occupational lung diseases

### 3.6.1 Hypersensitivity pneumonitis (or extrinsic allergic alveolitis)

Recent international estimates attributed 10 per cent of HP cases to workplace exposures, suggesting HP is not a rare OLD [3]. Evidence on the proportion of HP is limited in Australia to a few case series reported by respiratory physicians [24, 132]. No claims were lodged for HP in workers' compensation data between 2007–08 and 2016–17 period, although respiratory physicians reported a few cases among farmers (Table 4).

### 3.6.2 Alveolar proteinosis

A recent case report from Queensland reported on a 46-year old man working as a welder in the boat building industry and who had previously worked as a boilermaker between the ages of 19 to 25 years [133]. It was reported that he was diagnosed with secondary alveolar proteinosis probably because of his lifelong exposure to aluminium dust. Another case report, published in 1965, reported alveolar proteinosis in a man who had worked as a sand-blaster, tram conductor and later as a builder's labourer [134]. No other data sources identified in this review had any workplace or population data on alveolar proteinosis.

# **Chapter 4**

## **Discussion**

This chapter discusses the current findings relating to each of the OLDs in Australia and important changes in the extent of OLDs since the 2006 Review. This chapter also discusses the current OLD data sources, peer-reviewed published literature and grey literature, including their strengths and limitations.

## **4.1 Overview of occupational lung diseases in Australia**

The findings of this review demonstrate that OLDs continue to be an important group of work-related diseases among workers in Australia. The review found that there are several sources of information on OLDs in Australia, ranging from peer-reviewed research studies in the published literature, reports and other information from government and professional bodies available online in the grey literature and information available from data holders on request. These different forms of information provide different perspectives, which can contribute to our understanding of the extent of OLDs in Australia, but they fail to provide a complete picture of the likely national burden of OLDs.

Despite this, a major conclusion that can be drawn from the information obtained during this review is that CWP and silicosis are of considerably greater importance in 2019 than they were in 2006, at the time of the last report. This is because of:

1. a resurgence of CWP and other CMDLD, such as COPD, in the Queensland coal mining industry over the past three years, and
2. the rising number of silicosis cases, including cases of an accelerated form of silicosis identified among engineered stone workers over the past two years.

These developments have occurred in diseases for which, up until recent years, the causative exposures were thought to be well controlled, leading to a decline in the number of cases of CWP and silicosis in Australia reported in the 2006 Review.

COPD is the other OLD for which there is available information suggesting it is of greater importance in 2019 compared with 2006, especially in some industry sectors, such as the Queensland coal mining industry. This is partly due to a greater recognition of work-related exposures contributing to COPD, whereas traditionally it had been thought of as a disease of smokers. With declining smoking rates in Australia, work-related exposures may play an increasingly important role in the development of COPD in exposed workers.

Of the other OLDs included in this review, non-cancer asbestos-related conditions, including asbestosis, appear to be continuing their decline in frequency, but have not disappeared altogether and continue to be reported, especially in NSW. WRA, which was the major type of OLD covered in the 2006 Review, is now overshadowed by the resurgence of CWP and silicosis.

Based on available data in recent years, especially workers' compensation data, WRA appears to be continuing to slowly decline nationally. However, of the two types of WRA, there is more information available to make conclusions about the current state and trends over time for true occupational asthma, rather than for work-exacerbated asthma. Of the remaining OLDs included in this report, HP, toxic

pneumonitis and alveolar proteinosis, there is little information available to document the current extent of these diseases in Australian workers, nor trends over time.

## **4.2 Data sources reporting occupational lung diseases in Australia**

There is no national comprehensive longitudinal systematic collection of information on the incidence or prevalence of OLDs in Australia, but there are many relevant data sources which collect one or more specific types of data relating to OLDs in Australia. These are epidemiological studies, usually relating to certain 'at risk' industry sectors, health monitoring or screening data for example engineered stone workers, OLD registries (SABRE etc.) and workers' compensation data.

A small number of epidemiological studies of Australian workers were identified, most were cross-sectional studies, so prevalence was calculated rather than incidence. Little further information was available from longitudinal studies of OLDs in Australian workers. Specific jobs, where some findings on OLDs were found included healthcare workers, miners, aluminium industry workers, military veterans, firefighters, tea packers, sawmill workers and asbestos workers.

No epidemiological studies were identified in other at-risk occupations such as:

- engineered stone workers
- construction workers
- agricultural workers
- painters
- pharmaceutical workers
- bakers, and
- food processing workers.

Epidemiological studies of OLDs, particularly population-based studies, may not provide accurate and complete information, as they often rely on self-reported disease/symptoms and are less commonly based on clinical diagnosis [72]. In these studies, there may be no strictly defined criteria for the OLD under investigation.

Estimates of the occupational contribution to lung disease can be difficult to make, because at an individual level the work-related disease may be attributed to other factors, for example smoking [3, 135]. In addition, some work-related disease may only be diagnosed after retirement and so may be missed in surveys of the working population, for example CWP [136]. Underreporting of OLDs is most likely to occur in older patients who are no longer at work, but whose health may have been affected by their past work history [68].

A potentially useful source of OLD data for this review is the findings from routine health monitoring. This is undertaken as a duty under WHS laws in each jurisdiction including for workers exposed to hazardous substances. Unfortunately, such data are not collected or reported in a systematic way to regulatory bodies and so it was not possible to access such health monitoring data for the purposes of this review.

The health screening programs recently established in some states to screen engineered stone workers for case finding purposes and to identify those workers with early lung changes and/or high exposure histories has provided some data. These findings were available for inclusion in this report. Such screening programs based on a set protocol and diagnostic criteria are likely to provide higher quality data on silicosis than through other health monitoring programs.

Another potentially important data source for OLDs is registry data. Registries are common in overseas countries, such as in the UK, where respiratory and occupational physicians systematically report OLDs to the SWORD scheme [68], however, such registries do not currently exist in Australia.

The coverage of the SABRE scheme was incomplete, as it was a voluntary scheme covering Victoria, Tasmania and later NSW. It did not include all physicians who saw cases of OLDs. Findings from the SABRE scheme in NSW showed that the spectrum of reported cases of OLDs was quite different from that in Victoria. This may simply reflect the differences in industry between the two regions, for example more mining in NSW [96]. Findings from the SABRE scheme, therefore, may not be representative of Australia resulting from the incomplete coverage across and within states [89]. Of course, this may change as a result of the work undertaken by the National Dust Disease Taskforce. Its interim advice states that 'We strongly recommend that all workers at risk of developing accelerated silicosis be identified. Early intervention is critical to minimising harm and maximising positive health outcomes. A national approach for case finding for all people who have worked with engineered stone needs to be developed. A National Dust Disease Registry should be implemented to capture jurisdictional disease notification data (where available), with the capability to identify early new cases of disease/s' [128]. The Minister for Health issued a statement on 23 January 2020 which accepted this recommendation.

National workers' compensation data have been routinely compiled and published on a regular basis by SWA. These data can provide useful indications about the extent of the more common OLDs at any one point in time and also trends over time. Compensation data do have limitations, which are more pronounced for OLDs and other occupational diseases compared with traumatic injury claims. They require the work contribution to the condition to be considered and reported by treating practitioners. This is less likely to occur for conditions such as asthma and COPD, which are common in the community. In contrast, for OLDs, such as asbestosis, CWP and silicosis, which each have a single documented causative exposure. These conditions are specific to a limited number of jobs and so the link with work is more likely to be recognised and claims data are more likely to be complete.

The NDS only reports accepted workers' compensation claims by eligible workers and does not cover all those potentially affected by OLDs. For example, the NDS does not include WA police or Commonwealth defence personnel. Self-employed workers, such as independent contractors, may be under-represented because workers' compensation schemes do not generally cover these workers.

Workers may decide not to pursue a claim for a variety of reasons, resulting in no data capture if a claim is not lodged [93]. Furthermore, because of long latent periods, some OLDs may not develop until after retirement, when the worker may not still be eligible for benefits. Therefore, for the above reasons, compensation data are likely to underestimate the true extent of OLDs, particularly for WRA and COPD, where the connection to work may be more difficult to make.

During the 2007–08 to 2016–17 period, 69 per cent of all accepted claims for respiratory diseases from the NDS were coded as either ‘other diseases of the respiratory system, unspecified’ or ‘other diseases of the respiratory system, not elsewhere classified’. This high proportion of unspecified cases may be a result of the difficulties in providing a specific diagnosis associated with respiratory diseases. With such a high proportion of unclassified OLDs, this makes it difficult to interpret trends over time for specific OLDs.

## **4.3 Incidence, prevalence and trends of occupational lung diseases**

### **4.3.1 Work-related asthma**

National rates of compensated WRA are only available from the NDS (Figure 6). These data show that rates have varied from 2000–01 to 2010–11 between approximately 10 and 35 claims per million employees and show an overall slight downward trend over time. Accepted asthma claims remain the highest proportion of accepted OLD claims, although it needs to be kept in mind that 69 per cent of respiratory claims do not specify a particular OLD diagnosis. WRA represented over one-third of the total number of cases of OLD reported to the SABRE scheme in Victoria.

