Key points

- Mineral dust exposure causes a number of conditions, including those specific to dust exposures, such as the pneumoconioses (or pulmonary fibroses due to mineral dust exposure), and others that may additionally be related to other causes, such as COPD.
- Identification of multiple conditions using respiratory investigations requires expert interpretation and understanding of the range of potential conditions.
- The frequency and content of a respiratory surveillance programme will vary according to the relevant occupational exposures, and be affected by both medical and nonmedical factors, including the background prevalence of local diseases. A programme will also need to consider other factors such as local legislation, availability of resources, worker convenience and cost.

Educational aims

- To identify the large range of respiratory diseases caused by exposure to mineral dusts and identify the range of tests that may be used in a surveillance programme for occupational respiratory disorders.
- To highlight difficulties that might be experienced by medical practitioners in designing and operating an effective surveillance programme, while incorporating rapidly advancing medical technology and practice.
Organising and interpreting respiratory surveillance for mineral dust-exposed workers requires specialist knowledge and understanding of the potential range of diseases, as well as a detailed occupational history. This has generated renewed interest in the utility and scope of respiratory surveillance in workers.

Among the general public, and even among healthcare practitioners, there is often confusion about what exactly is meant by health screening, respiratory surveillance and case finding. Broadly speaking, health surveillance is the process of collecting data about a specific disease or group.
of diseases in an exposed population. Respiratory surveillance is the process whereby a group of exposed workers are regularly tested to ensure that they are not developing respiratory diseases that are known to occur from specific work exposures. The ideal of such surveillance is to reduce the incidence of such disease to zero but in practice, it often just reduces disease to a low, acceptable level. The respiratory surveillance for CWP in UK mines after World War II is a good example of such a programme. Such dedicated, large respiratory surveillance programmes greatly improved understanding about the role of coal mine dusts in lung disease and improved knowledge about lung disease generally [2]. These programmes clearly demonstrated that reduction in coal dust levels resulted in a marked reduction in pneumoconiosis prevalence in coal miners [3].

Respiratory surveillance should be distinguished from population health screening, which is the usual type of screening that most patients and doctors will experience. This is the process of applying a specific test or set of tests to a healthy population in order to identify risk factors or early disease that can be treated or managed [4]. Its aim is to reduce the impact of the condition on the affected individual and on the incidence of a disease in a society. This screening is often used for cancer detection; for example, for breast cancer.

Respiratory surveillance also differs from case finding, which is a technique commonly used for investigating disease outbreaks and for protecting individuals from contracting an infectious disease. Here, exposed or potentially exposed people are identified by examining contacts of those who have tested positive for a particular disease. An example is tuberculosis contact tracing. The techniques used for case finding can also be used to identify diseases in an occupational or environmental setting (e.g. the South Korean humidifier disinfectant epidemic [5]). In Australia, recently, some case-finding activities have been initiated in artificial stoneworkers after it became clear that they were at high risk of progressive silicosis.

In this review, we will use the term “respiratory surveillance” to describe the process of screening exposed workers for early signs of disease.

**When is respiratory surveillance useful?**

According to the World Health Organisation (WHO), screening is defined as the presumptive identification of unrecognized disease in an apparently healthy, asymptomatic population by means of tests, examinations or other procedures which can be applied rapidly and easily to the target population [6]. The WHO has a list of criteria [4] for a cancer screening testing programme to be acceptable. These are not the same as those applied in workplace respiratory surveillance schemes, which are not cancer detection schemes.

Where workers are exposed to coal mine dusts and silica, the primary aim is to screen for pneumoconiosis (while there are other diseases related to mineral dust exposure). Reduction in dust levels has clearly reduced the incidence of pneumoconiosis in populations of workers over the last 60 years in many countries. These are long latency diseases and a corresponding time lag for a reduction in incidence is to be expected. Thus, long-term surveillance is important. Pneumoconiosis is specific to dust inhalation and can potentially be avoided, and progression can be reduced by reduction or cessation of exposure. Thus, the lack of any current accepted treatment for pneumoconiosis is irrelevant, unlike in population screening programmes.

Lung conditions that are potentially detectable in respiratory surveillance programmes include CWP and silicosis, COPD, diffuse dust fibrosis (interstitial pulmonary fibrosis related to inhalation of mineral dusts; strictly speaking, a pneumoconiosis), lung cancers, hypersensitivity pneumonitis and potentially several others. Although lung cancers may be diagnosed, a pneumoconiosis surveillance programme is not designed for the detection of cancer. Occupational respiratory surveillance may be considered more complex than population screening because the programme is looking for early signs of several diseases, some of which are very common in the general population and may have a variety of different risk factors. There is no “golden ticket” – a test that is a definite diagnostic test for any or all of the possible lung diseases – and therefore, a programme relies on a combination of tests to provide indicators of possible disease, to detect the individuals who require further specialist and individualised follow-up and to assess the severity of any work-detected disease. Respiratory surveillance is thus the first stage in diagnosis and not the final answer.

