Review and Update of the Prescription for Prescribed Disease D1 (Pneumoconiosis)

November 2023

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Dear Secretary of State,

The Industrial Injuries Advisory Council (IIAC) has undertaken an extensive review of the prescription for the industrial disease pneumoconiosis (includes silicosis and asbestosis) PD D1, which is covered by industrial injuries disablement benefit.

Pneumoconiosis is the generic term for a class of interstitial lung disease where inhalation of dust (for example, coal dust) has caused interstitial fibrosis. The three most common types are asbestosis, silicosis, and coal miner's lung (black lung). It is an occupational lung disease, typically from years of dust exposure during work in mining; textile milling; shipbuilding, ship repairing, and/or shipbreaking; sandblasting; industrial tasks; rock drilling (subways or building pilings); or agriculture. It is one of the most common occupational diseases in the world.

The current PD D1 prescription has been in place for nearly 80 years and has not undergone any substantial revision in that time, despite changes to modern working practices. The current prescription is now unsatisfactory in several aspects, including being difficult to navigate for both claimants and administrators. Many of the occupations specified are largely historic and do not reflect occupations where current exposures occur, particularly for silica. Substantial exposure to silica can occur, for example, in the construction industry, in worktop manufacture and fitting and in dental prosthetic production.

This command paper outlines the historic background to PD D1 and the current prescription and then recommends four simplified categories of occupational exposures to be included in a revised prescription, namely:

1. Asbestos
2. Coal or coal mine dust
3. Silica-containing dusts
4. Metal dusts: aluminium, beryllium, cobalt, indium tin oxide, rare earth metals, and tungsten carbide

This report also sets out IIAC's recommendations that the disease considered in the prescription should be 'pneumoconiosis' without any further qualification as this term will be understood by respiratory specialists. IIAC takes the view that a diagnosis of pneumoconiosis normally requires specialist investigation to confirm the presence of diffuse lung disease and exclude any alternative possible diagnoses. To bring the PD D1 prescription in line with others, IIAC recommends a diagnosis of pneumoconiosis should not automatically lead to an award of benefit irrespective of the degree of disability; the award should be based on the extent of disability. This should be quantified as it is at present on an individual basis taking into account all available information including symptoms, lung function, radiological features, and co-morbid conditions.

Currently, the PD D1 prescription has an open category and as there are relatively few awards made under this provision, it is recommended that the open category be removed and that if any new causes of pneumoconiosis become recognised these should be assessed by IIAC and a decision made about their inclusion in the schedule of prescribed exposures.

By making these recommendations, IIAC anticipates the prescription will be easier to navigate for claimants as it removes the specifications for employment, also making it easier from an administrative perspective.

Yours sincerely,

Dr Lesley Rushton

Chair, Industrial Injuries Advisory Council

Review and Update of the Prescription for Prescribed Disease D1 (Pneumoconiosis)

Summary

The current prescription for pneumoconiosis dates back in part over 100 years and has not undergone any major revision for almost 80 years. In that time the causes of the disease and the diagnostic techniques have changed substantially. The current prescription can be difficult to navigate for both claimants and administrators. In addition, the specified occupations are largely historic and do not include all those where current exposures may occur, particularly for silica. The Council recommends that the disease considered in the revised prescription should be '*pneumoconiosis*' without any further qualification and that work involving exposures to the following should be included as potential causes of pneumoconiosis for the purpose of PD D1:

1. Asbestos
2. Coal or coal mine dust
3. Silica-containing dusts
4. Metal dusts: aluminium, beryllium, cobalt, indium tin oxide, rare earth metals, and tungsten carbide

The clinical and radiological features of pneumoconioses can be similar to those of other lung diseases and there are often alternative diagnoses and treatments. IIAC therefore expects that a specialist clinical opinion should normally be obtained before an application for IIDB for pneumoconiosis is considered.

IIAC recommends that awards of benefit for pneumoconiosis should be brought into line with those for other conditions, with no automatic award on diagnosis irrespective of the actual disability. Claims for comorbid chronic obstructive pulmonary disease (COPD) should now be considered separately. In addition, awards for complicating tuberculosis and non-tuberculous mycobacterial infection should be restricted to pneumoconioses associated with silica exposures.

No changes are proposed to the extent of exposure needed to establish a diagnosis for any of the prescribed exposures or to the assessment of disability.

Introduction

1. The current prescription for pneumoconiosis is an old one that refers back to compensation schemes from more than 100 years ago. It has not undergone any substantial revision for almost 80 years although it has been considered by the Industrial Injuries Advisory Council (IIAC) several times, and Council reports were published in 1953,1973,1996, 2005 and 2006. Over that period understanding, definitions and terminology relating to the condition have changed, as have diagnostic techniques. The current prescription is now unsatisfactory in several aspects, including being difficult to navigate for both claimants and administrators. Many of the occupations specified are largely historic and do not reflect occupations where current exposures occur, particularly for silica. The All-Party Parliamentary Group for Respiratory Health has recently drawn attention to the general lack of awareness across industry of the potential impact of silica exposure and the risk of development of silicosis (APPG 2023).

2. This command paper outlines the historic background to PD D1 and the current prescription and then recommends four simplified categories of occupational exposures to be included in a revised prescription, namely:

    1. Asbestos
    2. Coal or coal mine dust
    3. Silica-containing dusts
    4. Metal dusts: aluminium, beryllium, cobalt, indium tin oxide, rare earth metals, and tungsten carbide.

3. The report describes each condition and the specific substances of concern and reviews the potential for exposure, diagnosis, and any disability that might occur. Notes on the individual pneumoconiosis can be found in appendix 2.

4. The Council has considered a wide range of related substances and exposures where evidence relating them to pneumoconiosis is weaker and these have not been recommended for prescription.

5. Despite the concerns about PD D1 outlined above, a substantial number of claims are made under the current prescription. There were 12,245 Industrial Injury Disablement Benefit (IIDB) awards between 2010 and 2019, i.e., approximately 1200 per annum, most being related to exposure to asbestos, coal and silica with nearly 80% being for asbestosis. The Council anticipates that the recommended changes may reduce the number of successful claims for low level disablement amongst those with asymptomatic pneumoconiosis. However, for silicosis, the Council hopes that the changes will raise awareness of the disease amongst workers and employers and will contribute to improvement in the detection and diagnosis of the condition.