This proportion (32 per cent) was similar to British Columbia (33 per cent) [137] and Ontario (40 per cent) [16]. Recent international estimates have also shown that approximately 16 per cent of adult-onset asthma cases were related to work-related exposures [3] and when PAR estimates for WRA have been applied to the Australian situation, this suggests that the number of WRA claims in Australia greatly under-estimate the true extent of this OLD.

The available Australian studies showed that the incidence of asthma was elevated in some working groups, for example Gulf War veterans [99], healthcare workers and cleaners [102] and not elevated in others, for example aluminium workers [103], and firefighters [98].

### **4.3.2 Chronic obstructive pulmonary disease**

Estimates of the prevalence of work-related COPD in the Australian population range from eight per cent to 19 per cent [34, 37, 38], the NDS data show that only 70 claims were accepted from 2000–01 to 2010–11. The number of claims per year was too small to investigate trends over time. No Australian longitudinal occupational study was identified that estimated the incidence of COPD.

Recent international estimates have shown that approximately 14 per cent of COPD cases are probably associated with work-related exposures [3], which suggests that the current Australian data are largely underestimating the true extent of this OLD. The findings of a recent 20-year longitudinal follow-up study of 3,343 participants of the population-based European Community Respiratory Health Survey (ECRHS) found that work-related exposures, assessed by a JEM, increased the risk of developing COPD over time [138]. The large UK population-based Biobank cohort study of 94,551 participants found that several occupations were associated with an increased risk of COPD [33]. These associations were confirmed among never-smokers and never-asthmatics. The occupations at risk

included sculptor, painter, engraver, art restorer, gardener, groundsman, park-keeper, food processor, plastics processor, moulder, agriculture, and fishing occupations and warehouse stock handler and, stacker [33]. These international studies suggest that COPD associated with work-related exposures in Australia is underestimated.

### **4.3.3 Coal workers' pneumoconiosis**

Until late 2015, CWP was thought to have been eradicated in Australia. However, this disease has re-emerged in Queensland but has not been identified in other states. The 2016 review of the respiratory component of the health surveillance for Queensland coal miners made 18 recommendations to improve the scheme [113]. The Queensland mining industry now has 39 CWP cases diagnosed since 1984 [83]. The NDI data review found deaths from pneumoconiosis in men increased from 99 in 2009 to 140 in 2016. There were 127 deaths from pneumoconiosis in 2013, which gradually increased until 2016 [116]. The national hospitalisations data from 1998 to 2015 also found increased hospitalisation because of pneumoconiosis among people aged 30 years and over between 2011 and 2012. There is often a long latent period between exposure and diagnosis of CWP, so these deaths and hospitalisations are likely to have been associated with higher coal mine dust exposures experienced many years before.

The increased rate of CWP in Queensland is of concern, but coal mine dust exposure limits have been reduced in Queensland and DNRME have provided more guidance to stakeholders and introduced increased reporting requirements. Reported dust exposure for longwall miners in 2019 was lower in most mines than in 2018 [139]. The long lag for development of CWP means that a reduction in dust exposure may not show as a reduction in CWP for some years.

This recent increase parallels that observed in the USA. NIOSH had reported a decline in prevalence of CWP from 6.5 per cent in the 1970s to a low of 2.1 per cent in the 1990s. However, CWP prevalence in the USA subsequently increased to 3.2 per cent in the first decade of the 21st century. The rate of PMF in certain coal mining states in the USA has also recently increased to levels observed prior to the introduction of modern dust controls [140]. A longitudinal analysis by NIOSH reported a significant decline in CWP deaths in USA miners from 1999 to 2016 [141].

Higher rates of CWP have been observed in other countries which are likely to be associated with higher exposures and less effective exposure controls than would be found in Australia. For example, coal miners in Chinese state-owned coal mines who commenced work in the 1970s had cumulative rates of CWP of between four to 17 per cent [142]. In Colombia, the prevalence of CWP was recently reported as 36 per cent [142].

### **4.3.4 Silicosis**

Few epidemiological studies were found on chronic silicosis rates in Australia [119, 120]. There were no nationally representative silicosis disease registry data available.

Silicosis cases certified by medical assessment panels in NSW and hospital admissions in Queensland suggest that silicosis rates were low in these two states and have not significantly changed between 2013 and 2017. This contrasts with a recent analysis from the UK SWORD scheme between January

1996 and December 2017 that identified 216 cases of silicosis [143].

Over the past 18 to 24 months, however, there has been a rapid increase in the number of identified cases of accelerated silicosis, and associated PMF in Australia and overseas [47, 56, 144]. The increase is attributable to exposure to dust from engineered stone and was earlier identified in workers manufacturing engineered stone [145]. In response, several jurisdictions have implemented health screening programs for engineered stone workers. As a result of these active case finding investigations, more than 300 workers have been found to have respiratory abnormalities requiring follow up and more than 150 further silicosis cases in engineered stone workers have been identified, many of whom have the accelerated form of the disease.

Jurisdictions across Australia have identified workplaces handling engineered stone and have taken action to ensure that dust controls are implemented, and this should reduce the future burden of silicosis in this industry [121].

In addition, in December 2019, SWA published a new RCS exposure limit of 0.05 mg/m<sup>3</sup> [146]. This exposure standard is half the previous limit of 0.1 mg/m<sup>3</sup> and should result in a reduction in exposure to silica for a number of workers and a consequent reduction in the rate of silicosis.

#### **4.3.5 Asbestosis**

Despite the fact that the use of asbestos has been banned in Australia for more than 15 years, cases of asbestosis are still being diagnosed and compensated because of the long latency of the disease. From 1998 to 2015, there were 2,041 hospitalisations for asbestosis and 487 hospitalisations for asbestos-associated pleural plaques according to the AIHW [131].

From 2007–08 to 2016–17, 12 per cent of the accepted compensation claims in the NDS were for asbestosis, which is the second highest proportion after WRA, (although no OLD was documented for 69 per cent of respiratory claims). NDS claims data for asbestosis between 2000 and 2011 (Figure 6) suggest that asbestosis is declining but AIHW mortality data (Figure 10) shows an increase in mortality over the same period, continuing to 2016. The typical latency period for asbestosis is the likely reason for this increase.

#### **4.3.6 Other occupational lung diseases**

We were not able to identify data on HP or toxic pneumonitis rates among Australian workers.

Recent international estimates have shown that approximately 19 per cent of HP cases are related to work-related exposures [3]. A recent report from the UK Health and Safety Executive reported 70 deaths where farmer's lung was recorded as the underlying cause [67]. The SWORD scheme estimated that there were 38 new cases of HP per year over the last ten years, of which 30 per cent were among women [64]. In 2017, there were 57 incident cases in the UK, whereas there was a smaller number of 14 cases in the 2014–16 period [67]. Given Australian workers can have relevant work-related exposures, these overseas findings on rates suggest that there is an under-recognition of these conditions in Australia.

## 4.4 Trends in occupational lung diseases in Australia

The above section has identified where it has been possible to make some conclusions on trends over time for OLDs in Australia. This is mainly for WRA and asbestosis (slowly reducing based on NDS data) and CWP and silicosis (both increasing based on health screening and claims data).

More longitudinal data, which includes repeated follow-up of participants working in dust-related jobs over a specific period and targeted health screening and surveillance are needed. This will allow the identification of OLDs at an early stage and improve understanding of the trends in OLDs. Despite the fact that more trend data are available than in the 2006 Review [1], there is still insufficient information to accurately evaluate trends for many OLDs in Australia. Most epidemiological studies only examine data on a specific disease in a specific industry/occupation. Trends in overall incidence or prevalence of OLDs can be drawn from an analysis of the NDS (Figure 6) [107], even though there may be under-reporting as previously discussed. This shows an overall reduction in claims for respiratory disease over the last 10 years, in particular for asbestosis and WRA. On the other hand, AIHW NDI data (Figure 10) suggest that between 2000 and 2016, deaths from dust-related lung diseases increased, driven by the increasing number of deaths from asbestosis.

Asbestos importation, supply, installation, use or re-use has been banned in Australia since 2003. It has effectively not been used in domestic construction since the early 1990s. Asbestosis has a latent period of 20 to 30 years from first exposure, so it is expected that although new asbestosis cases will continue to appear in coming decades, the rate will continue to fall.

The apparent reduction in asthma rates could result from a number of factors, such as:

- workplace changes such as the use of powder-free latex gloves in the healthcare industry
- improved control measures such as ventilation when spraying cars with isocyanate paints
- a reduction in the size of an industry sector so that fewer people are exposed, for example fewer foundry workers after closure of car manufacturing, or
- lack of recognition of work-related exposures, leading to under-reporting of this condition.

