From: Tim Noonan >
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To: Law; Peta Leemen
Cc: James Mathison; Pauline Kavanagh; Christian Fanker; SIRA Ministerial Unit; Tim Noonan
Subject: 2021 review of the dust diseases scheme - SIRA - Professor Driscoll's evidence review report (dated September 2021)
Attachments: Prof Driscoll evidence review report_final_ September 2021.pdf

Good morning

Please find attached Professor Driscoll's evidence review report (dated September 2021) as requested by the Committee.

Regards

Tim

Tim Noonan
Manager | Governance
State Insurance Regulatory Authority

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Update of Schedule 1 of the New South Dust Diseases Act

Final report

Tim Driscoll

ELMATOM Pty Ltd

September 2021

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BACKGROUND OF THE AUTHOR

This report was prepared by Professor Tim Driscoll (MBBS BSc(Med) MOHS PhD FAFOEM FAFPHM). Professor Driscoll is a specialist in occupational medicine and public health medicine and an independent consultant in epidemiology, occupational health and public health.

GLOSSARY

AIHW	Australian Institute of Health and Welfare
CI	Confidence interval
CWP	Coal workers' pneumoconiosis
DALYs	Disability-adjusted Life-Year
GBD	Global Burden of Disease
IARC	International Agency for Research on Cancer
NSW	New South Wales
RR	Relative risk
SIRA	State Insurance Regulatory Authority
TSANZ	Thoracic Society of Australia and New Zealand
UI	Uncertainty interval

EXECUTIVE SUMMARY

Background

In New South Wales, compensation and other payments related to dust diseases are covered under the Workers' Compensation (Dust Diseases) Act 1942 (the Act). Schedule 1 of the Act contains a list of diseases covered by the Act. In 2018, the Law and Justice Committee undertook a review of the dust diseases scheme. Recommendation 6 of that review was "*That the State Insurance Regulatory Authority liaise with key stakeholders, including the Thoracic Society of Australia and New Zealand, regarding updating of the list of dust diseases contained in Schedule 1…*" Suggestions for expansion of Schedule 1 of the Act have been made by the Thoracic Society of Australia and New Zealand (TSANZ).

The State Insurance Regulatory Authority requested a project that addressed the above issues. The project required the investigator to provide advice on which of the disorders proposed by the Thoracic Society of Australia and New Zealand (and any other disorders not currently in Schedule 1 of the Dust Diseases Act) should be recommended to be considered a 'dust disease'. The required information is presented in this report.

Approach

Studies relevant to dust diseases were identified through a search of the published literature. Criteria for guiding judgement as to which disorders should be included or not included under the Act were developed. For each disorder or group of disorders suggested by TSANZ, information from the identified literature was considered in the light of the criteria and recommendations made regarding whether to include the disorder under the Act.

Findings

A disorder was considered eligible for inclusion under Schedule 1 of the Act if it met all three of the following criteria:

- It is caused by exposure to a dust (an airborne solid substance) that can result in chronic or permanent pathological damage to the lung.
- The exposure is known to occur in occupational circumstances.
- All, or the vast majority, of occupational cases of the disorder result from exposure to dust.

These criteria explicitly exclude airborne chemicals and other substances not in solid form.

Using these criteria, four additional specific disorders were recommended for inclusion - hypersensitivity pneumonitis, lung cancer due to silica exposure, diffuse dust-related pulmonary fibrosis, and systemic sclerosis (otherwise known as scleroderma). In addition, it was recommended that all pneumoconioses, not just those listed in the Schedule, be included. Suggested values for epidemiological measures associated with these additional disorders were also provided.

Claims in respect of the diseases not recommended for inclusion in Schedule 1 of the Act may be eligible for compensation under the NSW workers' compensation scheme.

1. INTRODUCTION

In New South Wales, compensation and other payments related to dust diseases are covered under the Workers' Compensation (Dust Diseases) Act 1942. Schedule 1 of the Act contains a list of diseases covered by the Act.

In 2018, the Law and Justice Committee undertook a review of the dust diseases scheme. Recommendation 6 of that review was "*That the State Insurance Regulatory Authority liaise with key stakeholders, including the Thoracic Society of Australia and New Zealand, regarding updating of the list of dust diseases contained in Schedule 1 of the Workers' Compensation (Dust Diseases) Act 1942 and commission an independent actuarial study to consider the implications of making any amendments.*"

Recommendation 6 was supported in principle by the Government, which noted: "*…that in order to consider inclusion of other occupational dust diseases not currently included in Schedule 1 of the Workers' Compensation (Dust Diseases) Act 1942, it will be necessary to undertake research into:*

- *epidemiological studies on the incidence of the dust diseases;*
- *an exposure profile of the NSW workforce over time;*
- *latency periods (i.e. The time between exposure to the hazard and onset of illness); and*
- *the severity of the diseases on its occurrence.*"

Suggestions for expansion of Schedule 1 of the Dust Diseases Act have been made by the Thoracic Society of Australia and New Zealand (TSANZ).

The State Insurance Regulatory Authority (SIRA) requested a project that addressed the above issues. The project required the investigator to provide advice on which of the disorders proposed by the Thoracic Society of Australia and New Zealand (and any other disorders not currently in Schedule 1 of the Dust Diseases Act) should be recommended to be considered a 'dust disease'.

As some disorders which may be caused by occupational exposure to dust might also be caused by non-dust-related exposures, the investigator was also required to provide recommendations regarding whether these disorders should be added to Schedule 1 of the Dust Diseases Act with appropriate exposure criteria.

The required information is presented in this report.

This report consists of six chapters:

- Chapter 1 provides a brief introduction
- Chapter 2 outlines the methods used
- Chapter 3 presents a review of disorders for possible inclusion under the Act
- Chapter 4 presents epidemiological information relevant to additional disorders recommended to be included under the Act
- Chapter 5 provides a summary of the recommendations
- Chapter 6 contains the references cited in the document.

2. METHODS

INTRODUCTION

This section summarizes the methods used in this study to identify relevant information on the conditions proposed by TSANZ (and any other relevant disorders).

INCLUDED DATABASES

Searches were primarily undertaken in PubMed. No comprehensive search was undertaken of the grey literature, but some grey literature articles identified during the search process were included where relevant.

SEARCH STRATEGY

Separate searches were undertaken for each relevant disorder, using the disorder name and variations of it and "work-related" or "occupational" or variations of these. The focus was to identify relevant systematic reviews, but single studies were included if there wasn't an appropriate systematic review and the single study was deemed to be of sufficient quality or particular relevance. The intention was not to attempt to identify all published literature relevant to a disorder, but to identify key recent articles. Relevant articles identified through previous searching were also included.

There was no specific time limit set on the searches but in practice, only articles from about 2000 onwards were included and only studies of humans. The final full searches were conducted in February and March 2020, with inclusion of some additional studies later in 2020 during the revision process.

REVIEW PROCESS

Relevant studies were identified by review of the title, abstract and, if necessary, the full text version. One person (the author) undertook all the searching and made the decisions regarding inclusion, exclusion and relevance.

DATA EXTRACTION, CRITICAL APPRAISAL AND SYNTHESIS

Critical appraisal of the relevant literature and consideration of the weight of evidence in regards to a particular disorder was undertaken. The results from the included studies were synthesized qualitatively as appropriate.

CONSIDERATION OF WHETHER TO RECOMMEND ADDING DISORDERS TO BE COVERED UNDER THE ACT

Criteria for guiding judgement as to which disorders should be included or not included under the Act were developed. For each disorder or group of disorders suggested by TSANZ, information from the identified literature was considered in the light of the criteria and recommendations made regarding whether or not to include the disorder under the Act.