Workers are often obliged to undergo respiratory surveillance before starting employment, and then regularly throughout their employment, often in conjunction with screening for other occupational disorders. Programs in Australia are usually mandatory but may be voluntary in other parts of the world. These programs also offer the possibility of early detection of an incidental non-work-related condition, allowing for earlier commencement of relevant treatment. The commonest examples would be hypertension and diabetes, with obesity an increasing issue, but findings depend on the exact questions asked and tests performed within the program. Employers usually fund the surveillance programs, directly or otherwise, and the costs of any work-related disease are usually borne by relevant insurance programs. Thus, there are often financial differences for the worker between work-related and non-work-related conditions.

The 1996 WHO document “Surveillance of workers exposed to mineral dusts” is used worldwide today [7] but surveillance practices are also highly dependent on the availability of funding.
Respiratory surveillance in mineral dust-exposed workers

and access to investigations. The most commonly used tools for respiratory surveillance are:

- a standardised exposure history
- respiratory symptom questionnaires
- lung function testing
- chest imaging

In Australia, the general trend is moving beyond traditional surveillance tests and into the modern world, where high-resolution computed tomography (HRCT) scanning and full lung function testing have increased the sensitivity and specificity for diagnosis of lung disease [8], but this is beyond the financial capacity of most nations.

Which diseases are we looking for?

Exposure to mineral dusts, including coal dust and silica, can lead to a wide variety of different lung diseases (table 1). These can occur in isolation or in combination with others, making diagnosis difficult. Concomitant exposures, particularly to cigarette smoke and other inhaled agents, make the clinical picture even more complex.

Most countries have legislation mandating workplace surveillance in industries where crystalline free silica and other dusts are present above a certain level. However, even the best of regulations need to be enforced and this does not always occur. Complacency, lack of expertise, financial factors and others may all play a part in poorly functioning programmes [9].

Pneumoconiosis

The “classical pneumoconioses” are CWP and silicosis. Pneumoconiosis just means fibrosis of the lung due to mineral dust. The pneumoconioses are interstitial lung diseases that represent the lungs’ response to longstanding dust inhalation. Currently, the understanding is that they are characterised by a threshold for disease, i.e. significant disease does not occur in the absence of dust exposure, and that there is a clear dose–response relationship between the inhalation of dust and development of disease. Thus, increasing dust exposures result in increasing severity of disease.

Classical CWP and silicosis are characterised by nodular scarring on the chest radiograph, usually in the middle and upper lobes. Lung function deterioration usually develops later than radiological change and in early stages, these conditions are often asymptomatic. If exposure continues, progressive disease causes symptoms such as cough and breathlessness. Generally, however, symptoms occur late. Thus, respiratory surveillance is needed to detect early disease and these conditions are usually diagnosed first through radiological abnormalities.

Classical CWP is characterised by fibrotic nodules, which may eventually coalesce to produce large fibrotic masses known as progressive massive fibrosis (PMF). PMF frequently proves fatal for the affected individuals, and also occurs in silicosis. The nodules in CWP are typically smaller and less well defined than those of silicosis but there is considerable overlap between the radiological appearances, contributed towards also by the mixed exposures that are the reality of mining. Silicosis tends to result in denser nodules, hilar lymphadenopathy and calcification but many cases are actually “mixed dust fibrosis”, arising from mixed silica, coal and other dust exposures.

Accelerated (or progressive) silicosis

Recently, there has been a worldwide increase in a subtype of silicosis called accelerated or progressive

Case example 1

- 47-year-old male
- Abnormal chest radiograph on B-reading (2/3)
- Normal spirometry
- Asymptomatic
- Occupational history: 17-year history in retail, then 18-month history working in an office-based role on a coal mine site
- Follow-up testing: CT scan consistent with sarcoidosis
- Lung function testing all within normal limits
- Diagnosis: sarcoidosis. No evidence of role of mineral dust exposure, incidental finding.
- Management: can continue at work, management by respiratory physician
- Good recovery

Table 1  Conditions caused by mineral dust exposure or other inhaled agents in coal mines

| COPD         |
|--------------|
| Pneumoconioses (CWP, silicosis) |
| Lung cancer (silica)          |
| Diffuse dust-related fibrosis |
| Hypersensitivity pneumonitis  |
| Sarcoidosis?                 |

The increased incidence of tuberculosis and fungal infections in silica-exposed workers should also be noted.
silicosis, which occurs after high exposure to respirable crystalline free silica (RCS). Accelerated silicosis progresses at a greater than usual rate and can occur with short, intense exposures. It is rapidly progressive, and severe, fatal lung disease can occur, sometimes <10 years from the first exposure. Accelerated silicosis may be associated with an interstitial ground-glass infiltrate, probably reflecting rapid protein accumulation within the alveoli. Severe disease may occur, initially with few symptoms [10].

