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THE INDUSTRIAL INJURIES ADVISORY COUNCIL

POSITION PAPER 23

Pleural Plaques

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Pleural Plaques

Position paper 23

Summary

1. On the 11th June 2008, the Secretary of State for Work and Pensions referred the question of pleural plaques to the Industrial Injuries Advisory Council (IIAC) under Section 171(3) of the Social Security Administration Act 1992.

2. The Council was asked to consider the current prevalence of pleural plaques, the occupational causation of pleural plaques currently found in the population, the likelihood of disability arising from pleural plaques, the likelihood of other more severe complications of asbestos exposure arising amongst those currently having plaques, and whether compensation through the Industrial Injuries Scheme would be appropriate for people diagnosed with this condition.

3. The Council referred the matter to its permanent sub-committee, the Research Working Group, which conducted a literature search covering some 200 pages of research abstracts and some 75 key original research reports, consulted with leading experts in the field of respiratory research and asbestos-related diseases, and made calls for

evidence on its website and through the news mailing of a specialist occupational medical society.

4. The inhalation of asbestos can cause disease in the lung tissues themselves and in the thin surface membranes that cover the lungs – the pleurae. Pleural plaques are discrete localised areas of fibrosis that typically affect the lining of the inner chest wall (the parietal pleura).

5. They arise from exposure to asbestos. To all intents and purposes pleural plaques currently found in the British population have been occupationally caused (in contrast to some parts of the world where environmental exposures to asbestos cause an important background incidence of plaque formation).

6. The degree of exposure sufficient to cause pleural plaques is much lower than that required to cause asbestos-related disease of lung tissue (for example asbestosis), and plaques are widely accepted to be the commonest medical manifestation of asbestos exposure in the population at large.

7. Estimates of the exact frequency are limited in several important ways. Firstly, plaques evolve slowly over time and become large enough to see only many years after first exposure. Thus, their reported prevalence increases the longer the latent interval (elapsed time from first exposure) and at older ages. Secondly, the likelihood of plaques

developing following asbestos exposure tends to be related to the cumulative level of exposure, and so depends on the patterns and levels and timings of exposure across the population at earlier periods of employment. Thirdly, most studies of plaques have used chest radiographs as the method of detection, but chest radiographs only detect a minority of all cases (as confirmed by more sensitive techniques like scanning and more invasive procedures, such as autopsy) – estimates of frequency vary with the method of detection and case definition, but most data derive from a relatively insensitive detection measure. Fourthly, because of the relation to cumulative dose, frequency varies markedly according to the population studied, being much higher in screening programmes of asbestos workers and in post-mortem series than the population as a whole. Even consecutive chest films from hospital clinics may give a misleading impression of disease frequency, as these are taken to investigate people's illness episodes and plaques seldom cause symptoms.

8. Unfortunately, no representative population-based screening data have been collected within the UK. Such data that exist from other countries are historic and more representative of exposure patterns overseas. No direct or precise estimate of the current prevalence of pleural plaques in the UK is available. However, in absolute terms the condition is likely to be common, with one expert suggesting that as many as 36,000 to 90,000 people a year may be developing plaques.

9. The nature and anatomical location of pleural plaques means that they do not alter the structure of the lungs or restrict their expansion. Therefore, they would not be expected to cause an important degree of impaired lung function or disability; and such studies as we have found and such experts as we have consulted agree that losses of lung function are likely to be either small or non-existent. Some loss may arise coincidentally from minor degrees of underlying lung fibrosis, undetected in the chest radiography studies that dominate the literature. In any event, any losses fall well short of the compensatable level of disability within the Industrial Injuries Scheme defined previously in relation to chronic obstructive pulmonary disease.

10. Most authorities hold that pleural plaques rarely cause major symptoms, just as they rarely cause major impairment of lung function. Research reports give slightly conflicting evidence on this point, but a difficulty exists in obtaining an unbiased estimate of symptom frequency, especially as individuals who attend medical services with chest symptoms are more likely to receive a chest radiograph, and so more likely to have latent pleural plaques discovered than other individuals. Data on the frequency of common symptoms in those with previously unrecognised pleural plaques, drawn from a representative sample of the general population, are seldom available; but a large general population survey from Uppsala in Sweden was reassuring, finding that practically all of 827 subjects in this position were symptom free at the time that their plaques were discovered.

11. Plaques tend to grow slowly over time, but they do not become cancerous. Neither are they a cause of cancer at other sites, such as lung cancer or mesothelioma. However, the balance of evidence suggests that they are a marker of *future risk* of lung cancer and mesothelioma, because they are a marker of exposure to asbestos.

12. Higher quality cohort studies, which allow for exposure history and (for lung cancer) smoking habits, suggest that the increases are a consequence of the degree of exposure to asbestos and that the presence of pleural plaques does not, of itself, independently affect risk levels. In other words, any increase in risk in those with pleural plaques arises because they *have been exposed to asbestos*, not because they *have pleural plaques*. Since plaques can arise from low as well as high levels of exposure, and are imperfectly diagnosed by most research inquiries, the predictive information about future risks is limited and imprecise, a more useful indicator being the employment history.

13. The Council recognises that plaques can be a source of distress and anxiety to individuals in whom the condition is diagnosed. However, it found no research evidence, (positive or negative), about the resulting scale and severity of psychological ill-health, and therefore no evidence against which to judge the case for prescribing any psychiatric sequelae.

14. The Council did not recommend prescription of pleural plaques when the matter was last considered in 2005, the main objection being that plaques rarely give rise to significant impairment of lung function and so are unlikely to cause compensatable disablement within the Industrial Injuries Scheme. The extensive evidence received in the present review does not give cause for the Council to revise its opinion, and we do not recommend prescription in relation to pleural plaques for either physical or psychological disability.

15. In civil proceedings different considerations may apply. The Ministry of Justice is currently consulting on the question of whether plaques should or should not constitute actionable or compensatable damage in civil law. This report may serve to supplement the medical evidence obtained during that review, but should not be construed as advice from the Council on the stance we feel should be adopted by civil courts of law.