The Industrial Injuries Disablement Benefit Scheme

6. The IIDB Scheme provides non-contributory, 'no-fault' benefits for disablement because of accidents or prescribed diseases which arise during the course of employed earners' work. The benefit is paid in addition to other incapacity and disability benefits. It is tax-free and administered by the Department for Work and Pensions (DWP).

7. The legal requirements for prescription are set out in The Social Security Contributions and Benefits Act 1992 which states that the Secretary of State may prescribe a disease where they are satisfied that the disease:

   (a) ought to be treated, having regard to its causes and incidence and any other relevant considerations, as a risk of the occupation and not as a risk common to all persons; and

   (b) is such that, in the absence of special circumstances, the attribution of particular cases to the nature of the employment can be established or presumed with reasonable certainty.

8. Thus, a disease may only be prescribed if there is a recognised risk to workers in an occupation and the link between disease and occupation can be established or reasonably presumed in individual cases.

The Role of the Industrial Injuries Advisory Council

9. IIAC is an independent statutory body established in 1946 to advise the Secretary of State for Work and Pensions on matters relating to the IIDB scheme. The majority of the Council's time is spent considering whether the list of prescribed diseases for which benefit may be paid should be enlarged or amended.

10. In considering the question of prescription, the Council searches for a practical way to demonstrate, in the individual case, that the disease can be attributed to occupational exposure with reasonable confidence; for this purpose, 'reasonable confidence' is interpreted as being based on the balance of probabilities.

11. Some occupational diseases are relatively simple to verify, as the link with occupation is clear-cut. Some only occur due to particular work, are almost always associated with work, have specific medical tests which prove their link with work, have a rapid link to exposure, or have other clinical features that make it easy to confirm the work connection. However, many other diseases are not uniquely occupational, and when caused by occupation, are indistinguishable from the same disease occurring in someone who has not been exposed to a hazard at work. In these circumstances, attribution to occupation depends on research evidence that work in the prescribed job or with the prescribed occupational exposures causes the

disease on the balance of probabilities. The Council thus looks for evidence that the risk of developing the disease associated with a particular occupational exposure or circumstance is more than doubled (previous reports of the Council explain why this threshold was chosen).

Historical Background to PD D1

12. The term pneumoconiosis (or pneumokoniosis) was coined in the mid-19th century to denote dust-related conditions of the lung. It is an umbrella term encompassing conditions such as silicosis, coal workers' pneumoconiosis and asbestosis. There are other dust-related lung diseases such as asthma, byssinosis, and COPD to which the term pneumoconiosis is not generally applied.

13. The *1906 Workmen's Compensation Act* was the first to make provision for compensation for occupational diseases (Meiklejohn 1954), but it did not include any lung disease. The possibility of inclusion of 'fibroid phthisis' (silicosis) was considered in 1907 but was not accepted for reasons that included the difficulty of establishing a diagnosis. At that stage chest radiography was still in its infancy.

14. The *1919 Refractories Industries (Silicosis) Scheme* provided for compensation to ganister (silica) miners and silica brickmakers, who suffered death, were totally disabled or who had silicosis accompanied by disabling tuberculosis. Silicosis is known to be associated with an increased risk of developing tuberculosis which at the time was a common disease.

15. The *1927 Metal Grinding Industries (Silicosis) Scheme* extended provision to workers engaged in grinding metals, and certain related processes. The *1928 Various Industries (Silicosis) Scheme* modified the 1919 Act to include additional mining and quarrying operations. A 1931 amendment added masonry work, tin mining and the manufacture of scouring powders. Various processes in the pottery industry were then added, as was sandblasting of metals.

16. The *1931 Asbestos Industry (Asbestosis) Scheme* extended compensation to those with asbestosis.

17. The *1943 Workmen's Compensation Act* added coal mining as a cause of pneumoconiosis and consolidated the other schemes, extending cover to 'any form of pneumoconiosis'. It introduced the current statutory definition of pneumoconiosis:

> '*Fibrosis of the lungs due to silica dust, asbestos dust, or other dust and includes the condition of the lung known as dust reticulation'.*

18. The various existing schemes were amalgamated into the *1946 National Insurance (Industrial Injuries) Act*. There was additional provision for sandblasting of glass and shot blasting. To ensure a smooth transition from the old schemes the 1946 Act incorporated largely unchanged a number of '*elaborate and detailed schedules'* that had built up over the years.

19. The categories in the 1946 Act were intended to be applied on a temporary basis and they were reviewed in a *1953 IIAC Command Paper (Cmd 8866: Pneumoconiosis)*.

20. Three main issues were addressed in the 1953 IIAC paper:

    (i) The definition of the disease:

        i. IIAC considered whether to define pneumoconiosis as dust-related lung disease without further clarification or to include a requirement for lung fibrosis. That reflected awareness of the developing problem of COPD in coal miners and its possible relation to work. IIAC elected to retain fibrosis as an essential component of the definition of pneumoconiosis. There was discussion about whether to prescribe separately the various pneumoconioses, but the final view was to retain the generic term.

    (ii) Causes of pneumoconiosis:

        ii. The review recognised the problems of an ever-increasing list of circumstances under which pneumoconiosis might occur. Already from 1946 three new exposures had been added to the list:
            - The use of power-driven tools to free metal castings;
            - The manufacture of carbon electrodes;
            - Boiler scaling.

        iii. IIAC discussed dispensing with a list of prescribed agents altogether but felt it should be retained. The Council made two main recommendations:
            - The introduction of an 'open' category for exposures not listed in the schedule:

    "Exposure to dust if the person employed in it has never at any time worked in any of the other occupations listed."

Those applying under the open category would not benefit from a presumption that their disease was caused by their work. Those applying under the other categories would.

- IIAC recommended that any future additions to the list of prescribed occupations should be expressed in broad terms rather than detailing specific circumstances or exposures.

(iii) The position of men with minimal disablement:

iv. At the time of the review, benefit was only payable for pneumoconiosis when disablement was quantified at 5% or more. IIAC argued that that should be modified and that a diagnosis of pneumoconiosis should automatically lead to an IIDB award:

"..*we think it reasonable to suppose that a person in whom the disease is diagnosed will almost always be disabled by it to the extent of one per cent or more*".

21. At that time awards were made for loss of earnings and the principal effect of the 1953 proposal was to allow underground miners with early-stage pneumoconiosis who were at risk of progression to be moved to surface work where exposures were lower without suffering any overall loss of income. The reduced earnings provision was abolished in 1994.

22. IIAC went on to argue that it was not possible to measure disablement by pneumoconiosis in steps of less than 10%, and so 1% disablement should automatically be upgraded to 10%, and 11% to 20%. The effect was that anyone with a diagnosis of pneumoconiosis would automatically receive a minimum 10% disablement award.