Accelerated silicosis occurred in Turkey recently with the production of sandblasted jeans. Textile workers were exposed to extremely high levels of RCS. Eventually, the practice was banned but not before silicosis had caused many deaths [11]. The recent epidemic of accelerated silicosis in Australian artificial stone workers is another example. Despite the emergence of silicosis elsewhere [12, 13], construction workers were increasingly installing benchtops made of imported artificial stone. Cutting, grinding and polishing this stone released large quantities of fine dust with a very high silica content (>90%). Most workers used very little ventilation, dust suppression or extraction. Masks were seldom used and were often unsuitable for protection. Studies have shown a prevalence of accelerated silicosis of up to 30% of in these workers [14] with progression to PMF in ~30% [15]. Although levels of RCS have undoubtedly been too high, artificial stone also contains several different constituents that could also be factors contributing to toxicity.

Because pneumoconioses are characteristically asymptomatic in the early stages and effects on lung function are late, they are usually diagnosed during a surveillance programme. Any cases diagnosed in secondary care should raise a warning flag.

**Chronic obstructive pulmonary disease**

COPD is one of the commonest diseases worldwide. COPD encompasses both chronic bronchitis (inflammation and remodelling of the small airways) and emphysema (destruction of the lung parenchyma) [16]. COPD is an “umbrella term”, and the disease can result from combinations of both environmental and host factors.

Although the commonest causes of COPD worldwide are cigarette smoking and biomass fuel exposure, COPD is also caused by mineral dust inhalation. COPD can occur in the presence or absence of radiological changes of pneumoconiosis, and can obscure the findings of classical CWP. Chronic cough with sputum production is strongly associated with dust exposure [17–19]. Dust inhalation stimulates bronchial goblet cell hyperplasia [20] and the prevalence of bronchitis correlates with the level of estimated coal mine dust exposure [21]. Centrilobular emphysema is the most common type of emphysema seen after coal mine dust exposure, although all types can occur. Emphysema severity is also related to the history of cumulative dust exposure [22] and to the dust content of the lungs.

Research studies from several different countries have shown that 1 year of underground coal mine work is approximately equivalent to a pack-year of tobacco smoking in causing the lung function decline associated with COPD [23]. Dust-induced COPD is pathologically and clinically identical to other types of COPD, and the causal link can easily be forgotten in the presence of other risk factors, notably tobacco smoking.

**Other interstitial lung diseases**

Other lung diseases of which early signs may be identified on surveillance include diffuse dust-related fibrosis (DDF) and sarcoidosis. Diffuse interstitial pulmonary fibrosis was first noted in Welsh coal miners in the 1940s, when irregular lower-zone opacities were seen on chest radiography, a pattern different from the upper-zone regular opacities found with CWP. However, these were initially attributed to other factors (smoking, extrinsic allergic alveolitis or idiopathic pulmonary fibrosis (IPF)). Clinical features can be identical to those of progressive interstitial pulmonary fibrosis of other causes, most notably IPF. DDF may occur alone or in conjunction with the upper lobe nodules of classical CWP and lower-lobe honeycomb may occur. Current information suggests that DDF tends to follow a less aggressive clinical cause than IPF [14, 24, 25]. Histopathologically, there may be pigmented as well as unpigmented areas in a lung biopsy, so it can be easily overlooked. The joint international statement on IPF states that a diagnosis of IPF requires exclusion of other known causes of interstitial lung disease and recommends a thorough occupational history [26].

Asymptomatic sarcoidosis may be identified in respiratory surveillance programmes and its early radiological stages can be very difficult to distinguish from CWP or silicosis. There is mounting evidence suggesting a relationship between sarcoidosis and occupational and environmental exposures [27, 28], supported by epidemiological studies [29]. Sarcoidosis likely arises from several triggers, with underlying predisposing factors. Thus, considering the current evidence base, cases of sarcoidosis should also be only diagnosed after a thorough occupational history has excluded dust exposures.

**Malignancy**

Respiratory surveillance may also detect lung malignancies. Lung cancer is well documented to be caused by exposure to RCS, even in the absence
of confounders such as tobacco smoking [30] and without radiological silicosis. Workers may also be exposed to other occupational carcinogens like diesel emissions or radon, and miners may also have relatively high rates of tobacco smoking. Although chest radiography has been disappointing in detection programmes for lung cancer, HRCT scanning has been more effective, with several studies demonstrating a clear mortality benefit in high-risk people (although at a significant financial cost) [31, 32]. Respiratory surveillance programmes are not currently set up with the intention of providing robust lung cancer screening but could well be adapted to this purpose.