3. REVIEW OF DISORDERS FOR POSSIBLE INCLUSION UNDER THE ACT

INTRODUCTION

This chapter presents a consideration of evidence regarding occupational disorders arising from dust exposure. It also considers specific disorders or groups of disorders proposed by TSANZ for inclusion. The focus is on the results of review papers, but individual papers were considered where there were few relevant papers. Disorders already included under the Act were not further considered.

DUSTS DISEASES IN THE ACT

The Act defines a 'dust disease' as "*any disease specified in Schedule 1, and includes any pathological condition of the lungs, pleura or peritoneum, that is caused by dust that may also cause a disease so specified.*" The current Schedule 1 diseases are:

- Aluminosis
- Asbestosis
- Asbestos-induced carcinoma
- Asbestos-related pleural disease
- Bagassosis
- Beryllosis
- Byssinosis
- Coal dust pneumoconiosis
- Farmer's lung
- Hard metal pneumoconiosis
- Mesothelioma
- Silicosis
- Silico-tuberculosis
- Talcosis.

THE DEFINITION OF 'DUST'

The Act defines 'dust' as "*…dust of such a nature that the inhalation thereof may give rise to a dust disease.*". The Act does not contain any further guidance as to which conditions should, or should not be covered by the Act, apart from a reference to the disorders in Schedule 1.

This definition of 'dust' is not overly helpful because it contains the word 'dust' in the definition, without defining what 'dust', as used in the definition, actually is. The Collins

Dictionary defines 'dust' as "*dry, fine powdery material, such as particles of dirt, earth or pollen*"[1]. This definition seems to meet the intention of the Act and so has been adopted for the purposes of this report. Therefore, for the purposes of this report, 'dust' is taken to mean any airborne solid substance that can result in chronic or permanent pathological damage to the lung (the pathological damage aspect is considered in the next section). It explicitly excludes airborne chemicals and other substances not in solid form.

GENERAL CHARACTERISTICS OF DISORDERS THAT SHOULD BE INCLUDED

The wording of the Act strongly suggests that the disorders that should be included are those caused by inhalation of a 'dust' that results in persisting pathology to the lung, where that inhalation is directly connected to the occupation or work activity of the affected person. The Act does not appear to be an instrument to provide compensation for all lung disorders. This is consistent with the fact that the Act arose out of an earlier instrument that focussed on silicosis. This has important implications for which disorders should be included under the Act. If the inclusion is confined to disorders caused by dust, it will necessarily exclude many important occupational respiratory disorders, such as infections and disorders resulting from airborne chemical exposure. If the inclusion is expanded to include all occupational respiratory disorders, the Act would no longer be a dust diseases act. Instead, it would be a compensation instrument for work-related respiratory disorders, and one that potentially provided different compensation rights to workers with respiratory disorders compared to workers with other types of disorders. This does not seem to be the purpose of the Act as currently worded. This interpretation was confirmed through discussions with SIRA. This is the basis for the first criterion listed in the next section.

Under the Act there does not appear to be any requirement for the disorder to result in symptoms. Indeed, Schedule 1 includes 'Asbestos-related pleural disease', which may be present without resulting in any symptoms.

DISORDERS THAT CAN BE CAUSED BY DUST AND BY NON-DUST EXPOSURES

A related issue arises because some respiratory disorders can be caused by dusts but also by other agents. Occupational asthma, chronic obstructive pulmonary disease (COPD) and lung cancer are examples. Using occupational asthma as an example, this condition can be caused by hundreds of substances. Many of these can be described as dusts of some sort; examples are hardwood dust, flour and detergent enzymes. However, occupational asthma can also be caused by exposure to chemical agents; examples include isocyanates and formaldehyde. These chemicals do not appear to meet the definition of 'dust' under the Act. This means that not all cases of occupational asthma should be included under the Act as it is currently written.

The exclusion of some cases of occupational asthma because the cause was a chemical agent not in solid form, but the inclusion of other cases caused by a solid agent, seems arbitrary. All cases of occupational asthma are covered under workers' compensation legislation and occupational asthma is included in the Deemed Diseases List developed by SafeWork Australia[2]. The exclusion under the Act arises from the focus of the Act being disorders caused by occupational exposure to dusts, rather than all respiratory disorders caused by any occupational exposure.

This leaves three main choices. One is to include some cases of occupational asthma but exclude others, depending on their cause, even if all the cases are accepted as being due to work. The second is to exclude all cases of occupational asthma under the Act and allow them to be covered through the NSW workers' compensation scheme. The third is to change the focus of the Act to cover all respiratory disease, regardless of whether it is caused by dust or not. This third option is discounted for the purposes of this report, as directed by SIRA. Including some cases of an occupational condition but excluding others, even though all arise from work, seems unreasonably arbitrary and may lead to argument as to precisely which agent is the cause of the disorder (asthma in this example), although in practice this precise agent would usually be known for occupational asthma. The second option seems the most workable and has been adopted for this report when deciding whether to recommend a disorder should be included. This is the basis for the third criterion listed in the next section.

CRITERIA TO BE USED WHEN CONSIDERING WHETHER A DISORDER SHOULD BE INCLUDED IN SCHEDULE 1 OF THE ACT.

Based on the above considerations, for the purposes of this report, a disorder will be considered eligible for inclusion under Schedule 1 of the Act if it meets all three of the following criteria:

- It is caused by exposure to a dust (an airborne solid substance) that can result in chronic or permanent pathological damage to the lung.
- The exposure is known to occur in occupational circumstances.
- All, or the vast majority, of occupational cases of the disorder result from exposure to dust.

These criteria explicitly exclude airborne chemicals and other substances not in solid form. They also explicitly exclude exposures that only occur in circumstances not related to occupation or work activity. The first two criteria are clearly consistent with Recommendation 6 of the 2018 Law and Justice Committee review of the dust diseases scheme – "*…regarding updating of the list of dust diseases contained in Schedule 1 of the Workers' Compensation (Dust Diseases) Act 1942…*". The third criterion does not

explicitly arise out of this recommendation because, as mentioned, there are some disorders which can be caused by dust and also by other, non-dust, exposures. The former fall under the scope of the Act and the latter do not. Inconsistencies will arise whether the disorders are included or excluded from Schedule 1. The recommended approach is designed to minimise this inconsistency – if most of the cases are likely to be due to dust, it seems sensible to have the disorder covered by the Act; if most cases are not due to exposure to dust, it would be more consistent and workable to exclude the disorder under the Act and allow it to be covered through the NSW workers' compensation scheme.

ADDITIONAL DISORDERS SUGGESTED BY TSANZ TO BE INCLUDED UNDER THE ACT

The Thoracic Society of Australia and New Zealand has suggested retaining all the disorders currently included in Schedule 1 and to include some additional disorders. They also proposed classifying disorders into relevant groups (e.g., all the pneumoconisoses in a single group, for example). Their specific suggestions were:

- Pneumoconiosis including coal workers' pneumoconiosis (CWP) and silicosis
- Occupational and work-related asthma
- Occupational hypersensitivity pneumonitis
- Occupational COPD including that due to coal mine dusts and silica dust
- Occupational respiratory infections including tuberculosis and transmitted respiratory infections such as SARS, MERS, avian influenza and "swine flu" in health care workers, Brucellosis in veterinarians, and pneumococcal pneumonia in welders
- Toxic inhalational injuries and resulting long term lung disorders
- Occupational lung cancers including those related to arsenic, bischloromethyl ether, beryllium, cadmium, nickel, chromium IV, chloromethyl ethers, vinyl chloride, radon, and mustard gas
- Diffuse dust-related pulmonary fibrosis
- Consideration should also be given to systemic disorders associated with occupational exposures e.g., connective tissue and renal disorders related to silica exposure.".

The disorders and approaches suggested by TSANZ are explicitly considered in the remainder of this chapter, using the inclusion criteria above to guide this consideration. For each disorder or group of disorders, a recommendation is made regarding whether the disorder should be included under the Act or instead be dealt with through the NSW workers' compensation scheme.