23. *The 1967 National Insurance (Industrial Injuries) Amendment Act* enabled the effects of COPD ("emphysema and chronic bronchitis") to be treated as if they were the effects of pneumoconiosis for those with of disability assessments of 50% or more. This was an early recognition that coal mine dust could cause COPD. COPD in coal miners became a separate prescribed disease in 1992.

24. The 1967 National Insurance (Industrial Injuries) Amendment Act also allowed the effects of tuberculosis to be taken into account.

25. IIAC reviewed the prescription of pneumoconiosis in 1973 following a request from the Minister for Social Security *(Cmd 5443: Pneumoconiosis and Byssinosis).* The Council noted that there had been considerable developments since 1953 in medical knowledge of the disease, in diagnostic methods and in working practices. The growing awareness of

the issue of COPD in coal miners was a particular concern; "…*special problems arose in assessing the extent of pneumoconiosis when found in conjunction with other respiratory conditions*".

26. The main conclusions and recommendations of the 1973 review were:

    - A revised definition of pneumoconiosis to specifically exclude COPD "*pneumoconiosis means permanent alteration of lung structure due to the inhalation of mineral dust and the tissue reactions of the lung to its presence but does not include bronchitis and emphysema*".

    - Simple pneumoconiosis in coal miners should not generally be considered to be a cause of disability. The effects of silicosis were held to be usually more severe than those of coal workers' pneumoconiosis.

    - The mere presence of pneumoconiosis should not automatically imply disability; "…*we cannot support the conclusion reached in 1953 that virtually everyone in whom pneumoconiosis is diagnosed ought to have an award of disablement benefit*"

    - Assessment of disability from pneumoconiosis should continue to be in increments of 10%.

    - Account should continue to be taken of the effects of coexistent tuberculosis and COPD ('chronic bronchitis and emphysema') in those with disability assessments of 50% or more.

27. IIAC undertook a review of asbestos-related diseases in a *1996 Command Paper (Cmd 3467 Asbestos Related Diseases)*. No changes were recommended to the prescription in relation to asbestosis.

28. IIAC reviewed asbestos-related diseases again in *2005 (Cmd 6553: Asbestos-related diseases).* The Council took the view that CT scans should be used to support a diagnosis of asbestosis when available but should not be necessary for diagnosis. At the time it was considered to be standard clinical practice for patients to be assessed initially by plain chest radiograph and only a proportion would subsequently be assessed by CT. The Council noted that it would reconsider the recommendation if in the future CT scans become universal in these investigations. No changes were recommended to the prescription.

29. IIAC considered the issue of interstitial fibrosis in coal miners in *2006 (Position paper 17: Interstitial Fibrosis in Coalworkers)*. There was no

evidence available that would allow prescription of diffuse interstitial fibrosis separate from coal workers' pneumoconiosis.

30. The Council addressed the issue of presumption i.e. when a condition can be presumed to have been caused by work without the need for additional evidence in 2014 *(Cmd 8880 Presumption that a disease is due to the nature of employment: coverage and time rules)* and 2015 *(Cmd 9030 Presumption that a disease is due to the nature of employment: the role of rebuttal in claims assessment)*. The Council noted that in most cases PD D1 was presumed to have been caused by work if there had been more than 2 years exposure in aggregate to the relevant exposure. Claims under the Open Category (13) do not have the benefit of presumption.

The Current Prescription

The prescribed disease

31. Pneumoconiosis is the only occupational disease that is defined within the Social Security Act. It is taken to be:

> '*Fibrosis of the lungs due to silica dust, asbestos dust, or other dust and includes the condition of the lung known as dust reticulation'.*

The requirement for fibrosis excludes the so-called benign pneumoconioses such as siderosis or baritosis which are generally not considered to cause disability. It also excludes conditions such as COPD that are not generally considered to be pneumoconioses. It is not entirely satisfactory. Acute silicosis for example is associated with the pathological appearances of alveolar proteinosis without necessarily involving fibrosis. Fibrosis is also not necessarily a feature of the early stages of coal workers pneumoconiosis.

32. The reference to 'dust-reticulation' was introduced in the Workmen's Compensation Act of 1943 and reflected the contemporary understanding of the appearance of irregular opacities on chest radiographs. These were taken to indicate the pathological features of early coal workers' pneumoconiosis:

> "…*a widespread though not necessarily great increase in the reticular connective tissue in the lungs which results from the accumulation of dust particles".*

33. At the time the pathological appearance of simple coal workers' pneumoconiosis was also referred to as 'reticulation', but the terminology was confusing and was never generally adopted. The difficulty with the terminology was recognised by the time of IIAC's 1973 command paper. It was recommended that the reference to reticulation should be removed

from the definition of pneumoconiosis, but the recommendation was not implemented.

The occupation

34. The current prescription (Appendix 1) includes 12 scheduled job categories and 14 sub-categories. Eight of the categories refer to work with silica; one to work with asbestos; one to mine work; and 3 other categories relating to the grinding of mineral graphite, the manufacture of carbon electrodes, and boiler scaling. There is a final open category (13) - '*exposure to dust if the person employed in it has never at any time worked in any of the other occupations listed*.'

35. The categories set out in the current schedule of occupations/exposures are in large part historic and refer back to compensation schemes that predate the 1946 National Insurance Act. Most relate to silica exposures which are now relevant to only a small proportion of claimants and they do not fully reflect current populations at risk. They include processes such as sandblasting, which, although it is likely still to occur, has not been in legal use in the UK for several decades. Other processes such as coal trimming no longer occur. Whilst disease arising from these processes should still be covered by a revised prescription it is no longer necessary for them to be specifically mentioned in the schedule of occupations. The complexity of the current schedule has the potential to inhibit potential applicants, and IIAC has anecdotal evidence of the failure of clinicians to recognise that some more novel conditions or circumstances of exposure are included within the regulations.

Awards of benefit

36. PD D1 is unusual in that benefit can be awarded if the condition is deemed to be present irrespective of whether or not it is causing any disability. The reasons for that are historic as discussed above. Similar arrangements apply to byssinosis and diffuse mesothelioma.

37. PD D1 is also unusual in that the effects of the comorbid condition COPD can be taken into account in assessing benefit, as can complicating tuberculosis. COPD is now generally treated as a separate condition and is prescribed in coal miners as PD D12. IIAC is undertaking a separate review of COPD associated with other occupational exposures. Tuberculosis is now much less common and is more easily treatable than it was in 1967 when provision was made for it in assessing disability from pneumoconiosis. On the other hand, non-tuberculous mycobacterial infection is more commonly diagnosed. Both are recognised complications of silica exposure but not of other causes of pneumoconioses.