Infections

Silica exposure is established as a risk factor for the development of tuberculosis and this risk is higher in the immunosuppressed. Patients with silicosis are also at increased risk of nontuberculous mycobacterial disease, cryptococcal infections and fungal infection [33]. The risk of exacerbation in chronic pneumoconiosis from other infections, including viruses, yet remains to be evaluated.

Other diseases

Other conditions linked to dust exposure, such as scleroderma and renal disease, are not currently screened for in most programmes, but there is a definite link between connective tissue disorders and silica exposure that has been recognised in many studies [34]. Much remains to be learnt about these associations. Autoantibody measurement has recently been suggested after artificial stone exposure in Australia [35].

Tests used in respiratory surveillance

Occupational history and exposure assessment

Several different tools are available to standardise taking a good quality, detailed occupational and environmental exposure history. At its most basic, the history needs to contain information about every job that the individual has ever held and potential exposures from these jobs. Other useful information includes the type of respiratory protection worn, exact tasks performed, and type of workplace or mine. Diagnosis of a work-related condition requires a detailed understanding of any exposures and their health effects, usually conducted by an occupational physician or others with expertise (e.g. occupational nurses or hygienists).

The US National Institute of Occupational Safety and Health (NIOSH) uses a simple mining-focused work history questionnaire in its Coal Workers’ Health Surveillance Program [36], and this has also been adopted by the Queensland Coal Mine Workers’ Health Scheme (CMWHS) and more widely. Using the same questionnaire allows for consistency in data collection across jurisdictions if the surveillance programme is to be used for large-scale surveillance or research.

Exposure assessment is generally not part of routine medical training and may be difficult for inexperienced practitioners, particularly in complex scenarios, or in situations with medicolegal ramifications. More complex standardised questionnaires to assist with exposure assessment are also available for use in complex cases.

Respiratory symptom questionnaire

There are a variety of well-validated respiratory symptom questionnaires, primarily designed for research or for diagnosis in symptomatic individuals. These include the modified Medical Research Council (mMRC) dyspnoea scale, the COPD Assessment Test, the St George’s Respiratory Questionnaire and the King’s Brief Interstitial Lung Disease questionnaire [37]. In the setting of a formal surveillance programme, the most commonly used questionnaire is the mMRC scale, supplemented by other standardised questionnaires if needed.

Questionnaire responses may be affected by other issues when screening exposed but generally asymptomatic individuals. There may be lack of
Spirometry and full lung function testing

Spirometry is essential for the diagnosis of respiratory disease and provides classification of severity in many disorders. Although in many diseases it may be completely normal at diagnosis, serial values are very useful for detecting disease. The Global Lung Function Initiative (GLI) [38] has collected respiratory function data from around the world and produced reference equations for spirometry. International collaboration involving >40 countries has resulted in standardised GLI spirometry reference equations that can be used globally for people aged from 3 to 95 years.

The process of collecting spirometric data from an individual is best performed by a technician with appropriate training and equipment. Training is required to ensure that all participants in the process, including the technicians and overseeing physicians, understand potential technical difficulties and the ways to overcome them. Accreditation of training and of laboratories is now available, and ensures that good-quality spirometry is available. Poor-quality spirometry cannot be interpreted and may significantly over- or underestimate an individual’s risk of respiratory disease. In such cases, the test should be performed until good-quality values are obtained.

Longitudinal, good-quality spirometry testing can be used to compare lung function in individuals over time. It is essential that identical reference values are used each test, as there is some variety between the ranges. Thus, recording of actual values every time, along with height and weight, is valuable. Several jurisdictions now mandate the use of a particular reference range for consistency, such as the Queensland CMWHS requiring the use of the GLI reference values [38], and it is likely that more countries will do so in the future.

No spirometric abnormalities or patterns of abnormality are, in isolation, diagnostic of any disease. All relevant abnormalities require further investigation by an appropriately qualified specialist. Only after this, and in combination with the exposure history, can a determination on the relationship to work be made.

Whilst most surveillance programmes rely on the collection of basic spirometric data (forced expiratory volume in 1 s (FEV₁), forced vital capacity (FVC) and FEV₁/FVC ratio), more sophisticated lung function tests, including lung volumes and diffusing capacity of the lung for carbon monoxide (D₅₀), are increasing available, and may provide better information for diagnosis and detection of earlier disease. D₅₀ testing is more sensitive than spirometry in the diagnosis and prediction of mortality from emphysema [39, 40], and may be adopted in future surveillance programmes after evaluation. For example, with artificial stone exposure, the Royal Australasian College of Physicians/Australasian Faculty of Occupational and Environmental Medicine recommends that formal, laboratory-based lung function testing be performed as standard [41].