Many changes have occurred in the Australian workforce since 2006. There has been a reduction in large manufacturing industries making large cohort studies more difficult to undertake. The mining industry has had periodic increases, for example the mining boom which started in 2003 that has since declined. Australian Government data identified that over the past five years, employment in the industry has decreased by 3.6 per cent [147]. These changes will have flow-on effects in OLD rates in mine workers.

Another group who may be at higher risk of OLDs now than previously, are firefighters who have experienced bushfire smoke exposure following an unprecedented bushfire season in the summer of 2019 to 2020 [148, 149].

Since the 1990s, Australia has had very few reported cases of CWP [150]. A mortality surveillance study revealed that out of over 1,000 pneumoconiosis-related fatalities in Australia between 1979 and 2002, CWP accounted for fewer than 100 fatalities, with the largest decline occurring between 1988 and 1996 [114]. However diagnosis of pneumoconiosis in Australia has increased in more recent years

[83]. There have been 39 CWP cases diagnosed in Queensland since 1984 [83] and in 2017 there were two further cases who had worked in both NSW and Queensland coal mines [118]. The NDS data do not yet reflect this increase, there were fewer than five cases per million employees for CWP from 2000 to 2008, whereas no claims were lodged between 2008 and 2011.

Diagnosis of accelerated silicosis in Australia has increased recently [47, 122]. Silicosis cases have emerged particularly in engineered stone workers; the evidence on the prevalence or incidence is still unknown and is being actively investigated. Data from current health screening programs have so far identified 446 individuals with lung abnormalities from three states in Australia (Table 5) and many of these cases are the accelerated form of silicosis, some are PMF. Not all the workers in the industry have been screened yet. As the screening programs continue, further cases can be expected to be identified.

Australia now has more epidemiological and other data sources available compared to 2006. However, the lack of consistency in data collection, limited coverage of high-risk industries and occupations and lack of health screening data remain limitations. Epidemiological data with repeated follow-up from which inferences can be drawn on the burden of WRA and COPD are minimal; sex-stratified data are even more limited. The sample sizes in many studies are often too small to generalise. More large-scale studies in industries with high risk exposures and a national registry of OLDs could assist to clarify the true burden of OLDs in Australia.

## 4.5 Conclusions

There are some key findings that arise from the summary presented above.

### **Finding 1: OLDs continue to be a health concern in Australia**

WRA, COPD and CWP are still a significant concern in Australia. Coal dust exposure in Australian mines, and RCS dust exposure, particularly for engineered stone workers, are currently major health concerns that require continuing education and awareness of the ongoing presence of pneumoconioses. COPD is increasingly being recognised as an important OLD, as more work-related causative exposures are identified. Other OLDs, for which there is very little Australian data, such as HP, may be grossly under-estimated in Australia, based on findings from overseas. This may be because of misdiagnosis and/or a failure to recognise the work-relatedness of these conditions.

### **Finding 2: Further research is needed**

Targeted research should be developed focusing on the identification of at-risk jobs, for example work with engineered stone, jobs in the construction, mining and quarrying industries, firefighters exposed to bushfire smoke and work with bioaerosols. Health screening for workers in these jobs or the establishment of cohorts of these workers would help to identify the incidence of specific OLDs. This research should include the analysis of existing health screening data and research projects being undertaken by universities, research agencies and specialist medical bodies.

Improvement in, and increasing coverage of, health screening schemes and longitudinal studies of workers in the above at-risk jobs, should collect both disease and exposure information. This can help

to identify other industries and occupations at risk and assist in more effective targeting of prevention activities by jurisdictions and industry.

**Finding 3: Thorough application of available data sources will assist in providing a comprehensive national perspective**

Further research can supplement screening activities by identifying emerging OLD risks from new hazards, better evaluating the extent of OLD risks to workers, helping in identifying acceptable workplace exposure limits and assessing the effectiveness of interventions. Hospital admissions and administrative data could be linked together to identify patients who had been hospitalised with an OLD. It is especially important to include former and retired workers, some of whom may have left the industry as a result of lung conditions and are likely to have had longer exposure to develop OLDs. Some OLDs, with long latency, for example COPD and pneumoconioses may only become symptomatic after retirement.

This review identified a range of data sources on OLDs, but most of them are focused on specific occupations and diseases rather than providing a national perspective on OLD or targeting high-risk occupations for further investigation.

There is a need for better and more comprehensive routine respiratory health monitoring where workers are exposed to hazardous substances and targeted health screening in emerging at-risk jobs. Routine health monitoring can allow the identification of early stages of disease so that action can be taken to reduce exposure for the individual, implement early treatment (where available) and perhaps limit or reduce the severity of the disease. Signs of disease in individuals may indicate that exposure is too high and that control measures should be implemented to protect the rest of the workforce and perhaps workers in other workplaces.

**Finding 4: Data collection and management**

The establishment of a national comprehensive dust disease registry, which the National Dust Disease Taskforce recommended, will also be a major step forward in improving estimates of OLD incidence in Australia in the future, as it will systematically collect data on new diagnoses of OLDs regardless of whether the cases are compensated. These data can then be used to provide statistical evidence of the overall scale of the problem and preventive actions can be appropriately targeted by industry and the regulators.

## Appendix

**Table 7 Summary of epidemiological studies which investigated work-related exposure and asthma (primary outcome) in Australia**

| First author, year           | Sample size | Location/ State/ Industry | Study type                            | Mean age (years) | Exposure assessed   | Methods of exposure assessment             | Primary outcome                          | Secondary Outcome/s                 | Key findings  |
|------------------------------|-------------|---------------------------|---------------------------------------|------------------|---|--|--|-------------------------------------|---|
| Barnes et al. (2019) [95]    | 1112        | VIC/ healthcare           | Workplace based cross-sectional study | 39               | Sensitivity to chlorhexidine hand hygiene                         | SR   | Asthma                                   | Respiratory symptoms                | Prevalence of asthma is 13.7% (51/371) in nursing, 12.7% (26/205) in allied health and 11.9% (35/294) in doctors but no association were found.   |
| Dennekamp et al. (2015) [97] | 454 men     | WA/ aluminium industry    | Workplace based cross-sectional study | 31               | Bauxite and mine dust exposure                                    | Self-reported, TEM†, TED                   | Symptoms of asthma                       | Respiratory symptoms, lung function | Prevalence of cough at baseline was 5.9% (11/187), wheeze 11.8% (22/187) and 5.6% (8/144), wheeze 2.1% (3/144) at sixth follow-up.<br>No association was observed between cumulative exposure to bauxite and respiratory symptoms and lung function<br>Overall 4% (24/570) metropolitan firefighters reported asthma, 7% (39/570) reported COPD/emphysema/chronic bronchitis and 10% (57/570) reported chronic respiratory conditions |
| Schermer et al. (2014) [98]  | 570         | SA (SAMFS)/ firefighters  | Workplace based cross-sectional study | 46.3             | Smoke, organic, inorganic dusts, vapours, fumes, organic solvents | SR   | Asthma                                   | COPD, emphysema, chronic bronchitis | Overall 4% (24/570) metropolitan firefighters reported asthma, 7% (39/570) reported COPD/emphysema/chronic bronchitis and 10% (57/570) reported chronic respiratory conditions  |
| Simpson et al. (2014) [100]  | 66          | NSW/ healthcare           | Hospital-based cross-sectional study  | 59.9             | 18 high-risk occupational agents by asthma-JEM                    | Current job history, Asthma-JEM            | Asthma                                   | ---                                 | 17% (11/66) with asthma had occupations with high-risk exposures to latex, cleaning products and wood dust in HCW.<br>14% (9/66) with asthma had low risk exposures such as exhaust fumes   |
| Hoy et al. (2013) [102]      | 792         | Nationwide [TAHS]         | Population-based cohort study         | 45               | 18 high-risk occupational agents by asthma-JEM                    | Lifetime work history calendar, Asthma-JEM | Asthma (New-onset asthma) after 13 years | ---                                 | 12% (94/792) exposed to latex, 10% (82/792) to bioaerosols, 15% (122/792) industrial cleaning agents, 16% (129/792) agriculture, 18% (145/792) to combustion particles/fumes, 11% (84/792) to tobacco smoke, 10% (77/792) to high reactive chemicals, & 8.3% (66/792) to metal fumes  |
| Abramson et al. (2010) [103] | 446         | VIC/ aluminium industry   | Workplace based cohort study          | Median age 30    | Fluorides, SO <sub>2</sub> , BSF, oil                             | Job history TED                            | Asthma                                   | Lung function, BHR used to          | The incidence of wheeze ranged from 10/100 to 28/100 person-years in the men  |