Recommended Changes to PD D1

The prescribed disease

38. The Council recognises that there is no generally agreed definition of pneumoconiosis that is suitable for the purpose of prescription. The most widely used is that of the International Labour Organisation: "*Pneumoconiosis is the accumulation of dust in the lungs and the tissue reactions to its presence*." (ILO 1998). Although not stated explicitly in this definition, the condition is generally taken to exclude malignant disease and airway diseases such as asthma and COPD.

39. The definition of pneumoconiosis in the Social Security Act makes reference to fibrosis of the lungs which is not present in all forms and at all stages of pneumoconioses, though in the majority of cases substantial disablement will only occur when there is lung fibrosis. In the vast majority of cases a diagnosis of pneumoconiosis is based on radiological features together with the exposure history and the pathological features are assumed rather than demonstrated.

40. The current definition also makes reference to dust reticulation, but that terminology is considered to be of historic interest only, potentially confusing, and no longer relevant.

41. The Council recommends that the disease considered in the prescription should be '*pneumoconiosis*' without any further qualification. The term will be understood by respiratory specialists, and a number of definitions and descriptions are available in relevant textbooks such as:

> *"Pneumoconiosis may be defined as the non-neoplastic reaction of the lungs to inhaled mineral or organic dust and the resultant alteration in their structure but excluding asthma, bronchitis, and emphysema*" (Parkes 1994)
>
> *"Pneumoconiosis is a generic term that is used to describe the lodgement of any inhaled dust in the lungs and its effects, excluding asthma and neoplasia*". (Newman Taylor et al 2016)
>
> *"Pneumoconiosis is a term used for any non-neoplastic disease of the lungs caused by the habitual inhalation of mineral or metallic particles or dusts excluding asthma, bronchitis or emphysema"*. (Hendrick et al 2002)

42. It is anticipated that in the majority of cases the diagnosis of pneumoconiosis will be established by a respiratory or related specialist. This will normally be a respiratory consultant working in the NHS. All respiratory specialists should undergo training in occupational lung diseases and a work history should form part of the normal clinical evaluation when there is a possibility of an occupational lung disease.

There is a network of occupational respiratory centres (GORDS[1]) that can offer advice on individual cases or undertake assessments when there is diagnostic doubt. IIAC takes the view that a diagnosis of pneumoconiosis normally requires specialist investigation to confirm the presence of diffuse lung disease and exclude any alternative possible diagnoses. There might be exceptional cases where a specialist assessment is not possible and allowance will be made for that.

43. Notes on the diagnoses of the individual pneumoconiosis are appended (Appendix 2).

44. The Council notes that a diagnosis of pneumoconiosis implies an environmental, and almost always an occupational, cause for the disease. It might be thought that no further clarification or restriction is needed. However, a list of substances/exposures which are likely to meet the terms for prescription will offer some guidance to potential claimants.

45. Occasionally a diagnosis of pneumoconiosis will be made from pathological rather than radiological findings. That is most likely to occur in posthumous claims. No change is envisaged in the pathological features or criteria necessary to establish a diagnosis.

The occupation

46. It is proposed that individual circumstances relating to the tasks and tools used as set out in the 12 categories of the current prescription (appendix 1) should no longer be specified. Many of these are of little more than historic interest but they reduce the clarity of the prescription and potentially inhibit applicants with disease caused by modern working practices or exposures.

47. The Council considers that work involving exposures to the following should be included as potential causes of pneumoconiosis for the purpose of PD D1:

    1. Asbestos
    2. Coal or coal mine dust
    3. Silica-containing dusts
    4. Metals: aluminium, beryllium, cobalt, indium tin oxide, rare earth metals, and tungsten carbide

48. It is not proposed that there should be any change to the extent of exposure in terms of frequency, duration or intensity necessary to establish a diagnosis for any of the pneumoconioses.

[1] https://www.hsl.gov.uk/centreforworkplacehealth/gords/contact-information

49. The revised prescription no longer makes specific mention of asbestos textiles, machinery or plant as workplaces or exposures likely to give rise to asbestosis. All asbestos exposures of sufficient extent and whatever their source are now equally included in the generic term 'any job involving exposure to asbestos'.

50. Likewise references to specific jobs, tasks or workplaces that involve exposure to silica are replaced by the term 'any job involving exposure to silica-containing dusts. It is hoped that this simplification in particular will assist with the recognition of silicosis caused by newer work techniques or exposures as a prescribed condition.

51. Any pneumoconiosis arising from boiler scaling is likely to be a consequence of contaminating silica and should be considered under that category. Likewise, any pneumoconiosis arising from the grinding of mineral graphite or the manufacture of carbon electrodes, insofar as the condition still exists in the UK, should be considered to be a consequence of contaminating silica.

52. Relatively few awards are currently made under the open category (category 13 "*Exposure to dust if the person employed in it has never at any time worked in any of the other occupations listed*"). It requires the claimant never to have worked in any of the 12 scheduled categories. With the decline of lifelong employment within a particular industry and the increased mobility of the workforce this is likely to lead to anomalies. Thus, for example, if someone worked with asbestos (category 9) and then developed silicosis as a consequence of work in an unscheduled industry they would be ineligible for benefit under the current terms of the prescription.

53. There are no guidelines about which exposures can be included within the open category. The Department for Work and Pensions (DWP) is required to decide not only whether a particular exposure has caused an individual's disease but also whether or not the exposure in question is capable of causing disease. As pneumoconioses are almost always diseases of long latency, there are no clinical features such as more severe symptoms on days of exposure on which to base that decision. It relies on epidemiological evidence of a doubled risk associated with the exposure which is normally an issue for IIAC itself rather than for the DWP to determine.

54. It is recommended that the open category be removed and that if any new causes of pneumoconiosis become recognised these should be assessed by IIAC and a decision made about their inclusion in the schedule of prescribed exposures.

55. Guidance about the nature and extent of exposures likely to attract an award of IIDB are appended (Appendix 2).

Award of benefit

56. It is not proposed that any changes be made to the extent of exposure necessary to allow a diagnosis of pneumoconiosis to be made, or in the assessment of disability.