INTRODUCTION

Background to the review

16. On the 11th June 2008, the Secretary of State for Work and Pensions referred the question of pleural plaques to the Industrial Injuries Advisory Council (IIAC) under Section 171(3) of the Social Security Administration Act 1992. The Council was asked in particular to consider the current prevalence of pleural plaques, the occupational causation of pleural plaques currently found in the population, the likelihood of disability arising from pleural plaques, the likelihood of other more severe complications of asbestos exposure arising amongst those currently having plaques, and whether compensation through the Industrial Injuries Scheme would be appropriate for people diagnosed with this condition.

17. The Council has reviewed the evidence relating to these questions in the context of potential prescription for pleural plaques under the Industrial Injuries Scheme.

The Role of the Industrial Injuries Advisory Council

18. IIAC is an independent statutory body established in 1946 to advise the Secretary of State for Social Security on matters relating to the Industrial Injuries scheme, which includes Industrial Injuries Disablement Benefit (IIDB). The majority of the Council's time is spent

considering whether the list of prescribed diseases for which IIDB may be paid should be enlarged or amended.

Industrial Injuries Disablement Benefit

19. IIDB is one of the benefits included under the Industrial Injuries scheme. IIDB provides non-contributory, 'no-fault' compensation payments for disablement because of accidents or prescribed diseases which arise during the course of employed earners' employment. IIDB is paid in addition to other incapacity and disability benefits. It is tax-free and administered by the Department for Work and Pensions (DWP).

The legal requirements for prescription

20. The Social Security Contributions and Benefits Act 1992 states that the Secretary of State may prescribe a disease where he is 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.

21. In other words, 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.

22. In seeking to address the question of prescription for any particular condition, the Council first looks for a workable definition of the disease. The Council then 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 according to available scientific evidence.

23. An accident at work is specifically catered for within the IIDB scheme. However, if the condition might result from occupational exposure in the absence of an identifiable accident, the Council must consider whether it should be included in the list of diseases that are prescribed for the purposes of IIDB. In these circumstances it may be possible to ascribe a disease to a particular occupational exposure in two ways – from specific clinical features of the disease or from epidemiological evidence that the risk of disease is at least doubled by the relevant occupational exposure.

<u>Clinical features</u>

24. For some diseases attribution to occupation may be possible from specific clinical features of the individual case. For example, the proof that an individual's asthma is caused by his occupation may lie in its improvement when s/he is on holiday and regression when s/he returns to work, and in the demonstration that s/he is allergic to a specific substance with which s/he comes into contact only at work. It can be that the disease only occurs as a result of an occupational hazard (e.g. coal workers' pneumoconiosis).

<u>Doubling of risk</u>

25. 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 on the balance of probabilities depends on epidemiological evidence that work in the prescribed job, or with the prescribed occupational exposure, increases the risk of developing the disease by a factor of two or more. The requirement for, at least, a doubling of risk is not arbitrary. It follows from the fact that if a hazardous exposure doubles risk, for every 50 cases that would normally occur in an unexposed population, an additional 50 would be expected if the population were exposed to the hazard. Thus, out of every 100 cases that occurred in an exposed population, 50 would do so only as a consequence of their exposure while the other 50 would have been expected to develop the disease, even in the absence of the exposure.

Therefore, for any individual case occurring in the exposed population, there would be a 50% chance that the disease resulted from exposure to the hazard, and a 50% chance that it would have occurred even without the exposure. Below the threshold of a doubling of risk only a minority of cases in an exposed population would be caused by the hazard and individual cases therefore could not be attributed to exposure on the balance of probabilities. The epidemiological evidence required should ideally be drawn from several independent studies, and be sufficiently robust that further research at a later date would be unlikely to overturn it.

26. Other practical requirements include the presence of a disease and exposure that are verifiable within the Scheme's constraints, and a meaningful level of disablement in at least some potential applicants.

Method of investigation

27. IIAC referred the matter of pleural plaques to its permanent sub-committee, the Research Working Group, which conducted a literature search covering some 200 pages of abstracts. Altogether some 75 key research papers were retrieved and reviewed. The Council also consulted with a number of leading experts in the field of respiratory medicine and asbestos-related diseases, nationally and internationally, and made a call for evidence on its website and through a newsletter of

the UK Society of Occupational Medicine. Appendix 1 lists the experts
and bodies contributing evidence.

Asbestos and asbestos-related lung diseases

28. The term asbestos refers to a group of natural fibrous silicates whose
    strength, heat resistance, and chemical and electrical properties have
    been widely exploited since the late 1800s. The most common forms of
    asbestos are chrysotile (white asbestos), crocidolite (blue asbestos),
    amosite (brown asbestos), anthophyllite and tremolite.

29. The different types of asbestos fibres have different physical properties
    and can be classified into amphiboles (straight fibres) and serpentine
    (wavy fibres). The physical properties of asbestos fibres, in particular
    their length and tendency to split longitudinally, are important
    determinants of their pathogenicity. Amphibole fibres persist longer in the
    lungs than chrysotile (a serpentine fibre), which probably accounts for
    the increased toxicity of these fibres. However, few workers will be aware
    of which asbestos fibre they were exposed to, and in the vast majority of
    cases exposures have been mixed.

30. Due to the widespread global use of asbestos, many adults have
    evidence of environmental exposure to asbestos in their lungs (fibres of
    asbestos, or so-called 'asbestos bodies' as lung tissue markers of
    exposure). Asbestos was widely used in the 1950s to 1970s, with peak
    exposures occurring in the UK in the mid to late 1960s. Restrictions on

the use of asbestos followed the introduction of the 1969 Asbestos

Regulations in the UK. The importation, supply and use of raw asbestos

or asbestos-containing materials (with a few exceptions) were banned in

the UK in 1992 for amphibole asbestos (blue and brown), and in 1999 for

chrysotile (white). The use of asbestos has now been banned in most of

the developed world, although substantial amounts of asbestos remain

present in business and domestic premises and may still be encountered

in significant concentrations in air if disturbed in the course of

maintenance, renovation and demolition work.

31. Inhalation of asbestos can cause disease in the lungs themselves (both

the conducting airways – the bronchi – and the peripheral gas

exchanging parts of the lungs - the alveoli) and also in the thin surface

membranes which covers the lungs - the pleurae. The pleurae are

comprised of two membranes: the outer membrane (parietal pleura)

which covers and is attached to the inside of the rib cage and the inner

membrane (visceral pleura) which is attached to the surface of the lung,

blood vessels, bronchi and nerves.

