Specialist Medical Review Council

Declaration and Reasons for Decisions

Section 196W

Veterans' Entitlements Act 1986

Re: Statements of Principles Nos. 53 and 54 of 2013 concerning fibrosing interstitial lung disease and Statements of Principles Nos. 55 and 56 of 2013 concerning asbestosis

Request for Review Declaration Nos. 27 and 28

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1. In relation to the Repatriation Medical Authority (the RMA) Statement of Principles No. 53 of 2013 concerning fibrosing interstitial lung disease and death from fibrosing interstitial lung disease, made under subsections 196B (2) of the *Veterans' Entitlements Act 1986* (the VEA), the Specialist Medical Review Council (the Council) under subsection 196W of the VEA:

   DECLARES that the sound medical-scientific evidence available to the RMA is insufficient to justify an amendment to the Statement of Principles to existing factors, or to include a new factor or factors for;

      asbestos bodies and pleural plaque; or

      shorter periods of exposure to respirable asbestos fibres.

2. In relation to the RMA Statement of Principles No. 54 of 2013 concerning fibrosing interstitial lung disease and death from fibrosing interstitial lung disease, made under subsections 196B (3) of the VEA, the Council under subsection 196W of the VEA:

   DECLARES that the sound medical-scientific evidence available to the RMA is insufficient to justify an amendment to the Statement of Principles to existing factors, or to include a new factor or factors for;

      asbestos bodies and pleural plaque; or

      shorter periods of exposure to respirable asbestos fibres.

3. In relation to the RMA Statement of Principles No. 55 of 2013 concerning asbestosis and death from asbestosis, made under subsections 196B (2) of the VEA, the Council under subsection 196W of the VEA:

> DECLARES that the sound medical-scientific evidence available to the RMA is insufficient to justify an amendment to the Statement of Principles to existing factors, or to include a new factor or factors for;
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> asbestos bodies and pleural plaque; or
>
> shorter periods of exposure to respirable asbestos fibres.

4. In relation to the RMA Statement of Principles No. 56 of 2013 concerning asbestosis and death from asbestosis, made under subsections 196B (3) of the VEA, the Council under subsection 196W of the VEA:

> DECLARES that the sound medical-scientific evidence available to the RMA is insufficient to justify an amendment to the Statement of Principles to existing factors, or to include a new factor or factors for;
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> asbestos bodies and pleural plaque; or
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> shorter periods of exposure to respirable asbestos fibres.

5. In relation to the RMA Statements of Principles Nos. 53 and 54 of 2013 concerning fibrosing interstitial lung disease and death from fibrosing interstitial lung disease, and Nos. 55 and 56 of 2013 concerning asbestosis and death from asbestosis, made under subsections 196B (2) and 196B (3) of the VEA, the Council under subsection 196W of the VEA:

> RECOMMENDS that the RMA, when it carries out any future investigation(s), considers whether there is relevant sound medical-scientific evidence concerning any direct measurements of asbestos fibre levels during tasks relevant to Australian Naval personnel or derived from the international literature, or alternative measures of exposure to asbestos and the required level of exposure used in the existing factors, for “inhaling respirable asbestos fibres in an enclosed space/open environment” (clinical onset) and for “inhaling respirable asbestos fibres” (clinical worsening) in the Statements of Principles.

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REASONS FOR DECISION

INTRODUCTION TO THE COUNCIL AND ITS FUNCTIONS

1. The Specialist Medical Review Council (the Council) is an independent statutory body established by the VEA. In general terms, upon receipt of a valid application the Council is to review as relevant:

   – the contents of Statement/s of Principles in respect of a particular kind of injury, disease or death; or

   – a decision of the RMA not to determine, not to amend, Statement/s of Principles in respect of a particular kind of injury, disease or death.

2. In conducting a review, the Council must review all of the information (and only that information) that was available to the RMA when it made the decision under review. This is information which was actually used by the RMA as opposed to information which was generally available but not accessed by the RMA. A list of the information that was available to the RMA is provided in **Appendix D**.

3. Fundamental to Statements of Principles (SoPs), and so to a Council review, is the concept of sound medical-scientific evidence (SMSE), as that term is defined in section 5AB(2) of the VEA[1].

4. The information to which the Applicant referred, being information which the RMA advised was new information, that is, information which was not available to the RMA

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[1] The SMSE is a subset of the available information. It comprises those articles which the Council considers:

a) are relevant to the matters within the proposed scope of review, and

b) satisfy the definition in the VEA of 'sound medical-scientific evidence'.