57. The Council recommends that a claimant's condition should be presumed to be due to work in one of the categories listed below if they have a diagnosis of pneumoconiosis or of a form of pneumoconiosis (e.g., asbestosis, silicosis etc) established by an appropriate specialist. The Council notes that on rare occasions the disease can develop within a short period of the onset of exposure and recommends that presumption should no longer be restricted to those with a cumulative duration of work of two years or more.

58. A diagnosis of pneumoconiosis should not automatically lead to an award of benefit irrespective of the degree of disability. The award should be based on the extent of disability. This should be quantified as it is at present on an individual basis taking into account all available information including symptoms, lung function, radiological features, and co-morbid conditions. If appropriate, it can be added to awards for other prescribed diseases.

59. COPD associated with coal dust is now addressed in a separate prescription and does not need to be included in disability awards for pneumoconiosis.

60. Tuberculosis and non-tuberculous mycobacterial infection are recognised complications of silica exposures and should be taken into account in disability assessments in those with pneumoconiosis caused by silica-containing dusts. Tuberculosis or non-tuberculous mycobacterial infection developing in association with other forms of pneumoconioses are likely to be coincidental.

Conclusions and Recommendations

61. The Council recommends that the disease considered in the revised prescription should be 'pneumoconiosis' without any further qualification and that work involving exposures to the following should be included as potential causes of pneumoconiosis for the purpose of PD D1. They are set out in the table below:

Name of disease	Type of job – Any job involving exposure to:
Pneumoconiosis	1. Asbestos 2. Coal or coal mine dust 3. Silica-containing dusts 4. Metals: aluminium, beryllium, cobalt, indium tin oxide, rare earth metals and tungsten carbide.

62. The Council also recommends that:

- Awards of IIDB for pneumoconiosis should reflect the degree of disability and should not be made automatically if there is no associated disability.
- A claimant's condition should be presumed to be due to work in one of the categories listed above if they have a diagnosis of pneumoconiosis or of a form of pneumoconiosis established by an appropriate specialist.
- The open category be removed and that if any new causes of pneumoconiosis become recognised these should be assessed by IIAC and a decision made about their inclusion in the schedule of prescribed exposures.

Prevention note

63. The Health and Safety Executive was consulted for advice on prevention and the following contribution was supplied.

64. The risks of pneumoconiosis due to asbestos fibres or respirable dust – including coal, coal mine dust, silica or metal containing dusts – should be mitigated by ensuring that workers are not exposed or that work is carried out in a way that minimises exposure.

65. The Control of Substances Hazardous to Health Regulations 2002 (COSHH) place robust and well-established requirements on employers to control exposure of their workers to substances hazardous to health, including dusts that can cause pneumoconiosis. COSHH applies in full in mines, and the Mines Regulations 2014 place additional duties on mine operators with respect to the control and measurement of inhalable and respirable dust underground. Work with asbestos is regulated specifically under the Control of Asbestos Regulations 2012 (CAR).

66. The COSHH and CAR regulations have equivalent duties. They require substances hazardous to health, such as respirable crystalline silica and asbestos, to be identified and risk assessed with effective management arrangements then put in place. Under both, the emphasis is on

avoidance, containment, or the application of good control practice, in that order. Both have similar duties for training workers, monitoring exposures and the requirement for either health or medical surveillance. There are additional specific requirements for work with asbestos, including licensing and a duty to manage asbestos materials in public buildings.

67. Under COSHH, where it is not reasonably practicable to prevent exposure by substitution with a less hazardous substance, exposure to respirable crystalline silica must be reduced as low as reasonably practicable and the workplace exposure limit (WEL) of 0.1 mg/m^3 must not be exceeded. Exposure must be reduced using appropriate work processes, systems and engineering controls. These include measures such as dust suppression and local exhaust ventilation, that control exposure at source. Suitable respiratory protective equipment (RPE) is the last control measure option to be considered and may be used in combination with other measures when adequate control cannot be otherwise achieved. The risk assessment should show where there is a need to introduce health surveillance procedures. Where workers are regularly exposed to respirable crystalline silica and there is a reasonable chance that silicosis may develop, health surveillance must be provided. Specific guidance on health surveillance in relation to work with silica is available on the HSE website.

68. The importation, supply, and use of asbestos in Britain has now been banned, but asbestos was extensively used as a building material up to the late 1970s. Those currently at risk of exposure to asbestos fibres include people who remove asbestos containing materials and building and maintenance workers who may unknowingly be exposed during the course of their work. The CAR regulations require an assessment of risk before any work with asbestos-containing materials, the use of appropriate control measures to reduce exposure and the provision of RPE. People who work with asbestos may be required to have health examinations. There is a requirement to obtain a license or to notify the Health and Safety Executive before starting some types of work with asbestos.

69. Advice about good practice control measures for different work circumstances, including advice on air sampling and health surveillance, is freely available on the Health and Safety Executive's website.

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Appendix 1

Current PD D1 prescription

Disease Number	Name of disease or injury– Miscellaneous conditions not included elsewhere in the list	Type of job – Any job involving:
D1	Pneumoconiosis. Includes silicosis and asbestosis.	(1) (a) The mining, quarrying or working of silica rock or the working of dried quartzose sand or any dry deposit or dry residue of silica or any dry admixture containing such materials (including any occupation in which any of the aforesaid operations are carried out incidentally to the mining or quarrying of other minerals or to the manufacture of articles containing crushed or ground silica rock); (b) The handling of any of the materials specified in the foregoing subparagraph in or incidental to any of the operations mentioned therein, or substantial exposure to the dust arising from such operations. (2) The breaking, crushing or grinding of flint or the working or handling of broken, crushed or ground flint or materials containing such flint, or substantial exposure to the dust arising from any such operations. (3) Sand blasting by means of compressed air with the use of quartzose sand or crushed silica rock or flint, or substantial exposure to the dust arising from sand and blasting. (4) Work in a foundry or the performance of, or substantial exposure to the dust arising from, any of the following operations: (a) the freeing of steel castings from adherent siliceous substance; (b) the freeing of metal castings from adherent siliceous substance: (i) by blasting with an abrasive propelled by compressed air, by steam or by a wheel, or

(ii) by the use of powerdriven tools.

(5) The manufacture of china or earthenware (including sanitary earthenware, electrical earthenware and earthenware tiles), and any occupation involving substantial exposure to the dust arising therefrom.

(6) The grinding of mineral graphite, or substantial exposure to the dust arising from such grinding.

(7) The dressing of granite or any igneous rock by masons or the crushing of such materials, or substantial exposure to the dust arising from such operations.