32. Within the lungs, inhaled asbestos can cause fibrosis or scarring

(asbestosis) and cancer of the bronchi (lung cancer). At the lung surface

it can also cause disorders of the pleura, of which four categories are

commonly distinguished - pleural plaques, pleurisy, diffuse pleural

thickening and malignant mesothelioma of the pleura (and peritoneum –

a similar lining membrane in the abdominal cavity).

33. This report focuses on pleural plaques – their frequency, causes, and consequences, and the scope for prescription. Other asbestos-related diseases have been the subject of earlier reports, and are described briefly for information in Appendix 2. Asbestosis, asbestos-related lung cancer, mesothelioma and diffuse pleural thickening are prescribed diseases for which IIDB is already payable (Appendix 3).

Pleural plaques

34. Pleural plaques are discrete, localised areas of fibrosis with a basket-weave type appearance, which typically affect the parietal pleura and only rarely the visceral pleura. These fibrous areas may become calcified over time (calcified pleural plaques).

35. Plaques have an almost specific relationship to asbestos exposure, and evidence for their occupational causation lies in the very well-established excess prevalence of plaques in exposed populations and rarity of plaques in the absence of recognised exposure to asbestos. They are the commonest manifestation of exposure to asbestos, generally appearing some 20-40 years after first exposure (that is, after a 'latent' interval).

36. In some parts of the world (but not as far as known in the UK), plaques are endemic in the general population, where they arise from natural or

man-made environmental contamination with asbestiform minerals (Hillerdal 1997). Thus, crocidolite exists in surface soil in rural China (Luo et al 2003) and tremolite in surface deposits in parts of Corsica (Rey et al 1994); naturally occurring tremolite and chrysotile have been used in the stucco whitewashing of houses in Eastern Turkey (Hasanoglu et al 2003) and in Macedonia (Sichletidis 1992); and pleural plaques appear to be common in residents of these communities. They have also been reported in people living near, but not working in, asbestos mines (Hiraoka et al 1998; Neuberger et al 1978) and among the families of asbestos workers (Luisi et al 2007), and are more common in urban than in rural settings. The overwhelming majority of cases in the UK, however, are caused by exposures in the occupational setting.

37. It is unclear how asbestos fibres cause pleural plaques, but believed possible that inhaled asbestos fibres induce an inflammatory response involving lung cells that engulf foreign matter (pleural macrophages) and which set up a slow fibrotic reaction.

38. In life, pleural plaques can be visualised by radiography and are often detected as an incidental finding on a chest radiograph taken for other purposes. However, even with systematic screening, simple radiology does not discover every case – particularly as plaques are more readily visualised on a plain chest radiograph when they calcify, and calcification occurs only after a delay of several years. More sensitive

imaging methods such as high resolution computer tomography detect

plaques that go unreported on chest radiographs, while the gold

standard of diagnosis is autopsy. In one survey, only 12.5% of plaques

found at post-mortem were seen on an ante-mortem chest radiograph

(Hillerdal et al 1980), and the range in other surveys has varied from

40% to as little as 8% (Wain et al 1984, Greenberg 1992). The false

negative rate is thus substantial. In addition, false positive diagnosis

may arise, as calcification in other forms of pleural thickening (e.g.

tuberculosis) may simulate the appearance of plaques and be mistaken

for them.

Evaluation of the research literature relating to pleural plaques

Prevalence of plaques; occupational and non-occupational causes

39. Estimates of the prevalence of pleural plaques depend on the method

of detection and case definition (see paragraph 38). They also vary

markedly according to the setting and population under study, the

degree of exposure to asbestos, the age of those investigated (and

hence the interval from initial exposure) and the calendar period of

inquiry.

40. Pleural involvement can arise at a much lower level of exposure than

required to cause disease within the lung tissue itself (e.g. asbestosis,

asbestos-related lung cancer). Even so, the risk of developing pleural

plaques tends to be related to dose of asbestos and especially latency,

and is greater in those with higher cumulative levels of exposure, longer durations of exposure and longer elapsed times since first exposure. Correlations with exposure can be found both in relation to work history (Selikoff 1965; Rubino et al 1980, Zitting et al 1995, Hilt et al 1986) and to estimated lung burden of asbestos fibres or bodies (Karjalainen et al 1994, Rubino et al 1980, Roberts 1971, Bianchi et al 1991). Not all reports have confirmed a clear-cut exposure-response pattern (van Cleemput et al 2002); but errors are likely to exist both in detecting plaques (paragraph 38) and in estimating a person's lifetime exposure to asbestos (Boffetta 1998), and most authorities assume there to be a general relation to dose and time since exposure, perhaps subject to biological variation of host defence response between individuals.

41. Against this background, the prevalence of pleural plaques can be expected to be much higher in a group of workers with heavy exposure than in the general population, among whom such workers will be a small minority; higher also in areas where heavy exposure to asbestos has been prevalent in local industries; and more common among older people who have incurred historically higher levels of occupational exposure and passed through the waiting time (latent interval) until plaques first appear.

42. The table in appendix 1 illustrates this marked variation. Studies that derive from autopsies in predominantly elderly subjects – often in the

context of communities with local asbestos industries – have reported very high rates of pleural involvement (25-70% in men; Andrion et al 1982, Svenes et al 1996, Karjalainen et al 1994, Bianchi et al 2000).

43. Similarly, pleural plaques have been frequent in surveys of heavily exposed workers in specific industries, such as construction and building and repair, as illustrated by data from Finland and Sweden (18-40%; Koskinen et al 1996, Oksa et al 1992, Sanden et al 1987).

44. The exact prevalence has varied not only between occupational groups, but also over time, tending to be lower in pre-1980 reports than implied by Table 1 (3% to 13% across most studies, and as low as 3-8% in British dockyard workers, according to Hillderdal et al 1980). The higher estimates in more recent surveys probably reflect the longer elapsed interval from first exposure: the prevalence of pleural plaques may be rising, as seen also with incident mesothelioma. In summarising more recent evidence, Parkes suggests that about 5-15% of those with occupational exposure to asbestos will have uncalcified pleural plaques 20 years after this exposure and about a third or more will have calcified plaques 30 years on.