| First author, year           | Sample size        | Location/ State/ Industry                | Study type                             | Mean age (years) | Exposure assessed   | Methods of exposure assessment | Primary outcome | Secondary Outcome/s  | Key findings  |
|------------------------------|--------------------|--|--|------------------|---|--------------------------------|-----------------|--|---|
|                              |                    |  |  |                  | mist and inhalable dust   |                                |                 | define asthma, Atopy, wheeze, chest tightness                                  | Prevalence of wheeze increased in men but not in women. Wheeze was associated with cumulative exposure to BSF, fluoride, inhalable dust, SO <sub>2</sub> but not oil mist. No association between work-related exposure with FEV <sub>1</sub> , FVC and BHR. Lower FEV <sub>1</sub> /FVC was associated with greater cumulative exposure to BSF, fluoride and SO <sub>2</sub> .             |
| Campbell et al. (2007) [151] | 54                 | NSW/ food processing industry            | Workplace based cross-sectional study  | 39.6             | Workplace exposure to lupin among food processing workers                               | SR                             | Asthma          | Lung function  | Overall, 21% (11/53) had positive SPT to lupin. 63.6% (7/11) is currently sensitised to lupin, symptoms include wheeze, rhinitis. 2 subjects had positive methacholine challenge test and 1 had positive bronchial provocation challenge test. Overall, 1 case of WRA, 1 case of work aggravated asthma and 5 cases of occupational rhinitis.   |
| Johnson et al. (2006) [96]   | 5331               | NSW                                      | Population-based cross-sectional study | 18-49            | Any work-related exposures  | SR                             | Asthma          | ---  | Overall 18% (910/5331) asthma cases were diagnosed by doctors. Exposures to ammonia and photographic development were significantly associated with adult-onset asthma. <i>The PAF of adult-onset asthma for work-related exposure was 9.5%.</i>  |
| Kelsall et al. (2004) [99]   | 1424 male veterans | Nationwide /Australian Gulf War veterans | Workplace based cross-sectional study  | NM               | Veterans exposed to asbestos, solvents, lead, radiation, fuels, vibration, noise, paint | SR                             | Asthma          | Respiratory symptoms, chronic bronchitis, lung function and airflow limitation | Overall, 13.7% (190/1424) had ever asthma, 11.5% (165/1424) had doctor diagnosed asthma, 4.8% (68/1424) had FEV <sub>1</sub> /FVC<70%, 10.3% (142/1424) had chronic bronchitis, 1% (11/1424) had chronic obstructive bronchitis and 11% emphysema. There was no difference in lung function. Increased risk of asthma using ECRHS definition (aOR 1.4, 95% CI; 1.1-1.9) but not with other. |

| First author, year            | Sample size   | Location/ State/ Industry | Study type                            | Mean age (years) | Exposure assessed  | Methods of exposure assessment                           | Primary outcome | Secondary Outcome/s                    | Key findings  |
|-------------------------------|---|---------------------------|---------------------------------------|------------------|--|--|-----------------|--|---|
| Wakefield et al. (2003) [152] | 382   | VIC/office workers        | Workplace based cross-sectional study | 38               | SHS at workplace   | SR   | Asthma          | Respiratory symptoms                   | Wheeze: 15%, shortness of breath: 12%, cough: 14%, phlegm: 14, asthma: 18%. SHS at work was associated with an increased risk of wheeze (OR 4.26), frequent cough (OR 2.26).  |
| Ikin et al. (2002) [153]      | 74 in 1 <sup>st</sup> phase in 1995 and 43 in 2 <sup>nd</sup> phase in 1996 | WA/ aluminium industry    | Workplace based cross-sectional study | Ranged 33-38     | Alumina, fluoride, bauxite, inspirable dust, oil mist, SO <sub>2</sub> | Job histories and TEM                                    | Asthma          | Lung function                          | 18.9% (14/74) had previous history of asthma in 1 <sup>st</sup> phase and 7% (3/43) in 2 <sup>nd</sup> phase, 23% (17/74) had previous history of bronchitis in 1 <sup>st</sup> phase and 7% (3/43) in 2 <sup>nd</sup> phase. High reproducibility of lung function was observed with dose-response relationship. |
| Abramson et al. (2001) [154]  | 192   | VIC/ tea packers          | Workplace based cross-sectional study | 40               | Tea packers dust exposure  | SR   | Asthma          | Respiratory symptoms and lung function | 14.7% (16/109) operators, 16.7% (1/6) blenders, 16.7% (4/24) administration workers and 2.9% (1/34) fitters had asthma diagnosed by doctors. 24.8% (27/109) operators, 8.8% (3/34) fitters, 8.3% (2/24) administration workers had shortness of breath in the last 12 months.                                     |
| Mandryk et al. (2000) [101]   | 94  | NSW/ sawmill workers      | Workplace based cross-sectional study | NM               | Hard and soft wood dust, respirable dust                               | Personal dust samplers were used to collect dust samples | Asthma          | Respiratory symptoms and lung function | Overall 7.5% had asthma, 38% had bronchitis, 30% had dry cough, and 25% had wheezing.   |

\* BSF= Benzene Soluble Fraction; HCW=Health care worker; NM=Not mentioned; OR= Odds Ratio; SAMFS=South Australian Metropolitan Fire Service; SHS= Second Hand Smoke; SR=Self-reported; SO<sub>2</sub>=Sulphur Dioxide; SPT=Skin Prick Test; TEM=Task Exposure Matrix; TED=Task Exposure Database;

**Table 8 Summary of epidemiological studies which investigated work-related exposure and COPD (primary outcome) in Australia**

| First author, year           | Sample size                   | Location/ State/Industry | Study type                             | Mean age (years) | Exposure assessed                          | Methods of exposure assessment                 | Definition of COPD   | Key findings   |
|------------------------------|-------------------------------|--------------------------|--|------------------|--|--|--|--|
| Slattery et al. (2017) [110] | 212                           | SA/firefighters          | Workplace based cross-sectional study  | 46.4             | Dust                                       | SR   | FEV <sub>1</sub> /FVC<LLN<br>FEV <sub>1</sub> /FVC<0.7 (pre-BD)  | 8% more lung function abnormality using LLN equation than FEV <sub>1</sub> /FVC ratio  |
| Alif et al. (2017) [37]      | 1335                          | Nationwide [TAHS]        | Population based cross-sectional study | 45               | Dust, gases, pesticides group of exposures | Lifetime work history calendar, ALOHA plus JEM | FEV <sub>1</sub> /FVC<LLN<br>FEV <sub>1</sub> /FVC<0.7 (post-BD),<br>Chronic bronchitis, cough, Phlegm, dyspnoea | Overall, 12% (28/226) exposed to pesticide had COPD, 29% (63/223) exposed to pesticide had chronic bronchitis, 19% (41/221) exposed to pesticides had cough, 18% (39/220) exposed to pesticides had phlegm, 26% (58/225) exposed to pesticides had dyspnoea, 10% exposed to biological dust (76/756), mineral dust (71/707), gases/fumes (91/960) had COPD, 21% exposed to biological dust (159/751), mineral dust (148/702), and 20% exposed to gases/fumes (189/951) had chronic bronchitis. 13-14% exposed to biological dust (104/745), mineral dust (103/696), gases/fumes (125/946) had cough. |
| Alif et al. (2017) [38]      | 1335                          | Nationwide [TAHS]        | Population based cross-sectional study | 45               | Solvents and metals                        | Lifetime work history calendar, ALOHA plus JEM | FEV <sub>1</sub> /FVC<LLN<br>FEV <sub>1</sub> /FVC<0.7 (post-BD)   | Women exposed to chlorinated solvents have more risk of COPD than men. 11% (63/392) exposed to aromatic solvents had COPD, 12% (35/293) exposed to chlorinated solvents had COPD, 10% (55/528) exposed to other solvents had COPD, 13% (41/315) exposed to metals had COPD.  |
| Schermer et al. (2010) [111] | 501 cases and 1324 controls * | SA/firefighters          | Population based cross-sectional study | 43.9             | Dust, smoke and fire exposures             | SR   | FEV <sub>1</sub> /FVC<0.7 (pre-BD)   | FEV <sub>1</sub> /FVC and FEF <sub>25-75</sub> were significantly lower in the firefighters compared to controls. A total of 18.6% (93/501) firefighters had an FEV <sub>1</sub> /FVC<70% (p<0.001). Firefighters with the highest rate of self-reported exposures showed significant lower FEV <sub>1</sub> , FEV <sub>1</sub> % predicted, and FVC values compared with who reported to be less  |

| First author, year          | Sample size | Location/ State/Industry | Study type                             | Mean age (years) | Exposure assessed               | Methods of exposure assessment            | Definition of COPD   | Key findings  |
|-----------------------------|-------------|--------------------------|--|------------------|---------------------------------|---|--|---|
| Matheson et al. (2005) [34] | 1213        | Melbourne, VIC           | Population based cross-sectional study | 45-70            | Dust, gases, group of exposures | Lifetime work history calendar, ALOHA JEM | FEV <sub>1</sub> /FVC<LLN<br>FEV <sub>1</sub> /FVC<0.7 (pre-BD), chronic bronchitis, cough, Phlegm, dyspnoea | exposed to these factors, but the difference was not clinically meaningful.<br>Mineral dust and gases/fumes increased risk of COPD in women<br>Overall 19% (226/1213) had COPD, 4% (49/1213) had chronic bronchitis, 10% (120/1213) had cough, 8% (90/1213) had phlegm & 23% (277/1213) had dyspnoea. |

\* ALOHA= A Lot of Occupational Hygiene Assumptions; BD= Bronchodilator; FEF= Forced Expiratory Flow; LLN= Lower Limit of Normal; SR=Self-reported.