PNEUMOCONIOSIS INCLUDING COAL WORKERS' PNEUMOCONIOSIS AND SILICOSIS

Pneumoconioses are interstitial diseases of the lung which by definition are caused by exposure to dust. They are characterised by a fibrotic interstitial lung disease and are chronic conditions of the lung that can result in symptoms[3]. Sufficient exposure to result in pneumoconiosis almost certainly only occurs in occupational exposure circumstances. There have been a very small number of reports of silicosis occurring in non-occupational environments, such as those with high exposure to wind-blown sand, but the evidence is unclear and the circumstances do not seem relevant to the Australian environment[4].

Pneumoconioses are pathological disorders caused by exposure to a dust. This exposure is known to occur in occupational circumstances. All occupational cases of the disorder result from exposure to dust. Therefore, it is appropriate that all pneumoconioses are accepted as dust diseases for the purposes of the Act.

The Act already includes several named pneumoconioses – aluminosis, asbestosis, beryllosis, coal dust pneumoconiosis (also known as coal workers' pneumoconiosis), hard metal pneumoconiosis, silicosis and talcosis. There are also many more uncommon but relevant pneumoconioses that are not currently named under the Act (e.g., stannosis, caused by exposure to tin dust; siderosis, caused by exposure to iron). Grouping all these under the one name ('Pneumoconiosis'), as suggested by TSANZ, seems sensible, as it allows the inclusion of all known pneumoconioses without having to separately name each of them. TSANZ suggested including "coal workers' pneumoconiosis" and "silicosis" in the name of the group. The vast majority of cases of pneumoconiosis are of one of three disorders – silicosis, asbestosis and CWP. Silicosis was the disorder that was the focus of the legislation that was the predecessor to the current Act. There might be merit in retaining this focus and keeping an explicit connection to these important occupational conditions, by calling the group '*Pneumoconioses, including asbestosis, coal workers' pneumoconiosis and silicosis*'. However, strictly speaking it is not necessary to explicitly include these specific disorders in the group name because they are all pneumoconioses.

Recommendation: Include all pneumoconioses in Schedule 1 of the Act under the group name "Pneumoconioses, including asbestosis, coal workers' pneumoconiosis and silicosis".

OCCUPATIONAL AND WORK-RELATED ASTHMA

Occupational asthma by definition is asthma caused by occupational exposure. Most researchers and practitioners distinguish between incident asthma caused by occupational exposures (this is usually called 'occupational asthma') and the exacerbation

of pre-existing asthma by occupational exposures (this is typically called 'work-exacerbated asthma')[5]. However, the literature is inconsistent with this terminology. Occupational asthma may be cured if it is identified early enough and exposure to the causative substance avoided. On-going exposure is likely to result in a chronic condition. Exacerbation of pre-existing asthma by exposure to workplace irritants (work-exacerbated asthma) may produce a similar level of severity and adverse socioeconomic effects to occupational asthma [6] and may result in more significant adverse effects than asthma unrelated to work[6], but the evidence about this is not definitive[7, 8].

Occupational asthma can be caused by exposure to dust. This exposure is known to occur in occupational circumstances. The resulting pathology can be permanent. On this basis, occupational asthma caused by exposure to dust could reasonably be accepted as a dust disease for the purposes of the Act. However, many cases of occupational asthma arise from exposures that are not considered dusts. Therefore, occupational asthma does not meet the third criterion for inclusion and should not be included under the Act. Instead, claims in respect of occupational asthma may be eligible for compensation under the NSW workers' compensation scheme.

Recommendation: Do not include occupational asthma in Schedule 1 of the Act.

OCCUPATIONAL HYPERSENSITIVITY PNEUMONITIS

Hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis) is a disorder of the small airways of the lung caused by an immune reaction resulting from repeated contact with small animal or vegetable dust particles. There are many occupational exposures that can result in hypersensitivity pneumonitis. Common causes of the disorder are mouldy hay, straw, grain and feathers. Most of these exposures meet the definition of dust, but some cases may occur due to exposure to non-dust agents[3]. The disorder can also occur from non-occupational exposures. There are well-documented connections to particular occupations[9-19].

Hypersensitivity pneumonitis can be caused by exposure to dust. This exposure is known to occur in occupational circumstances. The resulting pathology can be permanent. Organic dusts probably cause the vast majority of cases of hypersensitivity pneumonitis. Therefore, it is appropriate that hypersensitivity pneumonitis is accepted as a dust disease for the purposes of the Act.

The Act already includes "Farmer's lung" and "Bagassosis", which are forms of hypersensitivity pneumonitis. Hypersensitivity pneumonitis has some commonalities with occupational asthma. Both are due (or in the case of occupational asthma, primarily

due) to immune-mediated mechanisms resulting from respiratory exposures. Therefore, proposing including hypersensitivity pneumonitis but excluding occupational asthma raises some inconsistencies. However, nearly all cases of hypersensitivity pneumonitis should meet the requirements of the Act, as nearly all are likely to arise from exposure to dust, whereas that is not the case with occupational asthma. In addition, some forms of hypersensitivity pneumonitis are already included under the Act and these disorders cannot be removed from Schedule 1.

Specifying all the agents that can result in hypersensitivity pneumonitis is challenging. The recent Comcare instrument included, and specified, only four exposures – mouldy feathers, mouldy grain, mouldy hay and mouldy straw. Other dusts might also result in hypersensitivity pneumonitis, suggesting it would be better not to specify which exposure was responsible. However, this would allow persons with hypersensitivity pneumonitis due to a non-dust exposure to be covered by the Act, which presumably is not a desired outcome. There is no easy solution to this issue. It is recommended that hypersensitivity pneumonitis be included as a category, with a specification in the Schedule 1 that the condition has to be due to exposure to dust. The medical panel could appropriately determine this. Claims in respect of non-dust causes of hypersensitivity pneumonitis may be eligible for compensation under the NSW workers' compensation scheme.

Recommendation: Include hypersensitivity pneumonitis in Schedule 1 of the Act, with 'Farmer's lung' included under this category, and specify that the condition has to be due to exposure to dust.

OCCUPATIONAL COPD INCLUDING THAT DUE TO COAL MINE DUSTS AND SILICA DUST

Chronic obstructive pulmonary disease is a chronic disease of the lung characterised by a breakdown in the normal structure of the airways and gas-exchange areas of the lung[20]. Smoking is the main cause but various occupational exposures, including dusts, can cause COPD. It can also occur as part of the end stage of pneumoconiosis. As with occupational asthma, there are many occupational causes of COPD. These are often grouped together as "vapours, gases, dusts and fumes"[7].

Chronic obstructive pulmonary disease can be caused by exposure to dust. This exposure is known to occur in occupational circumstances. The resulting pathology can be permanent. On that basis, COPD caused by exposure to dust could reasonably be accepted as a dust disease for the purposes of the Act. However, many cases of COPD associated with occupation arise from exposures that are not considered dusts. Therefore, COPD does not meet the third criterion for inclusion and should not be

included under the Act. Instead, claims in respect of COPD may be eligible for compensation under the NSW workers' compensation scheme. This approach would not exclude COPD associated with pneumoconiosis, because the presence of pneumoconiosis would mean the case was covered under the Act. However, it would exclude COPD due to coal mine dust exposure in the absence of pneumoconiosis[21].

Recommendation: Do not include COPD in Schedule 1 of the Act.

OCCUPATIONAL RESPIRATORY INFECTIONS

Occupational respiratory infections were one of the groups of disorders proposed by TSANZ. Schedule 1 of the Act does currently include one infection –tuberculosis. However, this is only tuberculosis in association with silicosis and so would be included under the Act regardless. The infections proposed by TSANZ can occur in occupational circumstances. However, they are not (or are commonly not) caused by exposure to dusts. Therefore, respiratory infections do not meet the first criterion and should not be included under the Act. Instead, claims in respect of respiratory infections may be eligible for compensation under the NSW workers' compensation scheme.