45. By contrast, pleural plaques have been far less common in large-scale surveys that have sampled from the general population. Thus, plaques were found in 7.7% of a representative sample of 7,085 people aged 30 years or over who participated in the mini-Finland Health Survey in

1978-1981 (Zitting et al 1995) (a country with endemic environmental exposure to asbestos); and in 1.0% to 2.2% of men from large population screening programmes in Telemark in Norway and Uppsala in Sweden (Hillerdal 1978, Hilt et al 1986). Other reports from Germany and the USA (as cited in Hillerdal 1997) place the prevalence at 0.5% to 1% in surveys conducted in the 1970s. Such a difference in magnitude from autopsy and screening programmes in exposed workers reflects the lower likelihood of significant exposure in the population at large. (As may be imagined, autopsies are not conducted at random; by the same token, even consecutive chest radiographs in hospital departments may give a misleading impression of disease frequency.)

46. To the Council's knowledge, no population-based data have been collected nationally or regionally within the UK akin to those in other countries. Pleural plaques were estimated to be the second commonest occupational lung disorder, as judged by notifications by chest specialists and occupational physicians to the UK Surveillance of Work-related Occupational Respiratory Disorders (SWORD) Scheme (an estimated 590 cases in 1995; Keynes et al 1996) but this figure as a measure of national burden is likely to be highly conservative, given that plaques generally only come to attention and specialist care by chance; and that plaques are widely held to be commoner than diffuse pleural thickening, for which some 2,000 claims were made under the IIDB Scheme in 2002 and far commoner than mesothelioma, some

1,800 new cases of which are diagnosed annually. One expert has suggested that for every new person who develops mesothelioma in a given period there may be 20-50 people developing plaques – i.e. perhaps 36,000 to 90,000 people per year. Firm figures are hard to come by, given the many variables that influence prevalence and the lack of a UK-specific mass screening survey in the general population directed specifically at the question; but it is worth reflecting that if the prevalence were 1 to 2% in men aged 50 years and over (as in Telemark or Uppsala), this would equate to as many as 80,000 to 160,000 cases across the UK as a whole. Such cases would mostly go undetected during the lifetime of the individuals concerned.

Natural history of pleural plaques and their health impact

47. Pleural plaques tend to grow in size over time, and new ones may appear over follow-up, but the process is a very slow and by no means inevitable one. When de Klerk et al (1989) followed 384 men from the Wittenoom crocidolite mill and mine for two to 38 years, their plaques were not seen to progress beyond their initial thickness or extent.

48. The effect of pleural plaques on lung function has been examined in many studies. Those identified by the Council's literature search are discussed briefly below.

49. In a study of sheet metal workers, Schwartz et al (1990) compared lung function in 258 workers with plaques and 877 workers without. On

average, and without allowing for smoking or lung fibrosis, there were small losses in lung function in those with plaques (average decline in Forced Expiratory Volume in 1 second (FEV_1) of 250 mls and in Forced Vital Capacity (FVC) of 340 mls).

50. Kouris et al (1991) evaluated 913 asbestos workers from Minnesota, including 146 with plaques. Compared with workers with normal pleura, those with plaques had on average about a 5% reduction in FVC and a 6% reduction in FEV_1. The odds of an FEV_1 <80% of predicted were raised 1.5-fold in those with plaques and similarly for an FVC <80% predicted, although the report is unclear as to whether the analysis also allowed for smoking and lung fibrosis (although these factors were separately analysed).

51. Jarvholm et al (1986) considered 202 non-smoking shipyard workers with varying exposure to asbestos, including 87 with plaques but no other apparent lung involvement on their chest radiograph. Three of 115 workers with normal radiographs and 13 of 87 with plaques had FVCs below the reference limits and on average the FVC was decreased 6 to 9% in the group with plaques. The authors concluded that pleural plaques are associated with "slightly impaired lung function".

52. Bourbeau et al (1990) investigated 1,110 construction insulation workers, of whom more than half had pleural plaques. Compared with

those without any pleural abnormality, those with pleural involvement had a decrease in FEV_1 and FVC on average of 222 and 402 mls respectively; but the group included some individuals with diffuse pleural fibrosis, and when those with isolated pleural plaques were considered separately, the decrease was smaller still (200 and 350 mls after allowing for age, smoking status and lung fibrosis).

53. Garcia-Closas et al (1995) found a small and non-significant association between pleural plaques and restrictive deficit of lung function in 631 asbestos-exposed carpenters. The study had a low response rate and covered only relatively youthful workers. It found a mean loss of 121 mls in FEV_1 in those with pleural plaques as compared with those without.

54. Zavalic et al (1993) studied 299 shipyard workers, including 68 with pleural plaques and no other lung abnormality. They found no significant differences in FEV_1, FVC and various other lung measures by presence or absence of pleural plaques, although values tended to be somewhat lower among those with bigger plaques.

55. Lilis et al (1991) investigated 1,584 asbestos insulation workers with long-term exposure to asbestos, drawn from a cohort of 17,800 male asbestos insulation workers from the US and Canada. Altogether, 975 had circumscribed pleural plaques. A pleural index score was created according to the extent of fibrosis and the size of the plaques. A

significant inverse relationship was found between FVC and the pleural index, consistent with the idea that extensive plaques can have more of an impact on lung function.

56. Damian et al (2004) measured lung function on two occasions, three years apart, in 196 shipyard workers with exposure to asbestos and 109 healthcare workers. Among those with plaques, no difference was found in loss of lung function (mls per year) as compared with other groups. These findings were confirmed in a subsequent report in which computed tomography was used to exclude lung involvement in subjects with pleural plaques and statistical allowance was made for various other factors including smoking habits.

57. In a small survey of long-serving workers from an asbestos cement factory, van Cleemput et al (2001) found no relation between the presence or absence of plaques and measures of lung function, and no relation between the surface area of plaques (quantified by CT scanning) and lung function. However, most plaques were similarly small in size, limiting the power of investigation.

58. Ohlson et al (1984, 1985) investigated lung function in a small study of 75 asbestos cement workers, a third of whom had plaques, and compared their findings with local referents. Measures of FEV_1 and FVC proved to be somewhat lower than predicted (by 250 and 300 mls on average after allowing for age and smoking habits), but no

significant differences were detected between those with and without pleural plaques.