\* Controls were selected from the North West Adelaide Health Study (NWAHS).

**Table 9 Summary of epidemiological studies which investigated work-related exposure and pneumoconiosis (primary outcome) in Australia**

| First author, year        | Sample size | Location/State | Study type                                 | Mean age (years) | Exposure assessed                | Methods of exposure assessment | Outcome/s defined  | Key findings   |
|---------------------------|-------------|----------------|--|------------------|----------------------------------|--------------------------------|--|--|
| Reid et al. (2018) [129]  | 6500        | WA             | Workplace based retrospective cohort study | 29.9             | Asbestos                         | Employment records             | Pneumoconiosis cases were identified from National Death Index | Mortality from pneumoconiosis was higher in Italian born compared to Australian born. SMR=15.9 (95 CI 12.2 to 20.3) for Australian SMR=28.6 (19 TO 41.3) for Italian   |
| Smith et al. (2006) [114] | 1000        | Nationwide     | Population based cross-sectional study     | NM               | Coal dust, asbestos, silica dust | NDI                            | CWP  | In the first group (1979–1981), silicosis accounted for 60% of all deaths, followed by asbestosis (31%).<br>By 2002, asbestosis was causing 78% of all pneumoconiosis related fatalities, while silicosis was only accounting for 19%.<br>In both periods, 56% death from asbestosis, 38% from silicosis and 6% from CWP.<br>Crude mortality rates for asbestosis in 2002: men 5.3 cases per million, women 0.1 cases per million. |

\*CI=Confidence Interval; NM=Not Mentioned.

**Table 10 Summary of epidemiological studies which investigated work-related exposure and other lung conditions (not in above tables) in Australia**

| First author, year           | Sample size                  | Location/ State/Industry      | Study type                            | Mean age (years) | Exposure assessed   | Methods of exposure assessment                   | Outcome/s investigated                 | Key findings  |
|------------------------------|------------------------------|-------------------------------|---------------------------------------|------------------|---|--|--|---|
| <b>Lung function</b>         |                              |                               |                                       |                  |   |  |  |   |
| Alif et al. (2019) [39]      | 767                          | Nationwide [TAHS]             | Population-based longitudinal study   | 50               | Dust, gases, pesticides, solvents, metals exposures               | Lifetime work history calendar, ALOHA plus JEM   | Lung function decline                  | Women exposed to aromatic solvents were increased risk of FEV <sub>1</sub> and FEV <sub>1</sub> /FVC ratio decline than men.<br>Participants exposed to aromatic solvents had 14mL greater lung function decline compared to participants unexposed.  |
| Schermer et al. (2013) [40]  | 281 cases and 933 controls * | SA/firefighters               | Population-based longitudinal study   | 43.3             | Dust, smoke and fire exposures                                    | SR   | Lung function decline                  | Overall 26% firefighters and 39% controls showed accelerated FEV <sub>1</sub> decline.<br>2) Firefighters had a lower odd of accelerated FEV <sub>1</sub> decline compared to controls.<br>3) Firefighters who never or rarely used respiratory protection during fire knockdown had a higher odd of accelerated FEV <sub>1</sub> decline compared with those who used it often or frequently |
| Schermer et al. (2010) [155] | 488 men                      | SA/ firefighters              | Workplace based cross-sectional study | 43.8             | Smoke, organic, inorganic dusts, vapours, fumes, organic solvents | SR   | Lung function, chronic cough           | Overall 12% (58/488) abnormal spirometry, 9% (45/488) abnormal impulse oscillometry, 20% (100/488) firefighters had respiratory symptoms.<br>67% (39/58) had spirometry FEV <sub>1</sub> /FVC below the LLN.  |
| Wu et al. (2004) [112]       | 580 men                      | Nationwide/ coke oven workers | Workplace based cohort study          | 38.4             | Coke oven exposure  | Job histories were obtained from human resources | Lung function decline                  | Each year of working in the 'operation' group was associated with 0.7mL FVC and 0.8 mL FEV <sub>1</sub> decline.  |
| Wu et al. (2004) [156]       | 764 men                      | Nationwide/ coke oven workers | Workplace based cross-sectional study | 41.0             | Coke oven exposure  | Job histories were obtained from human resources | Respiratory symptoms and lung function | Working in "operation" increased the risk of cough (odds ratio [OR]=2.37), phlegm (OR=2.55) and shortness of breath (OR=1.52).<br>Cross-sectionally, each year of working in operation was associated with 10mL reduced FEV <sub>1</sub> and 21mL reduced FVC.  |

| First author, year                        | Sample size | Location/ State/Industry       | Study type                            | Mean age (years) | Exposure assessed  | Methods of exposure assessment      | Outcome/s investigated                 | Key findings  |
|---|-------------|--------------------------------|---------------------------------------|------------------|--|-------------------------------------|--|---|
|   |             |                                |                                       |                  |  |                                     |  | Longitudinally, each year of working in operation was associated with 1.5mL extra annual decline in FEV <sub>1</sub> .  |
| Fritschi et al. (2003) [157]              | 1615 men    | Nationwide/ aluminium industry | Workplace based cross-sectional study | 37.8             | Aluminium smelters, fluoride, inspirable dust, oil mist, SO <sub>2</sub> | Air monitoring data, TEM            | Respiratory symptoms and lung function | Cumulative exposure to fluoride, SO <sub>2</sub> , and inspirable dust in aluminium smelters were associated with wheeze and chest tightness. Subjects exposed to oil mist had lower lung function compared to unexposed.   |
| Beach et al. (2001) [158]                 | 690         | WA/miners                      | Workplace based cross-sectional study | NM               | Bauxite  | Self-reported job histories and TEM | Respiratory symptoms and lung function | Overall 31% (216/690) had reported rhinitis, 14.2% (88/690) had dyspnoea, 18.4% (120/690) had wheeze, 12.2% (76/690) had chest tightness. All these were reported as work-related symptoms. FEV <sub>1</sub> decreased by 7.3 ml. Significant association between FEV <sub>1</sub> and duration of total employment.  |
| Musk et al. (2000) [159]                  | 2639        | WA/ aluminium industry         | Workplace based cross-sectional study | 36-41            | Bauxite, alumina, caustic mist   | SR occupational histories           | Respiratory symptoms and lung function | Work related wheeze, chest tightness, shortness of breath, and rhinitis were reported by 5.0%, 3.5%, 2.5%, and 9.5% of participants respectively. After adjustment for confounders, most groups of production employees reported a greater prevalence of work-related symptoms than did office employees. After adjustment for confounders, subjects reporting work related wheeze, chest tightness, and shortness of breath had significantly lower mean levels of FEV <sub>1</sub> (186, 162, and 272 ml respectively). |
| <b>Interstitial lung disease</b>          |             |                                |                                       |                  |  |                                     |  |   |
| Dale et al. (2015) [160]                  | 48          | NSW                            | Workplace based cross-sectional study | 72               | Dust and physical activity   | Doctor diagnosed cases              | ILDs                                   | Significantly reduced physical activity in people with ILDs compared with healthy control group.  |
| <b>Non-malignant respiratory diseases</b> |             |                                |                                       |                  |  |                                     |  |   |
| Friesen et al. (2009) [161]               | 5770        | WA/bauxite miners              | Workplace based cohort study          | 32               | Alumina, bauxite dust  | Job history, TEM                    | Non-malignant respiratory diseases     | Ever exposure to bauxite was associated with 2.4 times higher risk of developing non-malignant respiratory disease  |

| First author, year            | Sample size | Location/ State/Industry | Study type                            | Mean age (years) | Exposure assessed      | Methods of exposure assessment  | Outcome/s investigated    | Key findings   |
|-------------------------------|-------------|--------------------------|---------------------------------------|------------------|------------------------|---------------------------------|---------------------------|--|
| <b>Respiratory symptoms</b>   |             |                          |                                       |                  |                        |                                 |                           |  |
| Wakefield et al. (2005) [162] | 91          | VIC/office, casino       | Workplace based cross-sectional study | 32.3-40.4        | SHS smoke at workplace | SR                              | Respiratory symptoms      | Wheeze: 22%, shortness of breath: 25%, cough: 27%, phlegm: 27% for casino and club workers. Compared to office workers, casino workers had higher levels of exposure to SHS. |
| <b>Asbestosis</b>             |             |                          |                                       |                  |                        |                                 |                           |  |
| Murray et al. (2016) [163]    | 906         | WA/asbestos workers      | Workplace based cross-sectional study | 68.8             | Asbestos               | WAARP occupational data sources | LDCT screening/asbestosis | Prevalence of pleural plaque was 64% (580/906), ILD was 232/906 (25.7%), and 8.5% (104/906) asbestosis cases were identified   |

\* ALOHA= A Lot of Occupational Hygiene Assumptions; FEF= Forced Expiratory Flow; LDCT=Low Dose Computer Tomography; LLN= Lower Limit of Normal; NM=Not Mentioned; OR=Odds Ratio; SR=Self-reported; SHS= Second Hand Smoke; TEM=Task Exposure Matrix; WAARP= Western Australian Asbestos Review Programme; WA= Western Australia

\* Controls were selected from the North West Adelaide Health Study (NWAHS).