Sound medical-scientific evidence is defined in section 5AB(2) of the VEA as follows:

“Information about a particular kind of injury, disease or death is taken to be sound medical-scientific evidence if:

a) the information:

(i) is consistent with material relating to medical-science that has been published in a medical or scientific publication and has been, in the opinion of the Repatriation Medical Authority, subjected to a peer review process; or

(ii) in accordance with generally accepted medical practice, would serve as the basis for the diagnosis and management of a medical condition; and

b) in the case of information about how that kind of injury, disease or death may be caused – meets the applicable criteria for assessing causation currently applied in the field of epidemiology.

The later requirement is held to mean ‘appropriate to be taken into account by epidemiologists’.

at the relevant times, and so was not considered by the Council in reaching its review decision is listed in **Table A3 of Appendix A**.

5. **Appendix B** sets out further details regarding the composition of the Council for this review and the legislation relating to the making of SoPs.

SCOPE OF THIS REVIEW

6. In his application the Applicant sought review of the contents of SoPs Nos. 53 and 54 of 2013 for Fibrosing Interstitial Lung Disease and Nos. 55 and 56 of 2013 for Asbestosis.

7. The Council, when reviewing the SMSE, must determine whether or not there is SMSE which indicated a reasonable hypothesis connecting the particular injury, disease or death to the relevant service.

8. In a reasonable hypothesis, the evidence 'points to' as opposed to merely 'leaves open' a link between injury, disease or death and the relevant service. In a reasonable hypothesis, the link is not 'obviously fanciful, impossible, incredible or not tenable or too remote or too tenuous.[2]

9. If Council is of the opinion that a reasonable hypothesis has been raised, the Council proceeds also to determine whether a connection exists to relevant service on the balance of probabilities,[3] i.e. whether the connection is more probable than not.

10. In these Reasons the association for both the reasonable hypothesis test and the balance of probabilities test are respectively referred to as the 'relevant association'.

11. The Council exercises its scientific judgement in weighing the evidence about the relevant association.

Council's Decision on the Scope of Review

12. The Council was asked to review the information that was available to the RMA at the relevant times to determine whether the evidence supports the possible inclusion

2 See the full Federal Court decision in Repatriation Commission v Bey (1997) 79 FCR 364 which cited with approval these comments from Veterans' Review Board in Stacey (unreported 26 June 1985), all of which were in turn cited with approval in the Moore J decision at [33].

3 Relevant service in balance of probabilities statements of principles refers to non-operational service having regard to the various definitions applying to types of 'service' as defined in the VEA and the MRCA.

of new factors, and / or the contents of the Fibrosing Interstitial Lung Disease (FILD) and Asbestosis SoPs in respect to asbestos bodies, pleural plaques, and the duration of asbestos exposure. The Council was also asked to form a view on whether the information gathered by the RMA included all the medical science relevant to the disease. The Applicant provided references to some new information, and contended that the Council should undertake a more extensive search of the published literature to determine the completeness of the information.

13. The Council determined that the scope of the review was as follows:

- whether the collection of information gathered by the RMA, on which it based its decisions, and sent to the Council as the information subject to review, is in some way incomplete; and
- the possible inclusion of a new factor(s) / amendment of an existing factor(s) in the above SoPs in the same or similar terms as:
  - to have the presence of pleural plaques or asbestos bodies recognised as evidence that **asbestosis, fibrosing interstitial lung disease and pulmonary fibrosis** can be related to asbestos exposure, even without long asbestos exposure during service; and
  - more generally, a reduction in the duration of the asbestos exposure requirements in the SoPs for both conditions.

14. The Council advised the Applicant and the Commissions of its decision on the proposed scope of review, at the Hearing of Oral Submissions. The Council invited the Applicant and the Commissions to comment on the proposed scope of review either at the hearing or afterwards as arranged with the Council’s Secretariat.

15. No comments were received on the proposed scope of the review.

Written and Oral Submissions

16. The Council took into account all the submissions made to it, both written and oral. The Council's brief summaries of the respective submissions made by the Applicant and the Commissions are set out at **Appendix C.**

COUNCIL’S EVALUATION OF THE SOUND MEDICAL-SCIENTIFIC EVIDENCE

17. In forming its decisions on the SMSE, the Council brings to bear its scientific expertise and judgement. The Bradford Hill criteria and other tools or criteria taken into account by epidemiologists were applied to the articles as the Council considered appropriate.