59. Ostiguy et al (1995) conducted a cross-sectional and seven year follow up survey on long-term workers from a copper refinery industry, including 54 individuals with pleural plaques. These were associated with a small average loss of FVC (196 mls) and a non-significant reduction of FVC over time.

60. Hedenstierna et al (1981) compared 36 construction workers with pleural plaques with 36 matched controls who had had no professional exposure to asbestos. Average losses in lung function were small (FEV_1 80 mls; FVC 230 mls).

61. Pleural plaques do not alter the structure of the lungs themselves and their anatomical location (on the parietal pleura of the inner chest wall) is such that they do not restrict the expansion of the lungs. There are reasons therefore to suppose that they will not give rise to an important level of respiratory disability, and studies such as those cited in paragraphs 49 to 60 are in keeping with this.

62. Such losses as have been described in the research literature may owe more to undetected underlying lung fibrosis or diffuse pleural thickening than to the plaques themselves. Most studies of lung function have employed chest radiographs to sub-classify according to

the presence or absence of plaques; these methods fail to detect a significant proportion of cases with lung fibrosis, just as they miss some cases with pleural plaques. However, a study by Copley et al (2001), which considered the functional consequences of pleural disease in patients evaluated both with chest radiography and with more sensitive computed tomography (CT) scanning, found that pleural plaques were not independently associated with deficient lung function once accurate assessment of lung tissue involvement had been made.

63. In any event, the losses reported in the research literature are typically small, a view endorsed by the experts we consulted. As judged by the criteria the Council applied in recommending prescription for chronic obstructive pulmonary disease in coal miners (Cm 2091, Cm 7253) they would fall well short of a compensatable level of disability within the IIDB Scheme.

64. Exceptionally, very extensive pleural plaques (created as large plaques become confluent) can lead to disability and breathless by restricting lung expansion. Although firm figures are hard to obtain, two of the experts we consulted estimated that this would occur in fewer than 1% of those with plaques. Insufficient research evidence was found on the extent of disablement in such rare extreme cases. Another symptom only very rarely reported (in fewer than 1% of cases) is that of a grating sensation in the chest while breathing.

65. Most authorities hold that pleural plaques rarely cause major symptoms, just as they rarely cause major impairment of lung function. However, the various research reports give slightly conflicting evidence on this point. According to Bourbeau et al (1990), for example plaques were associated with an increased risk of shortness of breath "with major activities", whereas Kouris et al (1991) found no increase in breathlessness. The small study by Hedenstierna et al (1981) found cough with sputum to be more common in those with plaques, although the 30 cases upon whom these conclusions were based were selected from 261 men with plaques and 162 other asbestos-exposed construction workers, and may not have been representative of the source population in terms of symptoms. In a brief report by Jarvholm et al, "having to stop to recover your breath when walking on flat ground at your own speed" was more common in men with pleural plaques, but based on only 4 men with symptoms and those with plaques tended to be older and more often smoked.

66. A particular difficulty in this area of investigation is how to obtain an unbiased estimate of symptom frequency. Individuals who attend medical services with chest symptoms are more likely to receive a chest radiograph, and therefore more likely to have latent pleural plaques discovered than other individuals. More informative are data on the frequency of common symptoms in those with previously unrecognised pleural plaques drawn from a representative sample of the general population, but such data are seldom available. However,

the large study by Hillerdal (1981) identified and followed 827 individuals with pleural plaques from the general population study in Uppsala in Sweden. The authors found that practically all of these subjects were symptom free at the time that their lesion was discovered.

67. It has been suggested that where symptoms do arise these may reflect involvement also of the underlying lung tissue and minor degrees of lung fibrosis which go undetected on chest radiograph or scan, rather than being caused by the plaques *per se*. Another possibility, as suggested by clinicians, is that personal awareness of plaques, and the understandable anxiety this engenders, can heighten an introspective search for health problems and a tendency to misattribute coincidental symptoms to lung 'damage'.

Future risks of cancer

68. Despite an initial case report that raised the concern (Lewinsohn 1974), it is now well established that pleural plaques do not in themselves become malignant. Neither are they a *cause* of cancer of the pleura or at other sites, such as the lung.

69. However, plaques have been linked with a greater *future risk* of these cancers, and this is unsurprising. The incidence of lung cancer increases with asbestos exposure, as does the probability of pleural plaques. Therefore, a statistical link can be expected between plaques

and lung cancer. However, it is well established that lung cancers do not arise out of the malignant transformation of plaques, the lung and lung lining being anatomically and histologically distinct. Rather, the relation is one of a *marker*, not a cause. Plaques are a marker of exposure to the causal agent, asbestos.

70. Risks of lung cancer and mesothelioma are increased in workers with pleural plaques, but only because of the risks associated with the underlying exposure to asbestos. In practice the amount by which risks are elevated is hard to assess, given the measurement errors that arise and especially the false negative rate of plaque detection (paragraph 38) and the imprecision of exposure assessment. However, several epidemiological studies have been performed which report on cancer risks.

71. In an early British survey, based in the shipyard in Barrow in Furness, Fletcher (1972) studied lung cancer incidence in men with and without radiographic evidence of plaques. Screening covered shipyard workers across various trades (laggers, welders, crane drivers, foundry workers, men working near radiological sources or using certain types of equipment, joiners, apprentices, new entrants, men with breaks in service, and men referred for reasons unspecified). 408 men aged 45 or older with pleural plaques were matched with 404 other men from the same trades of similar ages and with surnames that were close in an alphabetical listing. Observed deaths from lung cancer were

compared with those expected from local mortality statistics. Among men with plaques, 16 deaths were observed vs. 6.74 expected (Rate Ratio (RR) 2.37), as compared with 7 observed vs. 5.61 expected (RR 1.25) for men without pleural plaques. The higher risks in men with pleural plaques could have arisen because of differences in smoking habit or, more likely, higher levels of exposure to asbestos, and no estimates were made of these factors.

72. Hillerdal et al (1979) assessed all newly diagnosed cases of lung cancer from Uppsala in Sweden during 1971-1976 for their exposure history and chest radiograph findings. Around a quarter of cases worked in jobs with probable or definite exposure to asbestos, and among men aged 40 years or over with accessible chest radiographs, 6.7% were confirmed as having pleural plaques. For the county as a whole a prevalence of 1% was estimated across all men aged 40 or over in a separate survey by the same author.