(8) The use, or preparation for use, of a grindstone, or substantial exposure to the dust arising therefrom.

(9) (a) The working or handling of asbestos or any admixture of asbestos;
(b) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos;
(c) the cleaning of any machinery or plant used in any foregoing operations and of any chambers, fixtures and appliances for the collection of asbestos dust;
(d) substantial exposure to the dust arising from any of the foregoing operations.

(10)(a) Work underground in any mine in which one of the objects of the mining operations is the getting of any mineral;
(b) the working or handling above ground at any coal or tin mine of any minerals extracted therefrom, or any operation incidental thereto;
(c) the trimming of coal in any ship, barge, or lighter, or in any dock or harbour or at any wharf or quay;
(d) the sawing, splitting or dressing of slate, or any operation incidental thereto.

(11) The manufacture of carbon electrodes by an industrial undertaking for use in the electrolytic extraction of aluminium from

aluminium oxide, and any occupation involving substantial exposure to the dust arising therefrom.

(12) Boiler scaling or substantial exposure to the dust arising therefrom.

(13) Exposure to dust if the person employed in it has never at any time worked in any of the other occupations listed.

Appendix 2

Notes on the individual pneumoconiosis

(a) Asbestos

Asbestosis is diffuse lung fibrosis caused by one of the commercial forms of asbestos, principally chrysotile (white asbestos), and the amphibole mineral fibres amosite (brown asbestos) and crocidolite (blue asbestos). Radiologically it is characterised by a Usual Interstitial Pneumonia (UIP)–like pattern with subpleural, basal-predominant reticular abnormalities that are often associated with 'honeycomb' change and traction bronchiectasis.

Extensive UK exposures to asbestos occurred throughout much of the 20th century in the industrial manufacturing of products containing asbestos, insulation of boilers and other equipment, in shipbuilding, in onshore construction, and in many other industries. Jobs that involved heavy exposure to asbestos in the past included shipyard workers, plumbers, carpenters, electricians, construction workers, metal workers and many others.

In the first part of the 20th century exposures to asbestos could be very high and, for example, in shipbuilding in the 1960s concentrations up to 500 fibres/ml were recorded when removing asbestos insulation that had been sprayed onto steel structures. Applying or removing asbestos insulation could generate concentrations up to 100 fibres/ml.

Some example asbestos exposures expressed as number of fibres per ml of air (HSE 2006) are:

Stripping sprayed coatings	around 1000 fibres/ml
Stripping pipe or vessel lagging	up to 100 fibres/ml
Power sawing asbestos insulation board	up to 20 fibres/ml
Drilling asbestos insulation board without a vacuum trace	up to 10 fibres/ml
Hand sawing asbestos insulation board	5-10 fibres/ml
Careful removal of whole asbestos insulation board	up to 3 fibres/ml
Controlled wet stripping using manual tools	up to 1 fibres/ml
Drilling asbestos insulation board with dust extraction	up to 1 fibres/ml

A diagnosis of asbestosis depends primarily on the exposure history as the radiological appearances are identical to those of the idiopathic condition Idiopathic Pulmonary Fibrosis, and to lung fibrosis associated with connective tissue disorders such as rheumatoid arthritis or caused by some therapeutic drugs. In rare cases and usually at post-mortem the diagnosis is established from the pathological features and evidence of asbestos exposure in the form of asbestos bodies or an elevated asbestos fibre count.

Asbestosis generally develops after relatively heavy exposures in comparison with those needed to cause pleural plaques or mesotheliomas. There is no established threshold exposure necessary to cause asbestosis, but the risk increases as the extent of exposure increases. Asbestos fibres can persist in the lungs after exposures cease and asbestosis can first become clinically evident several decades after exposures cease.

The typical radiological features of asbestosis are of predominantly basal, peripheral, diffuse lung fibrosis. Its presence should normally be established or confirmed using CT scanning. Pleural plaques are often present and indicate some prior asbestos exposure though not necessarily sufficient to cause asbestosis. Localised fibrosis or focal abnormalities such as rolled/rounded atelectasis are not those of asbestosis.

Functional impairment in asbestosis generally increases in relation to the extent of radiological shadowing, and minor degrees of asbestosis as identified on a CT scan do not necessarily lead to disability.

(b) Coal/ coal mine dust

Coal can be mined by underground workings or from surface deposits. At its peak in the 1920s there were more than a million men employed in coal mining. However, the industry was in steady decline during the 20th Century and by the mid-1990s the industry had mostly closed in Britain.

Coal mine dust is a complex mixture of minerals, which in addition to carbon may include kaolin, mica and silica (quartz). Past exposures in underground mining were heavy but have generally decreased in recent decades. During the 1940s exposures in British coal mines sometimes exceeded 100 mg/m^3. (Bedford and Warner, 1943) but by the 1980s exposure to airborne respirable dust concentrations were mostly less than 6 mg/m^3 (Hurley et al 1982). Exposure to respirable dust was much lower in surface opencast mining than underground, generally below 1 mg/m^3. Workers in other industries, such as in coalfired power stations or docks processing imported coal, may also have been exposed to respirable coal dust (Love et al, 1997).

Coal worker's pneumoconiosis refers to the accumulation of coalmine dust in the lungs accompanied by radiological diffuse nodular abnormalities and typical pathological features. In the earlier stages coal macules are formed around the terminal bronchioles in the lungs. These can later coalesce to form the conglomerate abnormalities of progressive massive fibrosis.

The risk of the development of coal worker's pneumoconiosis is related to the extent of exposure. There is no established threshold exposure below which there is no risk although typically exposure durations of less than 10 years do not cause disease (Newman Taylor et al 2017).

A diagnosis of coal worker's pneumoconiosis is based on the history of exposure, the radiological pattern of disease, and the exclusion of other conditions with a similar radiological appearance. In rare cases the diagnosis is based on pathological features obtained from a lung biopsy or post-mortem examination.

The radiological features of coal worker's pneumoconiosis are of predominantly upper zone nodules with or without larger conglomerate shadows. The appearances are similar to those of silicosis and the two conditions can be difficult to distinguish radiologically. The idiopathic condition sarcoidosis and some other diseases can cause a similar radiological pattern but should be easily distinguished by the occupational history. UIP-pattern diffuse pulmonary fibrosis (similar to that of asbestosis) is not considered to be a feature of coal worker's pneumoconiosis as discussed in the 2006 IIAC Position Paper (Position Paper 17: *Interstitial Fibrosis in Coal workers*).