18. The Council also considered any methodological limitations or flaws (including such things as statistical power, control of confounders, bias, exposure assessment methods etc.) in the various articles.

19. For ease of reference, the Bradford Hill criteria (noting that these are not exhaustive) are:

- strength of association
- consistency across investigation
- specificity of the association
- temporal relationship of the association
- biological gradient
- biological plausibility
- coherence
- experiment
- analogy

20. The Council notes that these criteria are not necessary conditions of a cause and effect relationship. They act to provide some evidence of such a relationship.

21. While the Council considered, it did not focus its evaluation on those articles that did not provide data that the Council could draw conclusions on about the matters in scope (see [6]).

22. The Council took into account the submissions on the relevant SMSE made by both the Applicant and the Commissions.

23. The Council's decision on the relevant SMSE was that it should comprise the evidence, available to (before) the RMA, found in footnotes and endnotes in the Council's evaluation of the relevant SMSE in this document.

24. Information which the RMA advised was not available to it at the relevant times was not taken into account by the Council for the purposes of the review, as it could only

be considered as 'new information'. The Council identified from its own knowledge new information that would likely be relevant SMSE concerning asbestos-related pulmonary diseases, including FILD and Asbestosis (see **Appendix A. Table A4**).

25. Therefore, the Council considered the SMSE concerning the Applicant's contentions as set out by the Council in the scope of review (above), including SMSE relevant to the:

    a. Identical existing factors for clinical onset for both FILD and Asbestosis (see Table 1 below). That is "inhaling respirable asbestos fibres in":

        i. an enclosed space; and
        ii. an open environment.

    Each of these factors provides different cumulative periods of hours (equivalent to cumulative duration) and different latency periods for the reasonable hypothesis (RH) SoPs and the balance of probabilities (BoP) SoPs; and

    b. Existing clinical worsening factors for the Asbestosis[4] SoPs only (see below) are also for "inhaling respirable asbestos fibres", with different cumulative periods of hours and different latency periods for the RH SoPs and the BoP SoPs.

Preliminary Comment on Fibrosing Interstitial Lung Disease and Asbestosis

26. The Council noted that in addition to FILD and Asbestosis, there are other SoPs, which include asbestos-related factors. These are the SoPs for Pleural Plaque, Mesothelioma, and Lung Cancer, and each have similar exposure factors.

27. In the FILD[5] and Asbestosis[6] SoPs the RMA defines the diseases as:

> … "fibrosing interstitial lung disease" means one of a diverse group of lung diseases that are characterised by progressive fibrosis of the pulmonary interstitium with or without chronic inflammation. This definition excludes extrinsic allergic alveolitis,

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4 RMA. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013.

5 RMA. Statements of Principles No. 53 and 54 of 2013 for Fibrosing Interstitial Lung Disease. Brisbane, Australia: 2013.

6 RMA. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013.

bronchiolitis obliterans organising pneumonia, desquamative interstitial pneumonia, respiratory bronchiolitis-associated interstitial lung disease and pulmonary manifestations of systemic diseases.[7(p.1)]

… "asbestosis" means a form of lung disease caused by the deposition of asbestos fibres in the lung parenchyma, marked by bilateral interstitial fibrosis of the lung.[8(p.1)]

28. FILD resulting from asbestos fibre inhalation is defined as asbestosis, which leads to the application of the Asbestosis SoPs. However, for those patients with extensive asbestos exposure and a high suspicion of asbestosis, who do not have the classical radiological and or pathological features despite numerous investigations and assessments, the FILD SoPs would likely apply.

29. The Commissions' written submission[9] provided the following explanation on the operational use of the two SoPs:

The current situation, with asbestos-related pulmonary fibrosis covered by both the asbestosis and FILD SoPs, is now one of the only exceptions to the one disease – one SoP principle.[10(p.5)]

If a diagnosis of asbestosis can be established on the balance of probabilities, then the asbestosis SoP would be applied. If asbestosis cannot be so confirmed but fibrosing interstitial lung disease is present, then the FILD SoP would be applied (provided none of the conditions excluded by the SoP definition were present).[11(p.5)]

30. Terms relating to asbestos-related diseases are set out below:

Asbestosis is the scarring of lung tissue (beginning around terminal bronchioles and alveolar ducts and extending into the alveolar walls) resulting from the inhalation of asbestos fibres. Asbestosis specifically refers to interstitial (parenchymal) fibrosis from asbestos, and not pleural fibrosis or pleural plaques.[12] It is defined as diffuse interstitial pulmonary fibrosis

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7 RMA. Statements of Principles No. 53 and 54 of 2013 for Fibrosing Interstitial Lung Disease. Brisbane, Australia: 2013.