73. Wain et al (1984) found the prevalence of lung cancer to be similar in those with and without pleural plaques in a survey involving 434 autopsies. However, their study included only 25 cases with pleural plaques and had limited power to probe the study question.

74. Sanden et al (1987) conducted a prospective study of 3,787 men aged younger than 65 in 1978 who were employed as shipyard workers in Gothenburg. Cancer cases and deaths to the end of 1983 were

identified by record linkage. All of the shipyard workers had had a chest radiograph and a questionnaire about asbestos exposure and smoking habits. Mortality rates for lung cancer (vs. males in the same city) were slightly elevated overall (RR 1.12), but lower in men with plaques (RR 1.03) than in men without them (RR 1.40). Observed deaths for several other cancers were below the numbers expected; RRs were only raised for cancer of the prostate, and in this case lower in those with pleural plaques (1.32) than in all men (1.38).

75. Cullen et al (2005) followed 4,060 asbestos-exposed men prospectively for 9-17 years, in a trial to study whether vitamin A and ß-carotene could prevent lung cancer (CARET study). Recruitment was restricted to older male smokers with at least 5 years of work in an asbestos trade. After allowing for degree of lung fibrosis on the chest, age, amount smoked, and treatment received, RRs in those with pleural plaques were raised 1.43-fold vs. other plaque-negative asbestos workers. Among those with minimal or no lung opacities (i.e. without any evidence of asbestosis), RRs were even higher (1.91).

76. In a further report based on the cohort of shipyard workers described in paragraph 74, Sanden et al (1991) estimated exposure intensity and duration for each worker by questionnaire and categorised this as very heavy, heavy, low, or very low. In those with at least 20 years between the onset of exposure and health evaluation (20 year latency) there were 837 cases of pleural plaques, 55 cases of asbestosis and 1852

cases without pleural plaques or asbestosis. Altogether, there were 11 cases of mesothelioma, the incidence of which was greater in those with heavy or very heavy exposure to asbestos (0.5%) than in those with low or very low asbestos exposure (0.1%). However, the risk of mesothelioma was not increased in those with pleural plaques as compared to those without. (0.5% v 0.4%).

77. Koskinen et al (2002) studied 16,696 male Finnish construction workers, with at least 10 years of work experience during 1960-1990 through an asbestos screening campaign. At recruitment, workers received chest radiographs and questionnaires and were then followed for cancer during 1990-2000 through the Finnish cancer registry. Although rates of cancer overall, and lung cancer in particular, were close to expected (with standardised incidence ratios (SIR) of 1.03 and 1.07 respectively), excesses of mesothelioma were more marked (SIR 1.96). After allowing for age, smoking habits, lung fibrosis, trade, and estimated level of asbestos exposure, risks of lung cancer were 20% higher in those with pleural plaques than in those without, but risks of mesothelioma were 40% lower; and (despite the large scale of investigation) the findings were subject to substantial statistical uncertainty. Asbestosis proved a far more potent and certain marker of lung cancer and mesothelioma risk than presence of plaques.

78. In another Finnish study, based on mass radiographic screening of almost 8,000 adults from the general population, Partanen et al (1992)

identified 604 subjects with plaques but no other asbestos-related abnormalities and a similar number of controls matched by age, sex and locality. During a follow up which extended from 1972 to 1989, they found 25 cases of lung cancer among those with plaques as compared with 25 in the controls – a RR of 1.1. However, they cautioned that actual risks might be higher, citing the potential of chest radiographs to overlook plaques (paragraph 38).

79. Hillerdal (1994) identified 1,596 men from Uppsala with plaques on chest Radiograph in 1963 and followed them until 1985. Cases of mesothelioma and lung cancer were detected through the Swedish cancer registry and compared with expected age and year-specific incidence rates. After allowing for smoking, 50 lung cancers were found as compared with 32.1 expected (RR 1.6). Risks were higher among those who also had asbestosis (RR 2.3) than in those without (RR 1.4). Nine mesothelioma deaths were found as compared with only 0.8 expected, a RR of 11.25 after correcting for smoking habits.

80. Bianchi et al (1997) conducted 3,041 consecutive autopsies during 1979-1995 in an area of Italy with a very high local incidence of mesothelioma. The frequency of plaques was found to be higher in cases with mesothelioma than in cases without; in men, for example, the odds of finding plaques were raised 3.4-fold after allowing for age.

81. A study of similar design concerned 1,097 autopsies of adults from Turin, but on this occasion Mollo et al (1984) explored the association between plaques and lung cancer. Among men in whom this tumour was found, the RR of also having pleural plaques was raised 1.5-fold, although findings were not statistically significant. A more striking association was found with cancer of the larynx (RR 8.8), but a major limitation of this study was the failure to allow for smoking habits. Cigarette smoking is likely to have been more common in asbestos workers, and is a strong to very strong risk factor for these two cancer types (but not for mesothelioma, and so of less concern in the study by Bianchi et al mentioned in paragraph 80).

82. A survey by Reid et al (2005) followed participants from a cancer prevention programme in the crocidolite mining and milling town of Wittenoom in Western Australia. Benign pleural disease and asbestosis were identified on enrolment chest radiographs. Exposure to crocidolite was estimated, based on dust measurements and employment records for those exposed occupationally and from personal monitor fibre counts for former Wittenoom residents. Between 1996 and 2002 there were 76 cases of mesothelioma (56 pleural, 20 peritoneal). Pleural thickening and asbestosis were associated with an increased risk of peritoneal mesothelioma (but not pleural mesothelioma). Calcified pleural plaques, by contrast, were not associated with mesothelioma of either type, having taken cumulative asbestos exposure and age at the start of the programme into account.

83. In reviewing whether pleural plaques are associated with an increased risk of lung cancer or mesothelioma, the Council considers the best information to derive from cohort studies of well defined populations in which plaques have been identified at an initial time point, with subsequent follow up to assess causes of death in relation to the presence or absence of pleural plaques, having taken levels of asbestos exposure and (for lung cancer) cigarette smoking into account. The studies most closely meeting these criteria are described in paragraphs 74, 76, 77 and 82. These four cohort reports do not show evidence of an increased risk of lung cancer or of mesothelioma in cases with pleural plaques, having taken level of asbestos exposure into account. The inference is that the increased risk of lung cancer and mesothelioma in asbestos workers is a consequence of the level of exposure to asbestos and in the case of mesothelioma, interval from first exposure; the presence of pleural plaques does not independently affect the level of risk.