Simple coal workers' pneumoconiosis (where all the abnormalities are <1 cm in diameter) generally does not give rise to symptoms or cause disability (Parkes 1994). A diagnosis of coal workers' pneumoconiosis does not in itself necessarily imply any disability.

(c) Silica-containing dusts

Silica (silicon dioxide) is a common mineral that makes up about one quarter of the earth's crust. It can exist in crystalline form, amorphous forms, or combined with other minerals to form silicates.

Silicosis

Silicosis is a disease caused by exposure to respirable crystalline silica (RCS) mostly in the form of quartz but sometimes as tridymite or cristobalite which have different crystalline structures. The crystalline structure is important for the toxicity of silica, and amorphous forms as for example found in glass are much less harmful.

RCS is much more toxic than many other mineral dusts and is controlled more stringently. The workplace exposure limit (WEL) for silica is 0.1 mg/m^3 expressed as an 8-hour time-weighted average. That compares with an exposure limit of 4 mg/m^3 for low toxicity dusts.

High RCS exposure levels have been reported for many groups of workers in the UK and there has generally been poor compliance with the legal limits on occupational exposure. For example, in the 1990s RCS levels for specific work tasks in the construction industry were generally above 0.1 mg/m^3, with the highest levels reported for dry work with hand-held power tools (up to 7 mg/m^3) (Chisholm 1999). Average exposures would have been lower,

probably mostly between 0.1 and 0.5 mg/m^3. There has been poor use of respirators throughout industry to protect workers from RCS exposure.

Historically silicosis developed amongst workers in industries such as mining, quarrying, construction, foundries, sandblasting and ceramic manufacture. More recently silicosis has been reported in a variety of novel settings such as textile or artificial composite stone processing. The UK Work-related and Occupational Respiratory Disease (SWORD) scheme has reported cases in other non-traditional settings such as in dental technicians and jewellery manufacturing (Barber et al, 2019). The possibility of disease emerging in other novel settings remains a concern. Leung (2012) details jobs and work practices that can potentially give rise to silica exposures.

Three main forms of silicosis are recognised. These are primarily related to the intensity/extent of exposure which in turn determines the speed at which the disease develops:

An acute form with features similar to alveolar proteinosis that develops rapidly within weeks to a few years in those who are exposed to large amounts of RCS over a relatively short period (Leung et al 2012). It is associated with diffuse intrapulmonary shadowing of predominantly 'ground glass' density on CT scanning. The typical pathological appearance is of interstitial inflammation (alveolitis) and filling of the alveoli with denatured surfactant. The appearances are identical to those of the idiopathic condition alveolar proteinosis.

Chronic silicosis is more common has a characteristic appearance on biopsy with densely whorled fibrotic nodules. Radiologically it is characterised by small, rounded nodules with a posterior and upper zone predominance (simple silicosis) and confluent upper zone shadowing (progressive massive fibrosis).

A more rapidly progressive 'accelerated' form of silicosis can develop after a few years of relatively heavy exposure.

Cumulative exposure to RCS is the most important factor in the development of chronic silicosis but other characteristics can be important. Freshly fractured silica particles are more toxic than less fresh. The disease usually only develops after 10 years or more of exposure if dust concentrations are less than 1 mg/m3 but occasional cases can occur with exposures within statutory limits. It is often difficult to determine the extent of an individual's exposure and there are no firm guidelines about the threshold exposure that allows a diagnosis of silicosis to be established. A diagnosis of silicosis depends on a history of exposure to RCS and compatible radiological or pathological features together with the exclusion of any alternative more likely diagnoses.

Mild degrees of simple silicosis (where all the abnormalities are <1 cm in diameter) do not necessarily cause symptoms or abnormalities of lung function. More extensive disease, particularly when there is progressive massive fibrosis. is likely to be associated with disability. Progressive massive fibrosis can be associated with localised emphysema which can be considered to be part of the pneumoconiosis. More generalised emphysema/ chronic obstructive pulmonary disease is considered to be a separate condition.

Silicosis is associated with an increased risk of tuberculosis and of non-tuberculous mycobacterial infection, and when these develop their effects can be considered in the assessment of disability.

Mixed mineral dust fibrosis

Typical silicosis only develops after exposure to relatively pure RCS. When the RCS is mixed with other non-fibrogenic dusts and forms less than about 15% of the overall dust content a condition with different pathological appearances – mixed mineral dust fibrosis – can develop (Honma et al 2004). It is likely that the presence of the other dusts modifies the effects of the silica rendering it less toxic (Donaldson and Borm 1996).

Occupations that have been associated with mixed mineral dust pneumoconiosis include haematite mining, foundry work, pottery and ceramic work, stonemasonry, and jobs involving exposure to concrete dust. In haematite mining in the 1960s in the UK, respirable dust exposure levels were around 2 mg/m.

Pathologically, mixed mineral dust fibrosis is characterised by macules around the terminal bronchioles similar to those seen in coal worker's pneumoconiosis, with or without typical silicotic nodules. Radiologically, a variety of patterns are described including diffuse nodular and reticular shadowing, and progressive massive fibrosis. The condition is much less well characterised than silicosis, asbestosis and coal worker's pneumoconiosis.

Non-fibrous silicate pneumoconiosis

Silicates are a group of minerals formed from silica with a variety of other metals in their crystalline structure. They include minerals that form sheet-like crystals such as talc and mica, clays such as vermiculite, bentonite, and kaolinite (china clay), and fibres such as asbestos. Asbestos is generally considered separately from non-fibrous silicates because of its markedly greater fibrogenicity.

Traditionally, talc, mica and kaolin have been the most important of the non-fibrous silicates. Talc is still widely used in the manufacturing of rubber and paper, and in cosmetics. Historically, exposures were sometimes high,

ranging up to 30 mg/m^3 amongst production workers in French and Austrian plants (Wild et al 1995). Mica is used in electrical and electronics industries because of its insulating properties, as a filler and thermal insulator in construction, and in paints and plastics. In the past more than half of the world's kaolin (china clay) was mined in Cornwall. It is used in the filling and coating of paper, in the ceramics industry, and as a filler for rubber, paints, plastics and other substances.

While there is evidence that several non-fibrous silicates are associated with pneumoconiosis their potency overall appears to be less than that of RCS, and the adverse effects might in many cases be due to contaminating RCS. In the case of talc, some cases are described following domestic exposures, and a related condition is seen in intravenous substance abusers when the injected material is mixed with talc.