**Table 11 Summary of epidemiological studies which used workers' compensation, health screening or Coal Board data to report work-related exposure and lung diseases in Australia**

| First author, year                  | Sample size | Location/State            | Mean age (years) | Exposure assessed   | Methods of exposure assessment                  | Primary outcome/s assessed | Secondary outcome/s                                  | Key findings  |
|-------------------------------------|-------------|---------------------------|------------------|---------------------|---|----------------------------|--|---|
| <b>Asthma</b>                       |             |                           |                  |                     |   |                            |  |   |
| Donoghue et al. (2011) [104]        | 329         | Nationwide using AAC data | ---              | Aluminium smelters  | Workers' health monitoring data                 | Asthma                     | --   | Overall, 55% (180/329) had asthma occurred in potroom production, 74% (243/329) occurred in bath exposed areas, The mean proportion of employees exposed to bath was 50%. Incidence rate of asthma in 1992 was 9.46/1000/year and 0.36/1000/year in 2006, declined 96.2%. |
| Hannaford-Turner et al. (2010) [90] | 3856        | NSW                       | ≥50              | Any exposure        | Diagnosed cases from SABRE                      | Asthma                     | Asbestosis, Pleural Plaques, DPT, silicosis          | Overall 32% (1218/3856) had Pleural Plaques, 22% (836/3856) had DPT, 10% (366/3856) Asbestosis, 2% (90/3856) had silicosis, and 2% (89/3856) had asthma.  |
| Elder et al. (2004) [89]            | 520         | VIC and Tasmania          | 55.7             | Any exposure        | Doctor diagnosed cases                          | Asthma                     | Allergic alveolitis, bronchitis, pneumoconiosis      | Asthma was the most commonly reported case; the overall incidence was 30.9 [95% CI; 26.8-35.5] cases/million workers/year. The most prevalent cases were: 33% (170/520) had asthma, 8% (43/520) had pneumoconiosis, and 7% (37/520) had bronchitis.                       |
| <b>Silicosis</b>                    |             |                           |                  |                     |   |                            |  |   |
| Berry et al. (2004) [119]           | 1249        | NSW                       | NM               | Silica dust/RCS     | Diagnosed cases from NSW workers compensation   | Silicosis                  | --   | 35% (515/1467) silicosis patients died from non-malignant respiratory cause   |
| de Klerk et al. (2002) [120]        | 408         | WA                        | NM               | RCS, gold miners    | PMP and compensated cases, dust                 | Silicosis                  | --   | From 1974-1996, no compensated cases of silicosis in WA miners exposed to RCS were identified   |
| <b>Lung function</b>                |             |                           |                  |                     |   |                            |  |   |
| Park et al. (2014) [164]            | 577         | NSW                       | 66.7             | Asbestos            | Workers' compensation (dust disease icare) data | Lung function              | Asbestosis, DPT, asbestosis and DPT, pleural plaques | Overall, 222/577 (38.6%) had FEV <sub>1</sub> <80% predicted, 139/577 (24.2%) had FVC<80% predicted, 217/577 (37.7%) had FEV <sub>1</sub> /FVC<70%, 249/577 (43.8%) DLco<80% predicted, 75/577 (13.2%) had DLco/VA<80% predicted, 147/577 (25.6%) had PEF<80% predicted   |
| Kizil et al. (2002) [165]           | 33          | NSW                       | NM               | Coal dust on miners | Coal Board database                             | Lung function              | --   | The mean loss of lung function, FEV <sub>1</sub> was 73.7 mL  |

\*AAC= Australian Aluminium Council; DPT= Diffuse Pleural Thickening; DLco or TLco= diffusing capacity or transfer factor of the lung for carbon monoxide (CO); PMP=Pneumoconiosis Medical Panel; PEF=Peak Expiratory Flow; VA = alveolar volume.

**Table 12 Summary of clinical case reports and case series on work-related exposure and lung diseases in Australian workers**

| First author, year             | Number of clinical cases/reports | Study population           | Exposure assessed | Methods of exposure assessment | Outcome/s assessed                           | Key findings   |
|--------------------------------|----------------------------------|----------------------------|-------------------|--------------------------------|--|--|
| <b>Asthma</b>                  |                                  |                            |                   |                                |  |  |
| Hoy et al. (2010) [5]          | One clinical case                | VIC                        | Flour dust        | Doctor diagnosed               | WRA (Baker's asthma)                         | 18-year old man presented with a history of seasonal hay fever, itchy eyes, stuffy nose, cough, shortness of breath and wheeze from last 2 months. He has recently been on holiday and felt better during that time. He is working in a bakery for the last 12-months. His main role is to weigh the flour. His symptoms improve modestly over the weekend and recur within minutes of starting work.  |
| <b>Silicosis</b>               |                                  |                            |                   |                                |  |  |
| Turner et al. (2019) [53]      | Three clinical cases             | QLD                        | RCS dust          | Doctor diagnosed               | Engineered stone associated silicosis        | A 45-year-old man had a 15-year history of installing artificial stone kitchen benchtops and stonemasonry. He was unemployed for 18 months because of his medical condition was considered for lung transplantation but considered unsuitable and recently died in hospital.<br>A 47-year-old man with a 23-year history of dry-cutting marble blocks with a power-saw, installing artificial stone benchtops, monumental work and tombstones, was referred with progressive exertional dyspnoea and arthropathy.<br>A 53-year old man with a 20-year history of dry-cutting marble, granite and artificial stone benchtops was referred with dyspnoea and sicca symptoms. He underwent successful bilateral single sequential lung transplantation. |
| Levin et al. (2019) [166]      | Two clinical cases               | VIC                        | Silica dust       | Doctor diagnosed               | Engineered stone associated silicosis        | A 47-year-old man who had worked as a stone benchtop fabricator for nine years. His work frequently involved cutting and grinding artificial stone without water dust suppression, minimal exhaust ventilation, and only occasional provision of a face mask. He underwent right single lung transplantation.<br>A 36-year-old non-smoking man worked as a stonemason for six years fabricating benchtops, primarily from artificial stone material. He typically used high-powered hand tools to cut the stone without water dust suppression. He underwent bilateral sequential lung transplantation.  |
| McBean et al. (2018) [167]     | Five clinical cases              | Diagnosed cased with CMDLD | Mine dust         | HRCT and SR                    | CWP, Silicosis, MDP, PMF, DDF                | Mining industry workers developed CWP with 34 years' work, silicosis with 25 years', MDP with 37 years', MDP with 25 years' and DDF with 30 years of work  |
| Semasinghe et al. (2018) [168] | One clinical case                | QLD                        | RCS dust          | SR and doctor diagnosed        | Progressive worsening dyspnoea and silicosis | 35-year-old patient working as a stonemason developed chronic silicosis with progressive massive fibrosis (PMF)  |