84. In other words, the balance of evidence suggests that pleural plaques are a marker of future risk of lung cancer and mesothelioma, the relationship being stronger for mesothelioma; but any increase in risk in those with pleural plaques arises because they have been exposed to asbestos, not because they have pleural plaques.

Psychological Effects

85. Patients discovered to have pleural plaques on routine investigations, and workers found to have pleural plaques on screening, may understandably feel apprehensive about their future health. The potential exists for them to confuse benign and common pleural plaques with far rarer and more serious consequences of asbestos exposure such as malignant mesothelioma and asbestosis. And even with appropriate understanding, concern may still exist about the added future risk of cancer arising from past exposures (paragraphs 68-84). Workers may know, or have heard of colleagues diagnosed with pleural plaques who then died of fatal forms of asbestos-related disease, and may not feel wholly reassured even by careful professional counselling.

86. The scale and severity of psychological ill-health in individuals with pleural plaques appears to have been little studied. Whether the risk of mental illness amounting to a psychiatric disease is increased, and by how much, remains unclear. In particular, having searched the research literature against the question, the Council found no evidence to suggest a more than doubling of risks of psychiatric disorders in workers with pleural plaques as compared with other asbestos workers, such that attribution could be made on the balance of probabilities (according to the logic of paragraph 25).

The Council's view on the case for prescription

87. Pleural plaques are diagnosable by chest radiograph or CT scan, although likely to be under-diagnosed in some circumstances. In addition, the link to occupation can be reasonably presumed on the basis of clinical features (paragraph 24) and without recourse to elaborate epidemiology – to all intents and purposes, cases in the UK are likely to have arisen from occupational exposures. Thus two of the practical requirements for prescription – those of robust diagnosis and occupational attribution – can reasonably be addressed in the individual case.

88. However, the Council did not recommend prescription of pleural plaques when the matter was last considered in 2005 (Cm 6553), the main objection being that plaques rarely give rise to significant impairment of lung function and so are unlikely to cause compensatable disablement within the terms of the IIDB Scheme. The extensive evidence before it from this inquiry serves to support this position.

89. The Council recognises that plaques can be a source of distress and anxiety to individuals in whom the condition is diagnosed; and also that such individuals, to varying degrees, depending on prior exposure to

asbestos, may carry a greater future risk of mesothelioma and probably also of lung cancer. However, the IIDB Scheme as presently constituted compensates *actual* disablement, rather than *future risk* of disablement or *health anxiety*.

90. The evidence received in the present review does not give cause for the Council to revise the opinion outlined in paragraph 88. We do not recommend prescription in relation to pleural plaques, for either physical or psychological disability.

91. In civil proceedings different considerations may apply to those outlined in paragraph 89. The Ministry of Justice is currently consulting on the question of whether plaques should or should not constitute actionable or compensatable damage in civil law. This report may serve to supplement the medical evidence obtained during that review, but should not be construed as advice from the Council on the stance we feel should be adopted by civil courts of law.

Prevention

92. Asbestos diseases can be prevented by ensuring that workers who come into contact with asbestos containing materials are not exposed to the asbestos fibres which may be released when these materials are

handled or otherwise disturbed. The importation, supply and use of asbestos have now been banned but asbestos was extensively used as a building material from the 1950s through to the late 1970s. Those currently at risk from exposure to asbestos fibres include those who remove asbestos containing materials and building and maintenance workers who may unknowingly be exposed during the course of their work. To deal with the risks of exposure, there is a requirement in the Control of Asbestos Regulations 2006 to carry out a risk assessment and to take a series of actions depending on the assessment to prevent exposure to asbestos fibres so far as is reasonably practicable. This includes a requirement for training and medical surveillance in certain circumstances. Since May 2004, there has been a duty on those who have maintenance and repair responsibilities for non-domestic premises to assess those premises for the presence of asbestos and the condition of that asbestos and, again, to take a series of preventive actions depending on the assessment.

Conclusions and recommendations

93. The Council recommends against including pleural plaques among the list of prescribed diseases compensated under the IIDB Scheme.

Appendix 1: Table of Selected Estimates of the Prevalence of Pleural Plaques in Different Populations and Settings

Setting Authors	Subjects Studied	Prevalence of Pleural Plaques (%)			Other Comments
		Men	Women	All	
Post Mortem Series					
Andrion (1982)	1019 consecutive adult autopsies in Turin, Italy, 1977-1979	24.5	7.0	19.1	The prevalence rose with age in men (from 11.3% in 30-39 year olds to 37.7% in 60-69 years olds).
Bianchi (2000)	3640 consecutive adult autopsies in Monfalcone, Italy 1979-1988	70.5	23.8	-	Local shipyard building industry. In deceased male shipyard workers, the prevalence was 87%.
Svenes (1986)	402 consecutive autopsies in adults aged >40 years in Vest-Agder, Norway, 1981-1983	26.9	3.1	16.9	Industrialised area with shipyards, metal refineries and a textile industry.
Roberts (1971)	334 consecutive autopsies in South-west Glasgow, near the Clyde, late 1960s	-	-	12.3	40 of 41 cases were in men, with a peak in the 7th decade. Many came from the local shipyard.
Karjalainen (1994)	288 consecutive autopsies in men aged 33-69 years, Helsinki, Finland, 1991-1992	58	-	-	Prevalence increased with age, with likely exposure, and with pulmonary concentration of asbestos fibres.
Selected Occupations*					
Kishimoto (2000)	2951 construction workers from Okayama, Japan, screened by chest Radiograph and confirmed by CT scan	-	-	5.4	Cases especially found in carpenters, plasterers, and concrete board cutters.
Koskinen (1996)	18,943 Finnish construction, shipyard and asbestos industry workers screened in 1990-1992	-	-	27.0	96% of participants were male (so the overall prevalence approximates the prevalence in men); 95% were employed in construction.
Oksa (1992)	437 Finnish construction workers screened as part of the Koskinen study	-	-	18-40	Prevalence varied by occupational group.