Recommendation: Do not include respiratory infections in Schedule 1 of the Act.

TOXIC INHALATIONAL INJURIES AND RESULTING LONG-TERM LUNG DISORDERS

Toxic inhalational injuries resulting in long-term lung disorders can occur in the occupational setting. They can be caused by breathing in particles or substances that cause acute chemical or thermal damage to lung tissue due to the chemical composition, reactivity or heat of the agent.

Toxic inhalational injuries can be caused by exposure to dust. This exposure is known to occur in occupational circumstances. The resulting pathology can be permanent. On that basis, toxic inhalational injuries could be considered for inclusion as a dust disease for the purposes of the Act. However, many (probably most) cases of toxic inhalational injury associated with occupation arise from exposures that are not considered dusts. Therefore, toxic inhalational injury does not meet the third criterion for inclusion and should not be included under the Act. Instead, claims in respect of toxic inhalational injury may be eligible for compensation under the NSW workers' compensation scheme.

Recommendation: Do not include toxic inhalational injury in Schedule 1 of the Act.

OCCUPATIONAL LUNG CANCER

The TSANZ list of disorders to include under the Act included "*Occupational lung cancers including those related to arsenic, bischloromethyl ether, beryllium, cadmium, nickel, chromium IV, chloromethyl ethers, vinyl chloride, radon, and mustard gas*". There are other agents that occur in occupational circumstances and that are known to increase the risk of lung cancer. It is not clear why only some of these agents were specified. It is assumed here that the TSANZ proposal is to include all agents that can occur in occupational circumstances and that are known to increase the risk of developing lung cancer. Smoking is the main cause of lung cancer.

Lung cancer can be caused by exposure to dust. In fact, asbestos causes the great majority of cases of occupational lung cancer and silica is probably the next most common cause. These exposures are known to occur in occupational circumstances. The resulting pathology can be permanent. On that basis, lung cancer caused by exposure to dust could reasonably be accepted as a dust disease for the purposes of the Act. However, many cases of lung cancer associated with occupation arise from exposures that are not considered dusts. Therefore, lung cancer as a whole does not meet the third criterion for inclusion and should not be included under the Act. Instead, it should be covered through the NSW workers' compensation scheme.

Schedule 1 of the Act already includes cancer associated with one dust – asbestos ("asbestos-induced carcinoma") – but does not include any other lung cancer. (Note that malignant mesothelioma is included in Schedule 1 of the Act but is not a form of lung cancer.)

The decision to exclude lung cancer raises some concerns and potential inconsistency. Schedule 1 of the Act already includes two entries related to cancer, both related to asbestos - "asbestos-induced carcinoma" and "mesothelioma". In addition to mesothelioma, asbestos is known to increase the risk of lung cancer, laryngeal cancer and ovarian cancer. It is not intended that any disorders currently covered by the Act would be excluded. Therefore, some cases of lung cancer will be covered by the Act regardless of any other recommendations made here. Exposure to silica can also increase the risk of lung cancer and there does not seem any justification for including lung cancer associated with asbestos and not lung cancer associated with silica. This is especially so given that the antecedent of the Act was specifically established to cover silica-related disease. Some other occupational causes of lung cancer are also dusts. Including asbestos-related lung cancer (and possibly silica-related cancer) but not lung cancer caused by exposure to other dusts appears inconsistent, given the Act explicitly covers dust diseases. There would also be inconsistency if only lung cancer caused by dusts were included and other occupational-related lung cancers were excluded. This

approach would also be inconsistent with the approach proposed for occupational asthma and COPD. Whatever recommendation is made, there will be inconsistency.

Since asbestos-related lung cancer is included under the Act, it seems appropriate to include silica-related lung cancer and this is what is recommended. Claims in respect of other occupational causes of lung cancer may be eligible for compensation under the NSW workers' compensation scheme.

The TSANZ list only covered lung cancer but the Act actually covers non-respiratory cancers, given that ovarian cancer is included (since it is an "asbestos-induced carcinoma") and mesothelioma occurs in other areas of the body in addition to the chest. It might therefore be argued that other cancers caused by dusts should be considered for inclusion under the Act. Silica is not known to cause cancers other than lung cancer, and no dusts apart from asbestos and silica are proposed to be included under the Act in terms of cancer. Therefore, no other cancers apart from silica-related lung cancer are proposed for additional inclusion under the Act.

Recommendation: Include silica-related lung cancer in Schedule 1 of the Act. Do not include lung cancer caused by other exposures (apart from asbestos-related lung cancer, which is already included under the Act).

DIFFUSE DUST-RELATED PULMONARY FIBROSIS

Diffuse dust-related pulmonary fibrosis is a fibrotic condition of the lung parenchyma caused by exposure to coal mine dust. It is distinct from CWP and almost certainly only occurs due to occupational exposures[22-24].

Diffuse dust-related pulmonary fibrosis is a pathological disorder caused by exposure to a dust. This exposure is known to occur in occupational circumstances. All occupational cases of the disorder result from exposure to (coal mine) dust. Therefore, it is appropriate that diffuse dust-related pulmonary fibrosis is accepted as a dust disease for the purposes of the Act.

Recommendation: Include diffuse dust-related pulmonary fibrosis in Schedule 1 of the Act.

SYSTEMIC DISORDERS ASSOCIATED WITH OCCUPATIONAL EXPOSURES

The list proposed by TSANZ included a category for "*systemic disorders associated with occupational exposures*". Connective tissue and renal disorders related to silica exposure were specified as examples of such disorders. There are several systemic disorders that might be relevant when considering this proposal. For example, exposure to silica has been associated with several disorders, most notably systemic sclerosis (otherwise known as scleroderma) and chronic renal failure.

Two recent systematic reviews and meta-analyses that examined the relationship between occupational silica exposure and the occurrence of systemic sclerosis found a raised risk of systemic sclerosis in persons occupationally exposed to silica[25, 26]. Several potentially relevant papers have been published since then, including a recent brief Australian case series[27], but none add materially to the systematic reviews described earlier[28-36]. Earlier narrative reviews provide some evidence supportive of a causal connection between occupational silica exposure and the occurrence of systemic sclerosis[37-40] but also do not provide evidence over and above the more recent reviews. The two systematic reviews provide moderate evidence that occupational silica exposure does increase the risk of developing systemic sclerosis. However, the methodological limitations of the original studies and the inability to examine for evidence of a dose-response relationship means that bias, confounding and chance can't be excluded with confidence. Also of note, systemic sclerosis may include interstitial lung disease. This was found to be present in about one third of people with systemic sclerosis in a recent French population study[41], but such interstitial lung changes may be much more common if patients who do not have respiratory symptoms are included, based on CT scanning and autopsy findings{Suliman, 2017 #1173}. Such patients would meet the criterion for inclusion related to the need for lung pathology ("*…caused by exposure to a dust … that can result in chronic or permanent pathological damage to the lung*"). However, instances of systemic sclerosis that do not involve interstitial lung disease would not meet this criterion.

One recent review considered the relationship between exposure to silica and the risk of chronic kidney disease[42]. A meta-analysis identified an increased risk of chronic renal disease with silica exposure in a variety of occupational exposure circumstances. However, there was inconsistent evidence of an exposure-response relationship. The authors also identified a number of methodological problems with the included studies.

Several other diseases, mainly auto-immune disorders, have been associated with dust exposure, particularly silica exposure, including recently in silicosis patients exposed to artificial stone[27, 43]. In addition to systemic sclerosis (considered earlier), these disorders include rheumatoid arthritis[44, 45], systemic lupus erythematosus[46-48], glomerulonephritis

(which may explain the reported associated between silica and chronic renal disease) and anti-neutrophil cytoplasmic antibody (ANCA)-related diseases[46, 49-51]. Plausible immune mechanisms have been postulated for these possible relationships. However, the epidemiological evidence for a causal role of dust exposure is inconsistent.