Longitudinal interpretation of results is facilitated by good record keeping and test performance [42]. Decision making regarding the limits of expected longitudinal decline is facilitated by use of the free the SIPROLA program (www.cdc.gov/niosh/topics/spirometry/spirola-software).

Chest imaging

Historically, the mainstay of screening for pneumoconiosis detection was via a chest radiograph. Detecting nodules in early disease is difficult and requires much experience. One of the findings of the inquiry into the re-emergence of pneumoconiosis in Queensland was that a number of abnormal chest radiographs had been reported as normal [9] and it is widely acknowledged that the chest radiograph is a less sensitive tool for detecting early lung disease than more modern techniques such as HRCT.

The International Labor Organisation (ILO) recommends use of its International Classification of Radiographs of Pneumoconioses for interpretation of chest radiographs used in respiratory surveillance. There is also a training programme called the B-reader program, provided through NIOSH. This system, originally developed in the UK [43], provides standardised images and a grading system. Its original purpose was for epidemiological research, not diagnosis. However, many jurisdictions now include it in their diagnostic requirements for compensation purposes.

An ILO chest radiograph reading requires a specialist reader (usually a radiologist or other specialist physician) to compare the image with standardised images and provide a reading. There are four major categories (0 to 3) with each category having three subcategories, giving a total of 12 potential readings. Category 0/1 or below (0/0 or 0/−) are negative readings not consistent with a radiographic diagnosis of pneumoconiosis; 1/0 and above are potentially consistent. It is generally accepted that chest radiographs, even when read by experts, have a relatively high false-positive rate and interindividual variation [44]. For these reasons and for the purpose of excluding other diagnoses, further testing including HRCT is usually used for specialist diagnosis.

HRCT scanning is becoming increasingly advocated as a first-line tool for dust-exposed workers. Technical developments have resulted in a reduction in radiation dose, lower costs and
increased availability of HRCT. In Australia, the Royal Australian College of Radiologists guidelines have recently advocated the use of HRCT scanning rather than chest radiography for evaluation of artificial stone workers [45]. However, CT scanning facilities are not widely available in many countries and the costs may be excessive. Thus, it is likely that chest radiographs will remain the mainstay of respiratory surveillance programmes for the moment.

Examples of large-scale screening programmes

Many jurisdictions around the world require employers to provide health surveillance for their workers exposed to mineral dusts and many different surveillance schemes exist.

One example of a centralised programme is the CMWHS in Queensland. This is a mandatory surveillance programme run by the state government regulator, and conducted by specially trained and registered medical professionals. It is funded by the employers and requires workers to have an assessment at the start of their coal mining career and at least every 5 years while working in coal. Respiratory surveillance includes an exposure history, a standardised respiratory questionnaire, a chest examination, spirometry and a chest radiograph read by at least two B-reader trained radiologists. Since 2016, >50000 radiographs have been read to ILO standards in the scheme. Miners can continue to receive free surveillance testing after retirement from the industry on a voluntary basis.

In the USA, the Coal Workers’ Health Surveillance Program is a federal programme operated by NIOSH. Like the Queensland programme, workers undergo a chest radiograph read by at least two B-readers, and spirometry. However, several important differences exist. The US programme is mandatory before initiating work as a coal miner but voluntary thereafter. Workers provide answers to questionnaires but are not examined by doctors. In addition, former miners are not able to participate routinely in this programme. It is likely that limited participation in the CWHSP during coal mining careers has contributed to the increased finding of PMF in regions of the USA in recent decades.

Diagnosis and management

Diagnosis of dust-induced lung diseases is complex and requires detailed understanding of a broad range of nonoccupational respiratory factors and occupational issues. An abnormal set of results on respiratory surveillance is only the beginning of the investigative pathway, which may then include full lung function testing, exercise testing, bronchoscopy, biopsy and cultures, and eventually even thoracoscopic lung biopsy. Although there is a natural reluctance to embark upon invasive tests in a person with few symptoms, obtaining the correct diagnosis may have major implications for the worker and their family in the future, and could even prove lifesaving.

Diagnostic certainty is important. The impact of an incorrect diagnosis on an individual worker can be devastating, causing loss of income, psychological ill health and family dysfunction. One factor that is often underestimated is the effect of lack of certainty, which also may lead to lack of action or treatment. Patients and communities often have misplaced perceptions about the accuracy of diagnostic tests such as chest imaging and lung function, and may not appreciate the full range of different potential diagnoses. In such circumstances, it is probably better to be totally honest about a lack of certainty, and explain the need for further confirmatory tests and how even these can sometimes prove difficult to interpret.