Setting Authors	Subjects Studied	Prevalence of Pleural Plaques (%)			Other Comments
		Men	Women	All	
Ostiguy (1995)	494 long-term copper refinery workers from Montreal, employed in 1991	10.9	-	-	Asbestos was used to insulate foundry furnaces
Marcus (1987)	925 Swedish car mechanics, screened in 1977-1981	-	-	4.4	No plaques found in 109 office workers from car-repair firms.
Sanden (1987)	1721 workers employed for at least 20 years at a shipyard in Gothenburg, Sweden; participated in a health check in 1977-1979 and followed until 1983	-	-	36.3	
Oliver (1985)	377 railroad workers from a central Pennsylvania railroad yard, surveyed cross-sectionally	-	-	22.9	
Broderick (1992)	1211 active and retired members of the US Sheet Metal Workers' Association, employed for at least 25 years, surveyed in 1986	-	-	21.5	
Kouris (1991)	913 former workers of a plant in Minnesota that made asbestos-containing ceiling tiles and wall boards, screened in 1988	-	-	16.0	
General Population Surveys*					
Zitting (1995)	Stratified 2-stage population sample, representative of the Finnish population aged ≥30 years in 1978-1981 (n=7085)	12.5	3.6	7.7	
Hilt (1986)	Population screening survey of men aged 40 years from municipalities in the county of Telemark, Norway, 1982-1983 (n=21,483)	-	-	2.2	Local industries included electrochemical plants, ferralloy plants, chemical equipment industries, iron foundries, paper mills and shipyards.
Hillderal (1978)	In the county of Uppsala, Sweden, a general health survey was in effect for several years from 1963, covering every inhabitant over age 16 (70% participation). Estimates were provided for men aged >40 years.	1.0	-	-	The prevalence of plaques in men aged >40 years ranged across the six municipalities from 0.5 to 3.5% in 1976.

*For studies in this category, plaques were detected by chest Radiograph unless otherwise stated.

Appendix 2:

List of experts submitting evidence

Professor Anthony Newman Taylor	National Heart and Lung Institute at Imperial College, London
Dr John Moore-Gillon	St Bartholomew's and Royal London Hospitals, London
Dr Robin Rudd	London Cancer Group (formerly, London Chest Hospital and St Bartholomew's Hospital, London)
Professor Corbett McDonald	National Heart and Lung Institute at Imperial College
Professor David Hendrick	Royal Victoria Infirmary and University of Newcastle upon Tyne, Newcastle upon Tyne

Consultation responses were also received from:

Resolute Management Services Ltd

Zurich Financial Services

Association of British Insurers (ABI)

Professor John Cotes	School of Medicine, University of Durham
Dr JE Burgess	Consultant Occupational Physician
Mr Alan Care	Solicitor, Thomson Snell and Passmore

Appendix 2: Other asbestos-related lung diseases

1. <u>Asbestosis</u> – Inhaled asbestos can cause inflammation which leads to fibrosis of the lungs. Asbestosis is fibrosis of the lungs; it does not refer to all types of asbestos-related diseases. Risk of asbestosis is related to the level of exposure to asbestos, but this must be substantial. The interval from initial exposure to the development of asbestosis is typically 15-30 years.

2. <u>Mesothelioma</u> – this is a cancer of the pleura (the lining of the lung), the peritoneum (the lining of the abdominal cavity) and, rarely, other lining membranes. In adults, the disease is caused in the great majority of cases by inhaled asbestos. It can be caused by only a few months exposure to low levels of asbestos. Mesothelioma generally develops a long time after first exposure, sometimes 40 years or more, and seldom less than 20 years. The disease is incurable and proves fatal, usually within 12-18 months of symptom onset.

3. <u>Lung cancer</u> – Asbestos-related lung cancer is a malignant tumour of the bronchi of the lungs. It is indistinguishable from lung cancer due to other causes, such as smoking, and carries the same unfavourable prognosis. Lung cancer due to asbestos usually develops after an interval from initial exposure of 20 or more years and requires substantial degrees of exposure. Lung cancer and asbestosis may be seen in the same individual, and is a Prescribed Disease when this occurs, but also in workers from trades with a high established risk of asbestosis (Cm 6553). Some

authorities have argued that fibrosis is a necessary precursor to tumour development.

4. <u>Benign pleural disease</u> – The other main category of benign pleural disease, in addition to pleural plaques, is diffuse pleural thickening. This occurs when there are large areas of fibrosis extend continuously within the pleural cavity. Diffuse pleural thickening usually involves mainly the visceral pleura. If extensive, it can cause impairment and disability due to the widespread constriction of the lungs. Diffuse pleural thickening usually occurs from 10 to 15 years after initial exposure to asbestos.

Appendix 3: Terms of prescription for asbestos-related diseases

Prescribed disease or injury	Occupation
D1 Pneumoconiosis*	9) Any occupation involving: 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.
D3 Diffuse mesothelioma (primary neoplasm of the mesothelium of the pleura or of the pericardium or of the peritoneum)	Exposure to asbestos, asbestos dust or any admixture of asbestos at a level above that commonly found in the environment at large.
D8 Primary carcinoma of the lung where there is accompanying evidence of asbestosis	a) the working or handling of asbestos or any admixture of asbestos; or b) the manufacture or repair of

	asbestos textiles or other articles containing or composed of asbestos; or
	c) the cleaning of any machinery or plant used in any of the foregoing operations and of any chambers, fixtures and appliances for the collection of asbestos dust; or
	d) substantial exposure to the dust arising from any of the foregoing operations.
D8A Primary carcinoma of the lung	Exposure to asbestos in the course of :
	a) the manufacture of asbestos textiles; or
	b) spraying asbestos; or
	c) asbestos insulation work; or
	d) applying or removing materials containing asbestos in the course of shipbuilding.
	Where all or any of the exposures occurs before 1st January 1975, for a period of, or periods which amount in aggregate to, five years or more, or otherwise, for a period of, or periods which amount in aggregate to, ten years

	or more.
D9 Unilateral or bilateral diffuse pleural thickening with obliteration of the costophrenic angle.	a) the working or handling of asbestos or any admixture of asbestos; or b) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos; or c) the cleaning of any machinery or plant used in operations and of any chambers, fixtures and appliances for the collection of asbestos dust; or d) substantial exposure to the dust arising from any of the foregoing operations.

* Please note that only the occupations associated with pneumoconiosis (asbestosis) are included in the table.

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