In 2018, the United Kingdom Industrial Advisory Council reviewed the published literature with a view to deciding whether the connection between occupational exposure to silica and the occurrence of connective tissue diseases should be prescribed for compensation purposes. This was a similar purpose to that of the current report (albeit only focussed on one dust). The Council decided against prescription at that time, although it should be noted that their requirements were more restrictive than is the case for the current report. They concluded "*Collectively this provides reasonable evidence pointing to an occupational hazard, the evidence generally being deeper for systemic sclerosis/scleroderma than for the other two conditions [systemic lupus erythematosus and rheumatoid arthritis]. Prescription is hampered, however, by the difficulty of defining the qualifying levels of occupational exposure.*" The Council also considered requiring the presence of silicosis to determine eligibility for prescription (and thus compensation), but concluded "…*unresolved methodological concerns about the few available reports of this kind have proved to be a stumbling block.*"[52].

The evidence that silica exposure results in systemic sclerosis is moderately strong but not definitive. The evidence for silica and other dusts in regard to chronic renal failure and other systemic disorders is weaker. This means these systemic disorders do not definitely meet the first criterion for inclusion under the Act. However, the evidence is reasonably strong for systemic sclerosis. Also, systemic sclerosis is rare, meaning that it would be expected that there would not be many claims arising for this condition. Therefore, on balance, it is recommended that systemic sclerosis is included under the Act, but only if it features interstitial lung disease. Other systemic disorders linked with dust exposure, such as chronic renal failure, rheumatoid arthritis, systemic lupus erythematosus and glomerulonephritis, are not recommended for inclusion. Instead, claims in respect of these disorders may be eligible for compensation under the NSW workers' compensation scheme.

Recommendation: Include systemic sclerosis (otherwise known as scleroderma) involving interstitial lung disease associated with exposure to silica in Schedule 1 of the Act. Do not include systemic sclerosis not involving interstitial lung disease or other systemic disorders possibly related to exposure to dust.

OTHER DISORDERS THAT COULD BE CONSIDERED FOR INCLUSION UNDER THE ACT

There are no relevant additional disorders related to dust exposure that are not already included under the Act or proposed above for inclusion.

4. EPIDEMIOLOGICAL INFORMATION RELEVANT TO ADDITIONAL DISORDERS RECOMMENDED TO BE INCLUDED UNDER THE ACT

INTRODUCTION

This chapter provides requested epidemiological information on disorders suggested to be included under the Act. All pneumoconioses, regardless of whether they are currently listed in Schedule 1, were recommended to be included. Only four additional disorders were recommended for inclusion - hypersensitivity pneumonitis, lung cancer due to silica exposure, diffuse dust-related pulmonary fibrosis, and systemic sclerosis.

RELATIVE RISKS

The RRs for all pneumoconioses are essentially infinite (or undefined) because essentially all cases of pneumoconiosis are considered to be due to work-related exposure to dust.

There is no information available on the relative risk of hypersensitivity pneumonitis or diffuse dust-related pulmonary fibrosis.

Information on the relative risk of lung cancer arising from silica exposure is available. The proposed RRs for lung arising from silica exposure come from a study by Liu and co-workers[53]. This is considered the most appropriate study because it provides detailed information on cumulative silica exposure in a large cohort with good control of smoking. The RR for high exposure was 1.70 (95% confidence interval (95% CI) 1.23-2.34) and for lower exposure was 1.54 (95% CI 1.16-2.05), compared to unexposed workers.

The RR proposed for systemic sclerosis comes from the most recent systematic review[26]. Separate RRs were provided based on case control studies and on cohort studies. The RR for case control studies is considered the more appropriate because it is based on many more studies and is more precise than the RR for cohort studies, which was much higher but had a very wide confidence interval (RR=17.5 (95% CI 6.0–51.4)). The proposed RR is 2.81 (95% CI 1.86-4.23) in silica-exposed workers compared to unexposed workers.

INCIDENCE AND INCIDENCE RATES

There is no information on the incidence of hypersensitivity pneumonitis or diffuse dust-related pulmonary fibrosis in Australia. One review paper[15] reported estimates for the prevalence of farmer's lung that ranged from 1% to 19% of exposed farmers, and

prevalence estimates for pigeon breeder's lung ranging from 6% to 20% of exposed individuals, but the proportion of persons considered 'exposed' is difficult to estimate.

There is no direct information on the incidence of lung cancer arising from occupational silica exposure in Australia. The Global Burden of Disease study provides estimates on a global, regional and national basis, but for Australia does not provide estimates at a State or Territory level[54]. By combining information from several sources, it is possible to estimate the incidence of lung cancer arising from occupational silica exposure in NSW. This requires an estimate of the number of incident cases (or deaths) of lung cancer and an estimate of the proportion of these deaths that was due to occupational exposure to silica. This proportion, known as the Population Attributable Fraction (PAF), can be calculated using estimates of the relative risk of developing lung cancer from silica exposure (this relative risk is the risk of developing lung cancer in persons occupationally exposed to silica compared to the risk of developing lung cancer in unexposed persons); and estimates of the proportion of the population which is occupationally exposed to silica.

Information on cancer incidence is available from the Australian Institute of Health and Welfare's Australian Cancer Incidence and Mortality (ACIM) books. Information is available on incidence and mortality (both frequency and population-based rates) for most individual cancers and for all cancers combined. Information is available separately by age and sex and for all years from 1982 to 2015, inclusive. Separate information is available by State and Territory; age-specific information is not available by State and Territory but age-standardised rates are available[55]. Based on the AIHW data, in 2014 (the most recent year for which NSW incidence data are available) in NSW there were 3,736 cases of lung cancer - 2,132 in males and 1, 604 in females. In terms of deaths, in NSW in 2014 there were 2,719 deaths from lung cancer, 1,579 of males and 1,122 of females.

The most recent information from the Global Burden of Disease study provides an estimate of the PAF for occupational exposure for lung cancers arising from silica exposure[54]. Separate estimates of PAF are available based on deaths and on Disability-Adjusted Life Years (DALYs)[56, 57], using the GBD Compare site[56]. The PAFs based on deaths are used in the presented analysis. The data are available globally but also separately for Australia. (The AIHW provides PAF estimates for lung cancer from occupational exposures for Australia for 2011, but not separately for silica and only based on DALYs[58]). For Australia, the PAF was estimated to be 2.7% (95% Uncertainty Interval (95% UI) 0.7-4.8%) for males and 1.8% (95% UI 0.4-3.0%) for females. (For comparison, the corresponding PAFs based on DALYs were 3.5% (0.9-6.0%) for males and 2.3% (0.5-3.9%) for females.)

Using these data, it is estimated that in NSW in 2014 there were 58 cases of lung cancer in males, and 29 cases of lung cancer in females, due to silica exposure. Similarly, it is estimated that in 2014 there were 43 deaths of males, and 20 deaths of females, from lung cancer due to silica exposure (Table 1).

Table 1 Population-attributable fraction for lung cancer due to occupational exposure to silica – deaths and DALYs; estimated silica-related lung cancer cases and deaths – Australia, 2014

Cancer type	Male	Female	Total
PAF (deaths)+	2.7	1.8	2.3
Cases	58	29	87
Deaths	43	20	63

+: Global Burden of Disease study, based on IHME, 2019[54, 56].

As mentioned, the GBD study provides estimates of incidence and deaths from pneumoconiosis for Australia, but not by State and Territory. These estimates include all pneumoconiosis, but the only specific pneumoconiosis for which there is cause-specific data are asbestosis, CWP and silicosis. In Australia, and therefore in NSW, there are very few cases due to any other pneumoconiosis. Estimates for incident cases for Australia for 2017, the most recent year for which information is available, were 211 (95% UI 185-238) in males, 6 (95% UI 4-8) in females and 217 (95% UI 190-244) overall. The corresponding estimates for deaths were 150 (95% UI 122-182) in males, 5 (95% UI 2-8) in females and 155 (95% UI 128-187) overall. However, only two (95% UI zero to six) new cases and two (95% UI one to two) deaths were estimated for pneumoconioses other than asbestosis, CWP and silicosis.