Management of the whole situation of the impact of an occupational lung disease is a broader concept, and involves a worker’s individual medical and personal circumstances as well as employment factors. For example, a worker nearing retirement may find leaving work much easier than a man in the middle of his working career with a family to support. A worker who lives in a city with many available jobs may be much more willing to look for alternative employment than one who might have

Self-evaluation questions

1. Which of the following conditions is not associated with exposure to mineral dusts?
   a. COPD
   b. Silicosis
   c. Hypersensitivity pneumonitis
2. When assessing for the pneumoconiosis, which of the following radiological modalities is not useful for surveillance?
   a. Chest radiography
   b. Noncontrast computed tomography (CT)
   c. CT with contrast
3. Why is surveillance for occupational lung diseases associated with mineral dust exposure more challenging than some other surveillance programmes?
   a. There are multiple diseases that can be caused by the same exposure.
   b. There is no one test that is sensitive or specific for each potential disease.
   c. Both a and b are true.
4. How often should a worker undergo respiratory surveillance testing?
   a. Every year
   b. Every 3 years
   c. Every 5 years
   d. It is currently uncertain and may depend on the specific exposures or jurisdiction
**Suggested answers**

1. c. Hypersensitivity pneumonitis. This is not caused by exposure to mineral dust. It is caused by exposure to allergens in dust form; these are usually organic and can be associated with moulds, bacteria or allergens related to birds.

2. c. CT with contrast. Chest radiography is the currently the most commonly used modality for surveillance for occupational lung disease and the first line imaging. Although CT scanning is becoming more widely recognised as a future first-line option, particularly for accelerated silicosis, this is not currently recommended for respiratory surveillance. It is possible that this could change in the future. There is no evidence that use of contrast assists with the diagnosis of pneumoconiosis and, in fact, can make radiological interpretation more difficult, especially with silicosis.

3. c. Both a and b are true. It is challenging to design a programme for multiple diverse conditions that often require further specialised investigation for definite confirmation. Current tests used in respiratory surveillance are imperfect, lacking both sensitivity and specificity. With appropriate specialist interpretation, the results of a variety of tests in respiratory surveillance can be used to decide whether a disease requires further diagnostic testing or whether a diagnosis can be made.

4. d. It is currently uncertain and may depend on the specific exposures or jurisdiction. The frequency of surveillance currently varies. Some programmes require annual testing (usually excluding imaging, which involves exposure to radiation) with chest radiography performed every 3–5 years. More frequent radiological and clinical testing may be required for high exposures (e.g. in workers at risk of accelerated silicosis).

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difficulty finding another job. Financial factors are very important and should not be underestimated. Most people will prioritise their health highly but may need time to come to terms with this. Repeat consultations with adequate time allocated for open discussion of these issues are very valuable, as is access to relevant support services such as pulmonary rehabilitation, counselling and psychological support. Other sources of support can include smoking cessation advice, drug and alcohol and social services. Full discussion of these topics is outside the scope of this paper but their importance cannot be underestimated, as well as the benefits of close liaison with the caring primary health team.

Although there are currently no documented effective treatments for pneumoconiosis, removal from dust exposure in early disease should prevent progression to chronic disability. There are many potentially effective treatments for coal mine lung dust-related disorders that have yet to be evaluated, including antifibrotic therapies that have been shown to be effective in trials into lung fibrosis from other causes [46, 47]. Additionally, there is an excellent evidence base for modern treatment of COPD, lung cancer and respiratory infections, and proven efficacy of vaccines in promoting respiratory health. Pulmonary rehabilitation is effective in improving exercise capacity and health-related quality of life for benign dust-related diseases as well as for COPD [48], but more studies are needed. Smoking cessation is achievable and has long-term health benefits.

**Conclusions**

Respiratory surveillance programmes for mineral-dust exposed workers are an established method of reducing the burden of occupational lung diseases. With better understanding of the spectrum of disorders that can follow dust exposure, it is now recognised that several lung diseases can be identified and managed, in addition to classical pneumoconiosis. Important measures such as smoking cessation can also be implemented. While there are currently no documented effective treatments for pneumoconiosis, there are new options for treatment that are likely to prove useful in the near future.

Pneumoconiosis is a preventable disease. Even a single case in a respiratory surveillance programme represents a failure of dust control, which should prompt re-examination of working conditions. Surveillance schemes are not designed to be primary prevention measures but provide a useful check on past and existing control measures. They need to be continued after a worker retires due to the long latency before disease presentation. Any surveillance programme should be based on the best medical evidence, tailored to suit the individual disease and be provided at no cost to the worker. This should include appropriate specialist follow-up for those who test “positive” as well as broad support and case management. The detection of even a few cases of irreversible disease represents a system failure that is no longer acceptable in the modern working environment.