There is no consistent radiological pattern reported in cases of pneumoconiosis associated with non-fibrous silicates. The diagnosis depends on a history of sufficient exposure and the exclusion of other causes. The work is likely to have been for at least 10 years in environments with dust levels exceeding 1 mg/m^3, or the equivalent.

Metals

Aluminium

In the past the inhalation of aluminium powder was considered to be beneficial and was used to prevent or treat other pneumoconioses. Shaver's lung was described in the 1940s in the manufacture of the abrasive corundum but was probably a form of acute silicosis (Hendrick 2002). Further cases were described in the manufacture of aluminium pyrotechnic flake used for explosives in the 1940s. Occasional cases have been reported since then but reports of aluminium pneumoconiosis are relatively rare in relation to the frequency with which the metal is used (Newman Taylor et al 2016).

Beryllium

Beryllium (atomic number 4) is the lightest metal, is more rigid than steel, and is an excellent conductor of heat and electricity. As such it has a variety of uses in the aerospace, nuclear, defence, automotive, electronics and telecommunications industries. It is also used for example in dental prostheses and sports equipment. Exposures can occur in mining, smelting, manufacture, and recycling. Darby and Fishwick (2011) provide further details of the uses and applications of beryllium.

With acute heavy exposures beryllium can cause chemical pneumonitis and other upper respiratory problems.

Chronic beryllium disease (CBD) arises because of sensitization to beryllium with the development of CD4+ve T lymphocytes that react to the metal. Up to 20% of highly exposed workers have been reported to develop sensitization with genetic factors contributing to the risk (Balmes et al 2014).

Not all workers who become sensitized to beryllium go on to develop disease. The proportion has varied between 10% and 100% in a number of studies. Typically, disease develops between 10 and 20 years after first exposure but it has been reported to occur within months.

The key pathological feature of CBD is the presence of non-caseating granulomas identical to those seen in sarcoidosis. Beryllium particles are sometimes found within the granulomas, but this is not a consistent feature of the condition.

The lung is the organ primarily affected in CBD with insidious onset breathlessness, fatigue, cough, and chest discomfort. The chest radiograph features are similar to those of sarcoidosis, with predominantly mid- to upper-zone nodules, and mediastinal or hilar lymphadenopathy. CT scans are much more sensitive than chest radiographs in detecting early disease. Nodules are the most common finding, often clustered around bronchi, within interlobular septa, or in the subpleural region. Ground-glass opacities, bronchial wall thickening, and thickening of interlobular septa are also seen. A variety of lung function abnormalities are reported including airflow obstruction, restriction, and isolated impairment of gas transfer.

Because the appearances of CBD can be identical to those of sarcoidosis the diagnosis can easily be missed. After obtaining an occupational history an initial diagnosis of sarcoidosis has been revised to CBD in up to 40% in some studies (Muller-Quernheim et al 2016). The key diagnostic test is a lymphocyte proliferation assay carried out on peripheral blood or bronchoalveolar lavage fluid.

Rare earth metals/ Lanthanides/ Cerium

There are 17 rare earth metals (also known as lanthanides), the most abundant of which is cerium. They have important uses as catalysts, in high performance magnets used in electric motors or turbines, electronics and alloys. The use of these metals has increased markedly in recent decades.

There are fewer than 20 case reports in the medical literature of lung disease attributed to rare earth metals, mostly in carbon arc lamp operators, photoengravers and lens polishers (Sulotto et al, 1986). In most cases the patten of disease is of diffuse lung fibrosis though granulomatous disease has been described (Nemery 1996). The metals persist in the lung following exposure and the extent to which disease is caused by the exposure or is

coincidental can be unclear. No cases have yet been described in the UK though some potential for disease might exist for exampling in the recycling industry.

Indium tin oxide

Indium tin oxide is widely used as a coating in display screens, smart windows and the like on account of its electrical conductivity, optical transparency and ease of application as a thin film.

Fewer than 20 cases of indium lung have been reported in the medical literature (Chonan et al 2019). The commonest abnormalities are pulmonary alveolar proteinosis and pulmonary fibrosis with traction bronchiectasis and 'honeycomb' change. No cases have been reported in the UK but there is possibly some potential for exposure for example in the recycling industry.

Cobalt/ tungsten carbide/Hard metal disease (Cobalt Related Interstitial Lung Disease)

Hard metal is produced by heating a mixture of tungsten carbide, 5%-10% cobalt, and other metals including tantalum, titanium, nickel, niobium, and chromium to high temperature. The resulting material has a hardness that is only slightly less than that of diamond and is resistant to high temperatures. These properties make it ideal for use in tools designed to machine, grind, drill or cut metals or rock. Hard metal can also be used to coat softer metals to increase their durability. Exposures to cobalt and other metal particles occur particularly during the final steps in hard metal tool production such as grinding, polishing, machining, and coating.

Cobalt in hard metal rather than the metal itself is the likely cause of the pneumoconiosis. Diamond polishers use high-speed grinding tools that have their polishing surfaces cemented in a metal matrix that can contain up to 90% cobalt by weight. Their use is associated with a condition similar to hard metal disease (Demedts et al 1984). For that reason, it is often appropriate to use the more general term Cobalt Related Interstitial Lung Disease (CRILD) rather than hard metal disease.

The commonest pathological feature of CRILD is giant cell interstitial pneumonia (GIP) with intra-alveolar macrophages, multinucleated giant cells that are cannibalistic of other cells, an interstitial mononuclear cell infiltrate (Choi et al 2005) and fibrosis that is typically airway-centred (Adams et al 2017). Approximately 10-20% of subjects have features of diffuse lung fibrosis with a usual interstitial pneumonia (UIP)-type pattern or with 'honeycomb' features. Similar numbers of subjects have a UIP-type pattern of fibrosis without GIP. There are other less commonly reported pathological appearances including those of hypersensitivity pneumonitis. desquamative

interstitial pneumonia, nonspecific interstitial pneumonitis, and granulomatous lung disease. Because of their relative rarity and their occurrence in other contexts the nature of the association between these conditions and CRILD is less certain.

CRILD can develop following apparently low level exposures (Sprince et al 1994), suggesting that that immune hypersensitivity might play some role in its pathogenesis. Genetic susceptibility to CRILD has been reported in the form of an association with HLA-DP glutamate 69 which in turn is associated with cobalt binding and greater metal uptake (Potolicchio et al 1997).

The occupational history is key to the recognition of CRILD. Patients present with subacute or chronic breathlessness that is associated with radiological 'ground-glass' shadowing or a UIP-type lung fibrosis. Typical pathological features can be found on lung biopsies.

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