There is no direct information on the incidence of systemic sclerosis arising from silica exposure in Australia. However, the number is likely to be very low as these disorders are not common and the incidence in the general population appears to be low[59].

EXPOSURE PREVALENCE

There is no useable exposure information relevant to the occurrence of hypersensitivity pneumonitis.

Information on the prevalence of exposure of Australian workers to occupational carcinogens is available from the Australian Work Exposures Study (AWES), which obtained data from 2011 and 2012. The study was a cross-sectional telephone survey that looked at the prevalence of current occupational exposure to 38 known or probable priority carcinogens among Australian workers. The study found that about 2.7 million

men (58%) and 880,000 women (21%) appeared to be exposed to at least one of the priority carcinogens[60]. (Separate results just for New South Wales were not published.)

The AWES results estimated that 11.6% (95% CI 10.5-12.9%) of males in the workforce and 1.0% (0.7-1.5%) of females in the workforce are exposed to silica[60]. Note that carcinogens have a prolonged latency period and that people exposed to a carcinogen remain at risk of developing a resultant cancer for many years, usually decades, afterwards. Therefore, the proportion of people currently exposed is likely to be considerably less than the proportion of people who are at risk at any one time. The relevant proportion at risk will vary by cancer and age, but a rough estimate would be of the order of two and half to four times the current prevalence of exposure.

The AWES does not provide information about the prevalence of exposure to dusts that cause any of the pneumoconioses not currently covered by the Act.

DURATION OF EXPOSURE AND LATENCY

For many conditions included under the Act, there is a period of time (the latency) measured in months or years between first exposure and clinical occurrence of the resulting disorder. There is also likely to be a minimum exposure ('sufficient exposure') below which the disorder would not occur or is very unlikely to occur. The relevant measure of this exposure is usually the total exposure (cumulative exposure) rather than just a length of exposure or a level (concentration or intensity) of exposure. That is, a higher exposure (in terms of the concentration in air) for a shorter time is assumed to have a similar risk to a lower exposure for a longer time. This may not be strictly true for all disorders, but it should hold for most disorders most of the time.

Unfortunately, for most exposure-disorder pairs, the latency and minimum exposure are not well characterized. In addition, the level (concentration) of the relevant exposure is unlikely to be known with any accuracy for an individual worker. Therefore, any judgments on this can really only be based on a qualitative assessment.

In the absence of definitive information on required cumulative exposure and the likely absence of useful workplace exposure data to establish the cumulative exposure of an individual worker, the appropriate approach appears to be to recommend a minimum exposure time. This assumes that typical workers with exposure to a particular hazardous substance have similar levels of exposure, which means that if they are exposed for a similar length of time, they will have a similar cumulative exposure and thus a similar risk of developing the disease related to the exposure. This is the rationale for proposing a minimum exposure period rather than proposing a minimum cumulative exposure.

The minimum latencies and exposures to adopt depend on the degree of sensitivity (i.e., including all claims that do arise from occupational exposures and therefore that should be compensated) and specificity (excluding all claims that do not arise from occupational exposures and therefore that should not be compensated). Inevitably, the approach adopted must be a balance between the two, keeping in mind that individual cases that are deemed not to fall under the Act could still be the subject of a claim using the usual compensation methods.

The recommendations below have been developed with an assumption that the approach to be adopted for the Act should not be based on the minimum possible latency or exposure, but on latencies and exposures developed so there should be little question that the disease of interest could have developed as a result of the occupational exposures in question. Different recommendations would be made if the minimum possible latency (and minimum exposure periods) were to be adopted.

With the above provisos, recommendations on sufficient exposure are made for pneumoconioses not currently included under the Act, hypersensitivity pneumonitis, lung cancer arising from silica exposure, diffuse dust-related pulmonary fibrosis and systemic sclerosis. A shorter, intense, exposure might still result in the development of the condition, but there would be considerable uncertainty about this.

There is insufficient published information on the individual pneumoconioses suggested to be additionally covered under the Act to provide specific estimates of minimum latency and minimum exposure period for the terms of the Act. Guidance on these can be provided by information on asbestosis, CWP and silicosis. For most situations, the relevant latency and exposure period should be in terms of years – five years was recommended for the Comcare legislation. However, information that has become available in the last few years has shown that very high exposures to silica, which were thought to be historical and no longer an issue in Australia, have in fact occurred in the artificial stone product manufacturing industry. A recent Australian study found a median duration of exposure of seven years, with a range of four to 10 years[61]. It is very unlikely that those types of exposure would occur with other dusts that result in the rare pneumoconioses not currently explicitly listed in Schedule 1. Therefore, it would be reasonable to accept a minimum exposure period of five years and a minimum latency period of five years. Note that if an exposure period and latency for silicosis was required, it might need to be shorter than this, given the recent experience of rapid onset disease in the context of relatively short-term, very high exposures[61, 62].

Hypersensitivity pneumonitis is an immune-mediated disorder that arises due to sensitization to one or more organic agents in the workplace[15]. This is similar to most cases of occupational asthma. The required contact might only be for a short time before the sensitisation develops. Therefore, it would be reasonable to accept a minimum exposure period and latency of weeks. It is recommended that the minimum exposure period is one month, and the minimum latency period is one month.

For cancer, there is only a small amount of published information available on minimum latency periods. This issue has been considered in detail for the World Trade Centre Health Program, which required estimates of minimum latency in regards to providing appropriate health cover to people exposed to any of a variety of hazards following the World Trade Centre attack in 2001. The Administrator of the Program adopted minimum latencies of 11 years for mesothelioma, four years for most other solid cancers, 2.5 years for cancer of the thyroid and 0.4 years for lymphoproliferative and hematopoietic cancers[63]. These were deliberately very generous assessments, with the minimum period identified by any relevant publication adopted, and are too sensitive for the purposes of the Act. Median latencies for most of the cancers would be much longer than the periods used for the World Trade Centre Health Program, in the order of 20 to 30 years for mesothelioma, 15 to 20 years for other solid cancers, and at least one to two years for lymphoproliferative and hematopoietic cancers. However, using median latency is considered too conservative an approach to the identification of an appropriate minimum latency for lung cancer under the Act.

The minimum latency for lung cancer caused by silica exposure is recommended to be 15 years from first exposure. This recommendation is based on some limited useful information available about latency for lung cancer – the use of a latency of 15 years showed a good fit to the data in several studies of silica and lung cancer[64], although the best fit in another study was 25 years[53]; 27% of cases exposed to asbestos had a latency of less than 20 years[65]; and 14% of cases exposed to chromium had a latency of less than 20 years[66].

The minimum exposure period for lung cancer from silica exposure is recommended to be five years. There is no consistent evidence that provides strong guidance regarding required exposure. A large pooled analysis of silica-exposed workers found about a 20% increase in risk for workers exposed for less than 10 years (compared to never exposed workers)[67];and a similar study of asbestos-exposed workers found a 16% increase in risk for workers exposed for less than 10 years (compared to never exposed workers)[68].

There is insufficient published information on diffuse dust-related pulmonary fibrosis to provide a specific estimate of minimum latency and minimum exposure period for the

terms of the Act. Guidance on these can be provided by information on asbestosis, CWP and silicosis, as above. Therefore, it would be reasonable to accept a minimum exposure period of five years and a latency period of five years.