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Conflict of interest

C. Wood reports previous employment with the Dept of Natural Resources and Mines outside the submitted work. D. Yates has nothing to disclose.

References

1. Morgan J. Black lung is still a threat. *Lancet Respir Med* 2018; 6: 745–746.
2. Hurley JF, Copland D, Dodgson J, et al. Simple pneumoconiosis and exposure to respirable dust: relationships from twenty-five years’ research at ten British collieries. IOM Historical research report 1979; TM/79/12. Available from: www.iom-world.org
3. Health and Safety Executive. Pneumoconiosis. www.hse.gov.uk/lung-disease/pneumoconiosis.htm. Date last accessed: October 31, 2019.
4. World Health Organisation. Screening. www.who.int/cancer/prevention/diagnosis-screening/screening/en. Date last accessed: October 31, 2019.
5. Paek D, Koh Y, Park DU, et al. Nationwide study of humidifier disinfectant lung injury in South Korea, 1994-2011: incidence and dose-response relationships. *Ann Am Thorac Soc* 2015; 12: 1813–1821.
6. Wilson JMG, Jungner G. Principles and Practices of Screening for Disease. Geneva, World Health Organization, 1968.
7. Wagner GR, World Health Organization, WHO Meeting on the Screening and Surveillance of Workers Exposed to Mineral Dust. Screening and Surveillance of Workers Exposed to Mineral Dust. Geneva, World Health Organization. 1996.
8. Weissman DN. Role of chest computed tomography in prevention of occupational respiratory disease: review of recent literature. *Semin Respir Crit Care Med* 2015; 36: 423–448.
9. Monash Centre for Occupational and Environmental Health, School of Public Health, UIC. Review of Respiratory Component of the Coal Mine Workers’ Health Scheme for the Queensland Department of Natural Resources and Mines. 2016. www.business.qld.gov.au/industries/mining-energy-water/resources/safety/health/mining/medicals/dust-lung-disease/inquiries
10. Hoy RF, Baird T, Hammerschlag G, et al. Artificial stone–associated silicosis: a rapidly emerging occupational lung disease. *Occup Environ Med* 2017; 75: 3–5.
11. Akgun M, Mirici A, Ucar EY, et al. Silicosis in Turkish denim sandblasters. *Occup Med (Lond)* 2006; 56: 554–558.
12. Perez-Alonso A, Cordoba-Dona JA, Millares-Lorenzo JL, et al. Outbreak of silicosis in Spanish quartz conglomerate workers. *Int J Occup Environ Health* 2014; 20: 26–32.
13. Kramer MR, Blanc PD, Fireman E, et al. Artificial stone silicosis: disease resurgence among artificial stone workers. *Ches* 2012; 142: 419–424.
14. Edwards G, Knight R. Australia’s current workplace epidemic: accelerated silicosis. *Intern Med* 2019; 49: 26–26.
15. Matar E, Frankel A, Blake LKM, et al. Complicated silicosis resulting from occupational exposure to engineered stone products. *Med J Aust* 2017; 206: 385–386.
16. Singh D, Agusti A, Anzueto A, et al. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease: the GOLD science committee report 2019. *Eur Respir J* 2019; 53: 1900164.
17. Coggan D, Newman Taylor A. Coal Mining and chronic obstructive pulmonary disease: a review of the evidence. *Thorax* 1998; 53: 398–407.
18. Laney AS, Weissman DN. Respiratory diseases caused by coal mine dust. *J Occup Environ Med* 2014; 56: Suppl. 10, S18–S22.
19. Petsonk EL, Rose C, Cohen R. Coal mine dust lung disease: new lessons from an old exposure. *Am J Respir Crit Care Med* 2013; 187: 1178–1185.
20. Leigh J. 15 year longitudinal studies of FEV1 loss and mucus hypersecretion development in coal workers in New South Wales, Australia. In: Proceedings of the 8th International Pneumoconiosis Conference Part II. U.S. Department of Health and Human Services, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH), 112–21. www.cdc.gov/niosh/docs/90-108/. Date last accessed: October 23, 2019.
21. Henneberger PK, Atttfeld MD. Respiratory symptoms and spirometry in experienced coal miners: effects of both distant and recent coal mine dust exposures. *Am J Ind Med* 1997; 32: 268–274.
22. Kuepel ED, Wheeler MW, Smith RJ, et al. Contributions of dust exposure and cigarette smoking to emphysema severity in coal miners in the United States. *Am J Respir Crit Care Med* 2009; 180: 257–264.
23. Atttfeld MD, Hodous TK. Pulmonary function of US coal miners related to dust exposure estimates. *Am Rev Respir Dis* 1992; 145: 605–609.
24. Cockcroft A, Berry G, Cotes JE, et al. Shape of small opacities and lung function in coalworkers. *Thorax* 1982; 37: 765–769.
25. McConnochie K, Green FHY, Vallyathan V, et al. Interstitial fibrosis in coal workers: experience in Wales and West Virginia. *Ann Occup Hyg* 1988; 32: 5553–5560.
26. American Thoracic Society. Idiopathic pulmonary fibrosis: diagnosis and treatment. International consensus statement. American Thoracic Society (ATS), and the European Respiratory Society (ERS). *Am J Respir Crit Care Med* 2000; 161: 646–664. www.ncbi.nlm.nih.gov/pubmed/10673212
27. Newman KL, Newman LS. Occupational causes of sarcodosis. *Curr Opin Clin Immunol* 2012; 12: 145–150.
28. Hena KM, Yip J, Jaber N, et al. Clinical course of sarcodosis in World Trade Center-exposed firefighters. *Ches* 2018; 153: 114–123.
29. Kreider ME. Relationship of environmental exposures to the clinical phenotype of sarcodosis. *Ches* 2005; 128: 207–215.
30. Sato T, Shimosato T, Klinman DM. Silicosis and lung cancer: current perspectives. *Lung Cancer (Auckl)* 2018; 9: 91–101.
31. Horeweg N, van der Aalst CM, Vliegenthart R, et al. Volumetric computed tomography screening for lung cancer: three rounds of the NELSON trial. *Eur Respir J* 2013; 42: 1659–1667.
32. Aberle DR, Adams AM, Berg CD, et al. Reduced lung-cancer mortality with low-dose computed tomographic screening. *N Engl J Med* 2011; 365: 395–409.
33. Leung CC, Yu ITS, Chen W. Silicosis. *Lancet* 2012; 379: 2008–2018.
34. Industrial Injuries Advisory Council. Occupational Exposure to crystalline silica and its relation to connective tissue diseases. Position paper 42. June 2018. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/717051/occupational-exposure-to-crystalline-silica-and-its-relation-to-connective-tissue-diseases-iiac-position-paper-42.pdf. Date last accessed: October 31, 2019.
35. Turner MT, Samuel SR, Silverstone EJ, et al. Silica exposure and connective tissue disease: an under-recognised association in three Australian artificial stone workers. *Am J Respir Crit Care Med* 2019; 200: 510.
36. NIOSH. Miner Identification Document. www.cdc.gov/niosh/topics/cw/pdf/cw/miner-id-2.9.pdf
37. Williams N. The MRC breathlessness scale. *Occup Med* 2017; 67: 496–497.
38. The Thoracic Society of Australia and New Zealand. Spirometry Standards. www.thoracic.org.au/respiratorylaboratoryaccreditation/spirometry-standards. Date last updated: 2015.
39. Pellegrino R, Viegi G, Brusasco V, et al. Interpretative strategy for lung function tests. *Eur Respir J* 2005; 26: 948–968.
40. Boutou AK, Shrikrishna D, Tanner RJ, et al. Lung function indices for predicting mortality in COPD. *Eur Respir J* 2013; 42: 616–625.