| First author, year   | Number of clinical cases/reports | Study population   | Exposure assessed                                | Methods of exposure assessment | Outcome/s assessed                                      | Key findings  |
|--|----------------------------------|--|--|--------------------------------|---|---|
| Hoy et al. (2018) [47]   | Seven clinical cases             | Diagnosed cases with Engineered stone associated silicosis | RCS dust   | SR and doctor diagnosed        | Engineered stone associated silicosis                   | Cases were identified from QLD, VIC and NSW. All 7 cases were men with median age 44 years (26-61 years), 3 workers were qualified stonemasons born in Australia and 4 had no formal qualifications, and born overseas. All men performed dry cutting of artificial stone using hand tools. Only 3 were provided respiratory protection and in the form of disposable masks. Median duration of exposure was 7.3 years, ranged from 4-10 years. 6 cases with PMF and 1 chronic silicosis.   |
| Matar et al. (2017) [169]  | One clinical case                | NSW  | RCS dust   | Doctor diagnosed case          | Engineered stone associated silicosis                   | A 54-year-old man, born in Vietnam, came to Australia as a refugee, presented 6-year history of chronic cough and breathlessness without previous respiratory illness. Diagnosed as complicated silicosis with PMF  |
| <b>Hypersensitivity pneumonitis or extrinsic allergic alveolitis</b> |                                  |  |  |                                |   |   |
| Hoy et al. (2012) [24]   | One clinical case                | VIC  | Organic dust exposure                            | Doctor diagnosed case          | HP/extrinsic allergic alveolitis/ Mushroom workers lung | 23 years man presented with cough, shortness of breath, weight loss from last 2 months. He also reported intermittent fevers and flu-like symptoms over the same period. During a recent 2-week holiday to Bali he felt significantly better, but after returning home he has had a recurrence of symptoms. Occupational and exposure history identifies him as commencing work at a mushroom farm 12 months ago where he is exposed to dust from the mixing of mushroom compost. His cough and chest tightness usually start in the afternoon at work and persist into the evening. Other workers at the mushroom farm have reported similar symptoms and have had to leave the workplace as a result. |
| Hoy et al. (2007) [132]  | Two clinical cases               | VIC  | Organic dust exposure                            | Doctor diagnosed cases         | HP or Mushroom workers lung                             | <b>Case 1:</b> 36-year-old non-smoker man, employed at the mushroom farm for 8 years and had worked in the spawning shed for 3 years. He complained 4-months history of non-productive cough worse in the afternoons at work and improved on weekends. HRCT showed changes consistent with hypersensitivity pneumonitis.<br><b>Case 2:</b> 40-year-old former smoker man, employed at the mushroom farm for 6 years, had experienced in spawning shed. He complained about dyspnoea, cough, chest tightness, myalgias and pyrexia. Spirometry was normal but TLco was impaired. HRCT showed patchy ground glass opacities in both the lower lobes.  |
| <b>Alveolar proteinosis</b>  |                                  |  |  |                                |   |   |
| Chew et al. (2016) [133]   | One clinical case                | QLD  | Aluminium dust working in boat building industry | Doctor diagnosed case          | Alveolar proteinosis                                    | 46-year old man, lifelong non-smoker presented with increasing dyspnoea, dry cough and reduced exercise tolerance from last 12 months. After several investigation, he was diagnosed as secondary alveolar proteinosis  |

\*; DDF= Dust-related Diffuse Fibrosis; DLco or TLco= diffusing capacity or transfer factor of the lung for carbon monoxide (CO); HRCT=High Resolution Computed Tomography; MDP= Mixed Dust Pneumoconiosis; SR=Self-reported; PMF= Progressive massive fibrosis.

**Table 13 Summary of reports and reviews (published in a peer-review journal) reported the association between work-related exposure and lung diseases**

| First author, year                  | Study type       | Study population/source of data | Exposure assessed      | Methods of exposure assessment | Outcome/s assessed   | Key findings   |
|-------------------------------------|------------------|---------------------------------|------------------------|--------------------------------|--|--|
| <b>Coal workers' pneumoconiosis</b> |                  |                                 |                        |                                |  |  |
| Han et al. (2018) [118]             | Review           | Published papers                | Coal mine dust         | Doctor diagnosed cases         | CWP  | In Australia, 1000 pneumoconiosis fatalities were reported between 1979-2002. Queensland government reported 26 cases of CWP and 9 cases of silicosis from 1986 to 2018. Prevalence is lower than asbestosis and asthma.   |
| McCall et al. (2017) [170]          | Report           | Published papers and reports    | Coal dust              | Doctors diagnosed              | CWP  | 23 miners or former coal miners, who had worked in Queensland state, have been diagnosed with coal worker's pneumoconiosis (CWP), as black lung  |
| Zosky et al. (2016) [41]            | Review           | Published papers and reports    | Coal dust              | Published data sources         | CWP  | 2001 and 2003 there were 750 new cases of pneumoconiosis (including CWP, asbestosis and silicosis), with 92 deaths in 2003   |
| <b>Silicosis</b>                    |                  |                                 |                        |                                |  |  |
| Newbiggin et al. (2019) [171]       | Review           | Review of cases                 | RCS dust               | Retrospective review           | Silicosis  | 78 cases were men, with a mean age 34.1 years, 36 of them were diagnosed accelerated and 42 chronic silicosis.   |
| <b>Asbestosis</b>                   |                  |                                 |                        |                                |  |  |
| Soeberg et al. (2018) [57]          | Narrative Review | Published papers and reports    | Asbestos               | Hospitalised cases             | Asbestosis   | Between 1998 to 2015, 2041 cases were admitted to hospitals for asbestosis. In the same period, 833 hospitalisations for respiratory conditions because of the inhalation of chemicals, gases, fumes and vapours. From 1998 to 2015, 487 or 43% hospitalisations were recorded as pleural plaques with the presence of asbestos.   |
| Park et al. (2008) [60]             | Review           | NSW data sources                | Asbestos               | SABRE                          | Asbestosis, pleural plaques, DPT                           | 1) 30.1% cases with pleural plaques, 23.7% DPT, and 10.2% asbestosis. 2) New cases of DPT, pleural plaques, and asbestosis were 663, 4315 and 290 respectively.  |
| <b>Occupational lung diseases</b>   |                  |                                 |                        |                                |  |  |
| Hoy et al. (2017) [2]               | Narrative Review | Published papers and reports    | Work-related exposures | ---                            | Asthma, COPD, ILDs, obliterative bronchiolitis, asbestosis | This review highlighted some major gaps in Australian data sources. This includes workers' compensation statistics, which provide very little insight into specific OLDs and are insufficient to target preventive strategies into the workplaces. Most of the conclusions in Australia literature about OLDs are based on overseas data sources. This lack of information is a major barrier to develop targeted workplace interventions. |

\* DPT= Diffuse Pleural Thickening.

**Table 14 Summary of grey literature reporting on work-related exposure and lung diseases in Australia**

| First author, year                    | Publication type         | Study population | Exposure assessed                   | Methods of exposure assessment | Outcome/s assessed               | Key findings  |
|---------------------------------------|--------------------------|------------------|-------------------------------------|--------------------------------|----------------------------------|---|
| <b>Asthma</b>                         |                          |                  |                                     |                                |                                  |   |
| AIHW (2008) [106]                     | AIHW bulletin            | Nationwide       | All types of work-related exposures | Various data sources           | Asthma                           | NDS for compensation-based statistics from 2000-01 to 2004-05 reports that,<br><i>2660 accepted claims for OLDs, 395 (15%) for asthma, and 945 (36%) for asbestosis.</i><br>Number of accepted claims for asbestosis increased to 245 in 2004-05 from 155 in 2000-01 period.<br>Number of accepted claims for OA fallen from 95 (20%) to about 70 (11%).<br>In 2004–05, about 51% of the accepted asthma claims were for female workers, and about 33% were for workers aged 45–54 years.<br>Manufacturing (25%) and health/community services (23%) were the industries with the highest proportion of accepted claims.<br><b>PAR for asthma was reported 9-15%.</b><br>1850 and 3090 new cases of OA in 2003, giving an annual incidence rate of 195-325 cases per million workers per year.<br>PAR was reported 15%. |
| Sim et al. (2005) [6]                 | State Government report  | VIC              | All types of work-related exposures | Published studies              | Asthma                           |   |
| <b>Coal workers' pneumoconiosis</b>   |                          |                  |                                     |                                |                                  |   |
| Newbigin et al. (2019) [43]           | Case series              | QLD              | Coal mine dust                      | Confirmed cases                | CWP, Silicosis, MDP, DDF, COPD   | Total number of cases; CWP=27, Silicosis=11, MDP=18, DDF=5, COPD=22   |
| Queensland Government (2017) [83]     | State government website | QLD              | Coal mine dust                      | Diagnosed cases                | CWP, COPD, silicosis, asbestosis | From 1984-present, total reported number of 109 mine dust lung disease, cases were identified, of which, 72 pneumoconiosis, 37 CWP, and 18 silicosis cases [Updated on 30 June 2019]  |
| Parliamentary Committees (2017) [117] | State government report  | QLD coal miners  | Coal mine dust                      | Doctor diagnosed cases         | CWP                              | 21 QLD coal miners have now been diagnosed with CWP with the following features;<br>1) 17 cases were working at the time of diagnosis,<br>2) 3 were retired or former coal miners at the time of diagnosis,<br>3) 2 also worked in NSW and QLD coal mine<br>4) Age ranged from 38-74 with an average age of 56.   |
| Sim et al. (2016) [113]               | Technical report         | QLD              | Coal mine dust                      | CMWHC                          | CWP/CMDLD                        | The report identified concerns regarding the quality, reading and reporting of chest X-rays; the quality and reading of spirometry; failings in the handling of health assessment records.  |
| Queensland Government (2016) [172]    | State government report  | QLD              | Coal mine dust                      | Diagnosed cases                | CWP/CMDLD                        | 15 cases of pneumoconiosis were diagnosed<br>2 are described as complex (presenting with multiple conditions)<br>Current ages range from 38 to 73 with an average age of 56<br>1 involves an above ground worker with no underground experience   |