The systemic sclerosis arising from exposure to silica is thought to have an immune-mediated basis[49, 50]. Whether risk is based on cumulative exposure, length of exposure or intensity of exposure, or some combination of these, is not known definitively, but risk does appear to increase with increasing cumulative exposure. The length of time required for a meaningful increase in risk to be present is also not clear but is likely to be years. There is insufficient published information on systemic sclerosis related to silica exposure to provide a specific estimate of minimum latency and minimum exposure period for the terms of the Act. However, a recent Israeli case series of 40 workers with severe silicosis, who had worked with artificial stone and apparently been exposed to very high levels of crystalline silica, identified nine workers apparently with autoimmune disorders, all of whom had at least six years of exposure (range 6 to 26 years) by the time of presentation[69]. Based on this information, if an exposure period and latency period is required, it is suggested to accept a minimum exposure period of five years and a minimum latency period of five years, noting that this is based on information from workers likely to have had very high exposure intensities and high cumulative exposures, which means the latency might be expected to have been shorter than would be the case for workers with much lower exposures.

5. RECOMMENDATIONS

Include all pneumoconioses in Schedule 1 of the Act under the group name "Pneumoconioses, including asbestosis, coal workers' pneumoconiosis and silicosis".

Include hypersensitivity pneumonitis in Schedule 1 of the Act, with 'Farmer's lung' included under this category, and specify that the condition has to be due to exposure to dust.

Include silica-related lung cancer in Schedule 1 of the Act. Do not include lung cancer caused by other exposures (apart from asbestos-related lung cancer, which is already included under the Act).

Include diffuse dust-related pulmonary fibrosis in Schedule 1 of the Act.

Include systemic sclerosis (otherwise known as scleroderma) involving interstitial lung disease associated with exposure to silica in Schedule 1 of the Act. Do not include systemic sclerosis not involving interstitial lung disease or other systemic disorders possibly related to exposure to dust.

Do not include occupational asthma in Schedule 1 of the Act.

Do not include COPD in Schedule 1 of the Act.

Do not include respiratory infections in Schedule 1 of the Act.

Do not include toxic inhalational injury in Schedule 1 of the Act.

Claims in respect of the diseases not recommended for inclusion in Schedule 1 of the Act may be eligible for compensation under the NSW workers' compensation scheme.

6. REFERENCES

1. Hanks P, ed. *Collins dictionary of the English language*. 1981, Collins: Sydney.
2. Driscoll T. *Deemed Diseases in Australia*. 2015, Safe Work Australia: Canberra.
3. Hoy RF, Brims F. Occupational lung diseases in Australia. *Medical Journal of Australia*, 2017;**207**(10):443-448.
4. Bhagia LJ. Non-occupational exposure to silica dust. *Indian Journal of Occupational and Environmental Medicine*, 2012;**16**(3):95-100.
5. Hoy R, Burdon J, Chen L et al. Work-related asthma: A position paper from the Thoracic Society of Australia and New Zealand and the National Asthma Council Australia. *Respirology*, 2020;**25**(11):1183-1192.
6. Henneberger PK, Redlich CA, Callahan DB et al. An official american thoracic society statement: work-exacerbated asthma. *American Journal of Respiratory and Critical Care Medicine*, 2011;**184**(3):368-78.
7. Blanc PD, Annesi-Maesano I, Balmes JR et al. The Occupational Burden of Nonmalignant Respiratory Diseases. An Official American Thoracic Society and European Respiratory Society Statement. *American Journal of Respiratory and Critical Care Medicine*, 2019;**199**(11):1312-1334.
8. Lau A, Tarlo SM. Update on the Management of Occupational Asthma and Work-Exacerbated Asthma. *Allergy Asthma Immunology Research*, 2019;**11**(2):188-200.
9. Barber CM, Burton CM, Scaife H et al. Systematic review of respiratory case definitions in metalworking fluid outbreaks. *Occupational Medicine*, 2012;**62**(5):337-342.
10. Burton C, Crook B, Scaife H et al. Systematic review of respiratory outbreaks associated with exposure to water-based metalworking fluids. *Annals of Occupational Hygiene*, 2012;**56**(4):374-388.
11. Cartier A, Sastre J. Clinical assessment of occupational asthma and its differential diagnosis. *Immunology & Allergy Clinics of North America*, 2011;**31**(4):717-728.
12. Fishwick D. New occupational and environmental causes of asthma and extrinsic allergic alveolitis. *Clinics in Chest Medicine*, 2012;**33**(4):605-616.
13. Girard M, Cormier Y. Hypersensitivity pneumonitis. *Current Opinion in Allergy & Clinical Immunology*, 2010;**10**(2):99-103.
14. Girard M, Lacasse Y, Cormier Y. Hypersensitivity pneumonitis. *Allergy*, 2009;**64**(3):322-334.
15. Ohshimo S, Bonella F, Guzman J et al. Hypersensitivity pneumonitis. *Immunology & Allergy Clinics of North America*, 2012;**32**(4):537-556.
16. Rosenman KD. Asthma, hypersensitivity pneumonitis and other respiratory diseases caused by metalworking fluids. *Current Opinion in Allergy & Clinical Immunology*, 2009;**9**(2):97-102.
17. Sabin BR, Grammer LC. Chapter 17: Occupational immunologic lung disease. *Allergy & Asthma Proceedings*, 2012;**33**(Suppl 1):S58-60.
18. Vasakova M, Selman M, Morell F et al. Hypersensitivity Pneumonitis: Current Concepts of Pathogenesis and Potential Targets for Treatment. *American Journal of Respiratory and Critical Care Medicine*, 2019;**200**(3):301-308.
19. Zacharisen MC, Fink JN. Hypersensitivity pneumonitis and related conditions in the work environment. *Immunology & Allergy Clinics of North America*, 2011;**31**(4):769-786.

20. Vogelmeier CF, Criner GJ, Martinez FJ et al. Global Strategy for the Diagnosis, Management and Prevention of Chronic Obstructive Lung Disease 2017 Report: GOLD Executive Summary. *Respirology*, 2017;**22**(3):575-601.

21. Perret JL, Miles S, Brims F et al. Respiratory surveillance for coal mine dust and artificial stone exposed workers in Australia and New Zealand: A position statement from the Thoracic Society of Australia and New Zealand. *Respirology*, 2020;**25**(11):1193-1202.

22. Go LH, Krefft SD, Cohen RA et al. Lung disease and coal mining: what pulmonologists need to know. *Current Opinion in Pulmonary Medicine*, 2016;**22**(2):170-8.

23. Laney A, Weissman D. The classic pneumoconioses: new epidemiological and laboratory observations. *Clinics In Chest Medicine*, 2012;**33**(4):745-758.

24. Petsonk EL, Rose C, Cohen R. Coal mine dust lung disease. New lessons from old exposure. *American Journal of Respiratory and Critical Care Medicine*, 2013;**187**(11):1178-85.

25. McCormic ZD, Khuder SS, Aryal BK et al. Occupational silica exposure as a risk factor for scleroderma: a meta-analysis. *International Archives of Occupational and Environmental Health*, 2010;**83**(7):763-9.

26. Rubio-Rivas M, Moreno R, Corbella X. Occupational and environmental scleroderma. Systematic review and meta-analysis. *Clinical Rheumatology*, 2017;**36**(3):569-582.

27. Turner M, Samuel S, Silverstone E et al. Silica exposure and connective tissue disease: an underrecognized association in three Australian artificial stone workers. *American Journal of Respiratory and Critical Care Medicine*, 2020;**201**(3):378-380.

28. Bello S, Rinaldi A, Trabucco S et al. Erasmus syndrome in a marble worker. *Reumatismo*, 2015;**67**(3):116-22.

29. Ben Abdelghani K, Fazaa A, Souabni L et al. Association of pulmonary silicosis and systemic sclerosis. *BMJ Case Rep*, 2015;**2015**:bcr2013202509.

30. Chakrabarti S, Pan K. Erasmus Syndrome in a 42-year-old male: A rare case report. *Journal of Clinicial and Diagnostic Research*, 2015;**9**(5):Od01-3.