8 RMA. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013.

9 Repatriation Commission and the Military Rehabilitation and Compensation Commission. Submission by the Commission to the Specialist Medical Review Council on Asbestosis & Fibrosing Interstitial Lung Disease. Canberra, Australia: Australian Government Department of Veteran Affairs; 2014 April.

10 Repatriation Commission and the Military Rehabilitation and Compensation Commission. 2014 April. *ibid.*

11 Repatriation Commission and the Military Rehabilitation and Compensation Commission. 2014 April. *ibid.*

12 American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

secondary to asbestos exposure.[13] Asbestosis is recognised as having a dose-response relationship.[14, 15]

Pleural plaques are localised areas of pleural thickening usually affecting the parietal pleura.[16] They are related to the type of asbestos and dose.[17] Pleural plaques become more radiologically evident with time, typically 20 years or more after the inhalation of asbestos fibres.[18]

Diffuse pleural thickening is a different entity from pleural plaques, although it can co-exist with pleural plaques. Asbestos-related diffuse pleural thickening refers to extensive fibrosis of the visceral rather than the parietal pleura, with adherence to the parietal pleura and obliteration of the pleural space. It is more dose-related than pleural plaques and is probably also affected by individual susceptibilities.[19] It may occur with higher asbestos exposure and may be accompanied by asbestosis itself. When present, it may be difficult to diagnose accompanying asbestosis in these cases because the diffuse pleural thickening obscures the lung parenchyma on plain chest radiology, and high resolution computed tomography (CT) scanning is needed.[20] Clinically, diffuse pleural thickening decreases the audibility of the fine basal crackles. In this condition, there may also occur several features (e.g. parenchymal bands), which are easily confused with asbestosis.[21]

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13 Prazakova S, Thomas PS, Sandrini A, Yates DH. Asbestos and the lung in the 21st century: an update. Clin Respir J. 2014;8(1):1-10. (New Information).

14 Wagner JC, Moncrieff CB, Coles R, Griffiths DM, Munday DE. Correlation between fibre content of the lungs and disease in naval dockyard workers. Br J Ind Med. 1986;43(6):391-95. Cited by: Roggli VL, Gibbs AR, Attanoos R, Chung A, Popper H, Cagle P, et al. Pathology of asbestosis - An update of the diagnostic criteria: Report of the asbestosis committee of the College of American Pathologists and Pulmonary Pathology Society. Arch Pathol Lab Med. 2010;134(3):462-80. (RMA ID: 067548).

15 Roggli VL. Scanning electron microscopic analysis of mineral fiber content of lung tissue in the evaluation of diffuse pulmonary fibrosis. Scanning Microsc Suppl. 1991;5(1):71-83. Cited by: Roggli et al. Pathology of asbestosis - An update of the diagnostic criteria: Report of the asbestosis committee of the College of American Pathologists and Pulmonary Pathology Society. Arch Pathol Lab Med. 2010;134(3):462-80. (RMA ID: 067548).

16 Industrial Injuries Advisory Council. Position Paper #23. Pleural plaques. London, UK. 2009. Available from: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/328553/iiac-pp23.pdf (New Information).

17 Industrial Injuries Advisory Council. Position Paper #23. London, UK. 2009. (New Information). *ibid.*

18 American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

19 Schwartz DA. New developments in asbestos-induced pleural disease. Chest 1991;99(1):191-98. Cited by: American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

20 Roach HD, Davies GJ, Attanoos R, Crane M, Adams H, Phillips S. Asbestos: when the dust settles an imaging review of asbestos-related disease. RadioGraphics. 2002;22(Suppl 1):S167-84. (New Information).