| First author, year            | Publication type                               | Study population | Exposure assessed      | Methods of exposure assessment         | Outcome/s assessed          | Key findings   |                         |                           |
|-------------------------------|--|------------------|------------------------|--|-----------------------------|--|-------------------------|---------------------------|
|                               |  |                  |                        |  |                             | 4 have substantial overseas coal mine experience (UK and USA)<br>2 worked in NSW as well as in QLD.  |                         |                           |
| Joy et al. (2012) [173]       | Technical paper                                | NSW              | Coal mine dust         | NSW coal mine industry data            | CWP                         | Pneumoconiosis prevalence is near zero in NSW from early 2000. Prevalence was <2% in early 1980s.  |                         |                           |
| Coal Services NSW (2010) [85] | Annual report                                  | NSW              | Coal mine dust         | NSW coal mine industry data            | CWP                         | Since late 1970, the prevalence of pneumoconiosis in the NSW have started to decline and since then, it was continuing to be less than 0.5%.   |                         |                           |
| Other pneumoconioses          |  |                  |                        |  |                             |  |                         |                           |
| Poulos et al. (2014) [82]     | The Lung Foundation Australia technical report | Nationwide       | Work-related exposures | Various data sources                   | Other pneumoconiosis /CWP   | <b>Hospitalisation data:</b><br>1)In 2011-12 there were 197 hospitalisations for pneumoconiosis among people aged 30 years and over.<br>2)In 2011-12, there were 1,040 patient-days for pneumoconiosis among people aged 30 years and over. Men had many more patient-days because of pneumoconiosis (985) than women (55).<br>3)It was estimated that pneumoconiosis accounts for 1,959 DALYs in Australia with the burden being more than double in men (1,369 DALYs) than in women (589 DALYs).<br>4)In 2012, there were 120 deaths (0.14 per 100,000 adults aged 30 or more years) attributed to pneumoconiosis in Australia, with men accounting for 117 (97.5%) of the deaths. |                         |                           |
| Silicosis and asbestosis      |  |                  |                        |  |                             |  |                         |                           |
| icare (2018) [84]             | Annual report                                  | NSW              | Various Dust           | Medical assessment panel certification | Asbestosis, ARPD, Silicosis | <b>Cases certified by medical assessment panel by disease type and year as % of all diseases</b>   |                         |                           |
|                               |  |                  |                        |  |                             | <b>2017-18</b>   |                         |                           |
|                               |  |                  |                        |  |                             | Asbestosis<br>9.8% (32/324)  | ARPD<br>21% (68/324)    | Silicosis<br>2.5% (8/324) |
|                               |  |                  |                        |  |                             | <b>2016-17</b>   |                         |                           |
|                               |  |                  |                        |  |                             | Asbestosis<br>6.7% (21/311)  | ARPD<br>25% (79/311)    | Silicosis<br>1.9% (6/311) |
|                               |  |                  |                        |  |                             | <b>2015-16</b>   |                         |                           |
|                               |  |                  |                        |  |                             | Asbestosis<br>10% (30/289)   | ARPD<br>22.8% (66/289)  | Silicosis<br>3.1% (9/289) |
|                               |  |                  |                        |  |                             | <b>2014-15</b>   |                         |                           |
|                               |  |                  |                        |  |                             | Asbestosis<br>1.3% (4/312)   | ARPD<br>33.7% (105/312) | Silicosis<br>2.9% (9/312) |
|                               |  |                  |                        |  |                             | <b>2013-14</b>   |                         |                           |
|                               |  |                  |                        |  |                             | Asbestosis<br>7.6% (21/276)  | ARPD<br>25.7% (71/276)  | Silicosis<br>3.3% (9/276) |

| First author, year                     | Publication type                | Study population | Exposure assessed  | Methods of exposure assessment   | Outcome/s assessed                    | Key findings   |
|--|---------------------------------|------------------|--|--|---------------------------------------|--|
| Seneviratne et al. (2018) [174]        | Conference abstract             | NSW              | RCS dust   | SR   | Engineered stone associated silicosis | 36 stoneworkers were identified with abnormal x-ray, mean age was 55 years (range 23-75). Workers who had worked in the industry for many years had not undergone a complete health monitoring including chest x-ray and spirometry.                 |
| Thiruvarudchel van et al. (2017) [127] | Conference abstract/case series | NSW              | RCS dust   | Doctor diagnosed cases   | Engineered stone associated silicosis | Four cases were identified from NSW and one case was waiting for lung transplantation at the time of study.  |
| <b>Other lung diseases</b>             |                                 |                  |  |  |                                       |  |
| Abramson et al. (2018) [175]           | Conference abstract             | Nationwide       | Asbestos, organic dust, inorganic mineral dust, other dusts, metal dusts | FINJEM<br>AsbJEM   | IPF                                   | Work-related exposure to respirable dust and asbestos were independently associated with the risk of IPF.  |
| Yates et al. (2018) [176]              | Conference abstract/case report | NSW              | Hard metal dust  | Self-reported  | Rare type of OLD                      | Multinucleated giant cells were found in airspaces in HRCT. Metal particles were present in lung tissue indicate Hard Metal Lung Disease   |
| De Vos (2008) [177]                    | Doctoral thesis                 | WA               | Bushfire smoke   | Three types of filters were used. 1) Particular filter, 2) Organic vapour filter, 3) Organic vapor/formaldehyde filter | Lung function                         | Across the study population there was a significant decline in FEV <sub>1</sub> both after 60 minutes and 120 minutes compared to the baseline measurements. However, this decline was not associated with the type of filter used during the trial. |

\* ARPD= Asbestosis induced pleural disease; AsbJEM= Asbestos-specific Job exposure Matrix; CMWHC= Coal Mine Workers' Health Scheme; HRCT= High-resolution computed tomography; FINJEM=Finnish Job Exposure Matrix; SR=Self-reported.

**Table 15 Summary of national workers' compensation statistics to report work-related exposure and lung diseases in Australia**

| First author, year                  | Publication type                 | Study population | Methods of exposure assessment                   | Outcome/s assessed   | Key findings  |
|-------------------------------------|----------------------------------|------------------|--|--|---|
| Safe Work Australia (2016-17) [92]  | Workers' Compensation Statistics | Nationwide       | Claimed cases                                    | Respiratory diseases (all)   | 215 claims for respiratory diseases. Trends of claims for respiratory diseases also declined 27% from 2000-01 (285 claims) to 2016-17 (205 claims). Number of claims from chemical or chemical products also declined 47% from 2000-01 (1390 claims) to 2016-17 (735 claims).   |
| Safe Work Australia (2015-16) [93]  | Workers' Compensation Statistics | Nationwide       | Claimed cases                                    | Respiratory diseases (all)   | 185 claims for respiratory diseases. Trends of claims for respiratory diseases also declined 21% from 2000-01 (285 claims) to 2015-16 (225 claims).   |
| Safe Work Australia (2014-15) [94]  | Workers' Compensation Statistics | Nationwide       | Claimed cases                                    | Respiratory diseases (all)   | During 2014-15, there were 210 claims for respiratory diseases. Trends of claims for respiratory diseases also declined 20% from 2000-01 (285 claims) to 2014-15 (225 claims).  |
| Safe Work Australia (2014) [107]    | Technical report                 | Nationwide       | Compensation claim data and hospitalisation data | OLDs, Asbestosis, asthma, pneumoconiosis because of asbestos, mineral fibre, coal dust | <b><u>Workers compensation data:</u></b><br>Downward trend in compensation claims for all OLDs from 2000 to 2011. The rate of claims was 143 per million in 2000-01 and 73 in 2010-11 (Figure 1). Asbestosis claims declined to 7 claims per million from 36, asthma 15 claims per million over the 7-year period.<br><b><u>Hospitalisation data:</u></b><br>Increased hospitalisation in 2004-05 period for pneumoconiosis because of exposure to asbestos, mineral fibre, organic dust, coal dust. After 2004-05, the rate of OLD fell to 27 hospitalisations per million in 2009-10. |
| Safe Work Australia (2013-14) [178] | Workers' Compensation Statistics | Nationwide       | Claimed cases                                    | Respiratory diseases (all)   | 200 claims for respiratory diseases. Trends of claims for respiratory diseases also declined 28% from 2000-01 (285 claims) to 2013-14 (205 claims).   |
| Safe Work Australia (2012-13) [179] | Workers' Compensation Statistics | Nationwide       | Claimed cases                                    | Respiratory diseases (all)   | 205 claims for respiratory diseases. Trends of claims for respiratory diseases also declined 16% from 2000-01 (285 claims) to 2012-13 (235 claims).   |
| Safe Work Australia (2011-12) [180] | Workers' Compensation Statistics | Nationwide       | Claimed cases                                    | Respiratory diseases (all)   | 220 claims for respiratory diseases. Trends of claims for respiratory diseases also declined 5% from 2000-01 (285 claims) to 2011-12 (220 claims).  |

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