31. De Decker E, Vanthuyne M, Blockmans D et al. High prevalence of occupational exposure to solvents or silica in male systemic sclerosis patients: a Belgian cohort analysis. *Clin Rheumatol*, 2018;**37**(7):1977-1982.

32. Jain S, Joshi V, Rathore YS et al. Erasmus Syndrome: Silicosis and Systemic Sclerosis. *Indian Journal of Occupational and Environmental Medicine*, 2017;**21**(2):94-96.

33. Kim JY, Do SY, Moon YH et al. Systemic sclerosis due to crystalline silica exposure among jewelry workers in Korea: two case reports. *Annals of Occupational and Environmental Medicine*, 2017;**29**:18.

34. Pedro Gomes J, Shoenfeld Y. Morphea sculpted in silica: A case report of limited cutaneous systemic sclerosis in a woman with long-time exposure to silica dust. *Israel Medicial Association Journal*, 2017;**19**(7):459-460.

35. Rocha LF, Luppino Assad AP, Marangoni RG et al. Systemic sclerosis and silica exposure: a rare association in a large Brazilian cohort. *Rheumatology International*, 2016;**36**(5):697-702.

36. Sharma RK, Sharma AK, Sharma A. Erasmus Syndrome: Association of Silicosis and Systemic Sclerosis. *Indian Dermatology Online Journal*, 2018;**9**(3):185-187.

37. Dospinescu P, Jones GT, Basu N. Environmental risk factors in systemic sclerosis. *Current Opinion in Rheumatology*, 2013;**25**(2):179-183.

38. Mora GF. Systemic Sclerosis: Environmental Factors. *Journal of Rheumatology*, 2009;**36**(11):2383-2396.

39. Nietert PJ, Silver RM. Systemic sclerosis: environmental and occupational risk factors. *Current Opinion in Rheumatology*, 2000;**12**(6):520-6.

40. Nikpour M, Stevens WM, Herrick AL et al. Epidemiology of systemic sclerosis. *Best Practice and Research: Clinical Rheumatology*, 2010;**24**(6):857-869.

41. Nasser M, Larrieu S, Boussel L et al. Prevalence and mortality of systemic sclerosis-associated interstitial lung disease (SSc-ILD) using the French national health insurance system (SNDS) database in France. *European Respiratory Journal*, 2020;**56**(suppl 64):805.

42. Mohner M, Pohrt A, Gellissen J. Occupational exposure to respirable crystalline silica and chronic non-malignant renal disease: systematic review and meta-analysis. *International Archives of Occupational and Environmental Health*, 2017;**90**(7):555-574.

43. Shtraichman O, Blanc PD, Ollech JE et al. Outbreak of autoimmune disease in silicosis linked to artificial stone. *Occupational Medicine (Oxford)*, 2015;**65**(6):444-50.

44. Murphy D, Hutchinson D. Is male rheumatoid arthritis an occupational disease? A Review. *Open Rheumatol Journal*, 2017;**11**:88-105.

45. Schreiber J, Koschel D, Kekow J et al. Rheumatoid pneumoconiosis (Caplan's syndrome). *Europena Journal of Internal Medicine*, 2010;**21**(3):168-72.

46. Cooper GS, Miller FW, Germolec DR. Occupational exposures and autoimmune diseases. *International Immunopharmacology*, 2002;**2**(2-3):303-313.

47. Cooper GS, Parks CG. Occupational and environmental exposures as risk factors for systemic lupus erythematosus. *Current rheumatology reports*, 2004;**6**(5):367-374.

48. Parks CG, Cooper GS. Occupational exposures and risk of systemic lupus erythematosus. *Autoimmunity*, 2005;**38**(7):497-506.

49. Brown JM, Pfau JC, Pershouse MA et al. Silica, apoptosis, and autoimmunity. *Journal of Immunotoxicology*, 2005;**1**(3-4):177-187.

50. Miller FW, Alfredsson L, Costenbader KH et al. Epidemiology of environmental exposures and human autoimmune diseases: Findings from a National Institute of Environmental Health Sciences Expert Panel Workshop. *Journal of Autoimmunity*, 2012;**39**(4):259-271.

51. Parks CG, Conrad K, Cooper GS. Occupational exposure to crystalline silica and autoimmune disease. *Environmental Health Perspectives*, 1999;**107 Suppl 5**:793-802.

52. Industrial Injuries Advisory Council. *Occupational exposure to crystalline silica and its relation to connective tissue diseases: IIAC position paper 42*. 2018.

53. Liu Y, Steenland K, Rong Y et al. Exposure-response analysis and risk assessment for lung cancer in relationship to silica exposure: a 44-year cohort study of 34,018 workers. *American Journal of Epidemiology*, 2013;**178**(9):1424-33.

54. GBD 2016 Occupational Carcinogens Collaborators. Global and regional burden of cancer in 2016 arising from occupational exposure to selected carcinogens: a systematic analysis for the Global Burden of Disease Study 2016. *Occupational and Environmental Medicine*, 2020;**77**(3):151-159.

55. Australian Institute of Health and Welfare (AIHW). *Cancer Data in Australia: Australian Cancer Incidence and Mortality (ACIM) books: lung cancer*. 2018, AIHW: Canberra.

56. Institute for Health Metrics and Evaluation. *GBD Compare 2017*. 2019, IHME, University of Washington: Seattle, WA.

57. Lim S, Vos T, Flaxman A et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions,

1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*, 2012;**380**:2224–2260.

58. Australian Institute of Health and Welfare (AIHW). *Australian Burden of Disease Study: impact and causes of illness and death in Australia 2011*. 2016, AIHW: Canberra.
59. Shapira Y, Agmon-Levin N, Shoenfeld Y. Geoepidemiology of autoimmune rheumatic diseases. *Nature Reviews Rheumatology*, 2010;**6**(8):468-76.
60. Carey R, Driscoll T, Peters S et al. Estimated prevalence of exposure to occupational carcinogens in Australia (2011-2012). *Occupational and Environmental Medicine*, 2014;**71**(1):55-62.
61. Hoy RF, Baird T, Hammerschlag G et al. Artificial stone-associated silicosis: a rapidly emerging occupational lung disease. *Occupational & Environmental Medicine*, 2018;**75**(1):3-5.
62. Leso V, Fontana L, Romano R et al. Artificial Stone Associated Silicosis: A Systematic Review. *Int J Environ Res Public Health*, 2019;**16**(4).
63. Howard J. *Minimum latency and types or categories of cancer - World Trade Center Health Program*. 2015: Washington, DC.
64. Steenland K, Mannetje A, Boffetta P et al. Pooled exposure-response analyses and risk assessment for lung cancer in 10 cohorts of silica-exposed workers: an IARC multicentre study. *Cancer Causes and Control*, 2001;**12**(9): 773-784.
65. Harding A, Darnton A, Wegerdt J et al. Mortality among British asbestos workers undergoing regular medical examinations (1971-2005). *Occupational & Environmental Medicine*, 2009;**66**:487-495.
66. Luippold R, Mundt K, Austin R et al. Lung cancer mortality among chromate production workers. *Occupational & Environmental Medicine*, 2003;**60**(6):451-457.
67. Consonni D, De Matteis S, Pesatori AC et al. Lung cancer risk among bricklayers in a pooled analysis of case-control studies. *International Journal of Cancer*, 2015;**136**(2):360-71.
68. Olsson AC, Vermeulen R, Schuz J et al. Exposure-Response Analyses of Asbestos and Lung Cancer Subtypes in a Pooled Analysis of Case-Control Studies. *Epidemiology*, 2017;**28**(2):288-299.
69. Shtraichman O, Blanc PD, Ollech JE et al. Outbreak of autoimmune disease in silicosis linked to artificial stone. *Occupational Medicine*, 2015;**65**(6):444-50.