21 Roach et al. 2002. (New Information). *ibid.*

Benign asbestos pleural effusion is an exudative pleural effusion (a build-up of fluid between the two pleural layers) following asbestos exposure.[22] It is relatively uncommon and is usually the earliest manifestation of disease following asbestos exposure, typically occurring within 10 years from exposure.[23] Effusions may be asymptomatic, or cause pain, fever, and breathlessness.[24] Effusions usually last for 3-4 months and then resolve completely.[25] They can also progress to diffuse pleural thickening.[26] Diagnosis relies on a compatible history of asbestos exposure and exclusion of other probable causes.[27]

31. The Council noted that there have been changes in the understanding and use of the terminology for asbestos-related diseases over time. This may account for some of the difficulties in classification between different diseases. “Pleural asbestosis” is an obsolete term used in early studies, and is not equivalent to asbestosis as understood in this document, but rather refers to pleural plaques and/or diffuse pleural thickening. Diffuse pleural thickening is well described as a separate entity in the medical literature, and should be considered distinct from multiple pleural plaques. In many studies in the epidemiological literature, the term “parenchymal fibrosis” is often used as a substitute for “asbestosis”.

32. The Council also notes that updated diagnostic criteria for the diagnosis of asbestosis were published in 2010 by the Asbestosis Committee of the College of American Pathologists and Pulmonary Pathology Society.[28] One area of potential confusion is the acceptance of some pathologists of bronchiolar wall fibrosis in the absence of alveolar septal fibrosis as early asbestosis. The Asbestosis Committee believe that this bronchiolar wall fibrosis should not be referred to as asbestosis but

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22 Henderson DW, Jones ML, De Lerk N, Leigh J, Musk AW, Shilkin KB, et al. The diagnosis and attribution of asbestos-related diseases in an Australian context. Report of the Adelaide Workshop on Asbestos-related Diseases October 6-7, 2000. Int J Occup Med Environ Health. 2004;10(1):40-6. (RMA ID: 035222).

23 Chapman SJ, Cookson WOC, Musk AW, Lee YCG. Benign asbestos pleural diseases. Curr Opin Pulm Med. 2003;9(4):1-5. (New Information).

24 American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

25 Prazakova et al. Asbestos and the lung in the 21st century: an update. Clin Respir J. 2014;8(1):1-10. (New Information).

26 Epler GR, McLoud TC, Gaensler EA. Prevalence and incidence of benign asbestos pleural effusion in a working population. J Am Med Assoc. 1982;247(5):617-22. Cited by: American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

27 Chapman et al. Benign asbestos pleural diseases. Curr Opin Pulm Med. 2003;9(4):1-5. (New Information).

28 Roggli et al. Pathology of asbestosis - An update of the diagnostic criteria: Report of the asbestosis committee of the College of American Pathologists and Pulmonary Pathology Society. Arch Pathol Lab Med. 2010;134(3):462-80. (RMA ID: 067548).

as “asbestos airway disease”. This has also been called mineral dust airway disease. Similar fibrosis can be seen with a variety of dusts such as silica, iron, aluminium oxide and cigarette smoke as well as asbestosis. This area continues to be one of controversy and is relevant to the clinical diagnosis of asbestosis in that this type of fibrosis can produce small opacities visible on chest imaging, and may be one of the causes of false positive diagnoses.

33. An illustration of different types of asbestos-related pulmonary disease is shown in Figure 1.[29] Figure 1A shows the location of the lungs, airways, pleura, and diaphragm and Figure 1B shows asbestos-related diseases, including pleural plaques, asbestosis, lung cancer, and mesothelioma.