41. Royal Australasian College of Physicians. Accelerated Silicosis: Overview. www.racp.edu.au/advocacy/division-faculty-and-chapter-priorities/faculty-of-occupational-environmental-medicine/accelerated-silicosis/overview. Date last accessed: October 9, 2019.

42. Litow FK, Petsonk EL, Bohnker BK, et al. ACOEM guidelines: occupational interstitial lung diseases. *J Occup Environ Med* 2015; 57: 1250–1254.

43. Cotes JE. The Medical Research Council Pneumoconiosis Research Unit, 1945–1985: a short history and tribute. *Occup Med (Lond)* 2000; 50: 440–449.

44. Gevenois PA, Pichot E, Dargent F, et al. Low grade coal worker’s pneumoconiosis. Comparison of CT and chest radiography. *Acta Radiol* 1994, 35: 351–356.

45. Royal Australian and New Zealand College of Radiologists. Imaging of Occupational Lung Disease: Silicosis Position Statement (2019). www.ranzcr.com/fellows/clinical-radiology/professional-documents/?searchword=artificial+stone&ordering=published_date+DESC&direction=asc&limitstart=0

46. Rodríguez-Portal JA. Efficacy and safety of nintedanib for the treatment of idiopathic pulmonary fibrosis: an update. *Drugs R D* 2018; 18: 19–25.

47. Richeldi L, du Bois RM, Raghu G, et al. Efficacy and safety of nintedanib in idiopathic pulmonary fibrosis. *N Engl J Med* 2014; 370: 2071–2082.

48. Dale MT, McKeough ZJ, Troosters T, et al. Exercise training to improve exercise capacity and quality of life in people with non-malignant dust-related respiratory diseases. *Cochrane Database Syst Rev* 2015; 11: CD009385.