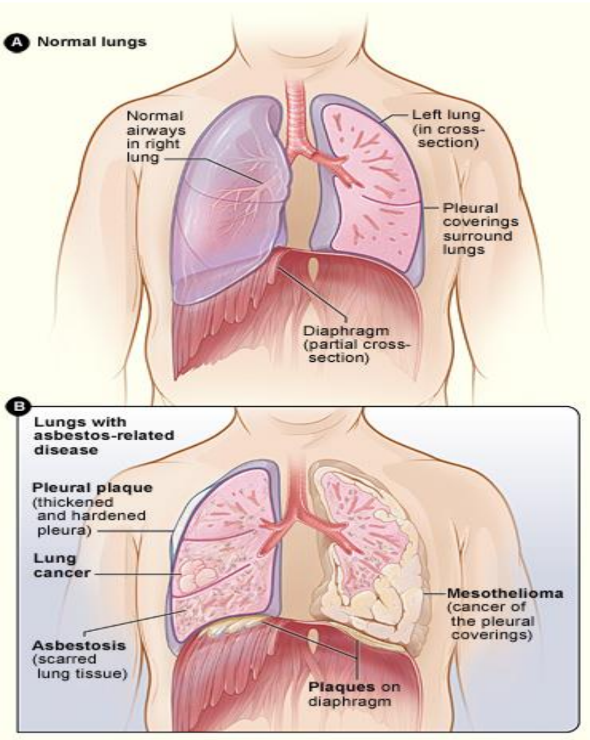

FIGURE 1. ASBESTOS-RELATED DISORDERS

29 Asbestos-Related Lung Diseases [Internet]. National Heart, Lung, and Blood Institute, NIH (US) 2014 [cited 2015]. [Figure 1]. Available from: http://www.nhlbi.nih.gov/health/health-topics/topics/asb (New Information).

34. In Figure 2[30] diffuse pleural thickening is shown.

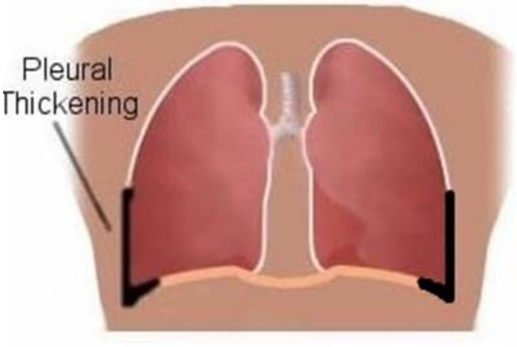

FIGURE 2. DIFFUSE PLEURAL THICKENING

35. The Council noted a number of other issues about the medical science studies concerning the level of exposure to asbestos required to produce FILD and Asbestosis. Despite much published research where respirable fibre levels have been assessed, measurement of actual exposure is difficult. In the various studies, methodologies have differed, the types of asbestos inhaled have varied, and other factors may not have been considered such as smoking, and other dusts inhaled. Different measurement methods also make comparisons between levels of exposure difficult.

36. The Council notes that the SMSE usually refers to fibre/mL/years to quantify asbestos exposure rather than simply chronological exposure duration. The Council's discussion regarding this is at [57].

COUNCIL'S EVALUATION OF SOUND MEDICAL-SCIENTIFIC EVIDENCE RELEVANT TO THE APPLICANT'S CONTENTIONS

The Relationship between Pleural Plaques and / or Asbestos Bodies and Fibrosing Interstitial Lung Disease and Asbestosis

37. Inhalation of asbestos fibres can cause fibrotic interstitial lung disease (asbestosis), pleural plaques, diffuse pleural thickening, malignant mesothelioma, and lung

30 Asbestos Victim Advice. Pleural Thickening. Asbestos related pleural thickening. [Internet]. WE Solicitors LLP - Asbestos compensation and claim lawyers and Mesothelioma compensations and claims [cited 2015]. [Figure 1]. Available from: http://asbestosvictimadvice.com/pleural-thickening/ (New Information).

cancer.[31] The interaction of asbestos fibres with alveolar macrophages in the lungs, in some instances, results in the formation of asbestos bodies, which are a marker of past exposure to asbestos fibres.[32] The detection of asbestos fibres (or asbestos bodies) in lung tissue, sputum, or bronchial washings/ lavage fluid (BAL) are a definite indication of asbestos exposure. A histological diagnosis of asbestosis is regarded as the gold standard for diagnosis, for which the American College of Pathologists has recently published criteria.[33] However, the uses of techniques such as surgically obtained lung biopsies are considered too invasive to be performed solely for compensation purposes. BAL is more available and less invasive, but laboratory expertise in identifying asbestos bodies is operator dependant and this skill is becoming rare.

38. Pleural plaques are the most common manifestation of the inhalation of asbestos fibres.[34] The presence of pleural plaques is an indicator of exposure to asbestos fibres.[35] Localised pleural plaques are usually asymptomatic, although diffuse pleural thickening is associated with decreased respiratory function.[36]

39. Diffuse pleural thickening is another marker of asbestos exposure. Diffuse pleural thickening, or extensive fibrosis of the visceral pleura secondary to asbestos exposure, may coexist with pleural plaques but has a distinctly different pathology with a different natural history, radiology and prognosis.[37] In contrast, pleural plaques primarily involve the parietal pleura.[38] Diffuse pleural thickening can develop within a year from exposure to asbestos often following a benign asbestos related pleural effusion.[39] Although symptoms are generally mild, severe restrictive lung disease

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[31] Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for asbestos. 035227. Atlanta, GA: U.S. Department of Health and Human Services. Public Health Service - ATSDR, 2001. (RMA: 035227).

[32] Roggli VL. Fiber Analysis. In: Rom WN, editor. Environmental & Occupational Medicine. 3rd ed. Philadelphia: Lippincott-Raven; 1998. p. 335-45. (RMA ID: 033719).

[33] Roggli et al. Pathology of asbestosis - An update of the diagnostic criteria: Report of the asbestosis committee of the College of American Pathologists and Pulmonary Pathology Society. Arch Pathol Lab Med. 2010;134(3):462-80. (RMA ID: 067548).

[34] American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

[35] Anonymous (International Expert Meeting). Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health. 1997;23(4):311-16. (RMA ID: 026517).

[36] American Thoracic Society. 2004. (RMA ID: 067594). *ibid.*

[37] Miles SE, Sandrini A, Johnson AR, Yates DH. Clinical consequences of asbestos-related diffuse pleural thickening: A review. J Occup Med Toxicol. 2008;3:20. (New Information).

[38] Miles et al. 2008. (New Information). *ibid.*

[39] Miles et al. 2008. (New Information). *ibid.*

with respiratory failure can rarely occur.[40] The Council noted that the presence of diffuse pleural thickening rather than pleural plaques is more frequently associated with underlying asbestosis, but a history of substantial asbestos exposure is required before a diagnosis of asbestosis and or FILD can be made. Pleural plaques, diffuse pleural thickening and asbestosis can be distinguished from each other using internationally accepted evidence-based criteria.[41, 42, 43]

40. The American Thoracic Society[44] reported that for a diagnosis of non-malignant asbestos-related disease (asbestosis; pleural thickening or asbestos-related pleural fibrosis - plaques or diffuse fibrosis; non-malignant pleural effusion; and airflow obstruction) there must be:

    i. evidence of structural pathology consistent with asbestos-related disease as documented by imaging or histology;

    ii. evidence of causation by asbestos as documented by the occupational and environmental history, markers of exposure (usually pleural plaques), recovery of asbestos bodies, or other means; and

    iii. exclusion of alternative plausible causes for the findings.[45 (p.691)]

41. Similarly, the International Expert Meeting on Asbestos, Asbestosis, and Cancer [46] stated:

> Asbestosis is defined as diffuse interstitial fibrosis of the lung as a consequence of exposure to asbestos dust. Neither the clinical features nor the architectural tissue abnormalities sufficiently differ from those of other causes of interstitial fibrosis to allow confident diagnosis without a history of significant exposure to asbestos dust in the past or the detection of asbestos fibers or bodies in the lung tissue greatly in excess of that commonly seen in the general population. Symptoms of asbestosis include dyspnea and cough. Common findings are inspiratory basilar crackles and,

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40 American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

41 American Thoracic Society. 2004. (RMA ID: 067594). *ibid.*

42 Anonymous (International Expert Meeting). Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health. 1997;23(4):311-16. (RMA ID: 026517).

43 International Labour Office. Guidelines for the use of the ILO International Classification of Radiographs of Pneumoconioses. Occupational Safety and Health Series, No 22 Revised edition 2000. Cited by: American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715 (RMA ID: 067594).

44 American Thoracic Society. 2004. (RMA ID: 067594). *ibid.*

45 American Thoracic Society. 2004. (RMA ID: 067594). *ibid.*

46 Anonymous (International Expert Meeting). 1997. (RMA ID: 026517). *ibid.*

less commonly, clubbing of the fingers. Functional disturbances can include gas exchange abnormalities, a restrictive pattern, and obstructive features due to small airway disease.[47] (p.312)

Asbestosis is generally associated with relatively high exposure levels with radiological signs of parenchymal fibrosis. However, it is possible that mild fibrosis may occur at lower exposure levels, and the radiological criteria need not always be fulfilled in cases of histologically detectable parenchymal fibrosis. The recognition of asbestosis by chest radiography is best guided by standardized methods such as the classification of the International Labour Organisation (ILO) and its modifications. Standard films must always be used. For research and screening purposes, radiological findings of small opacities, grade 1/0, are usually regarded as an early stage of asbestosis.[48] (p.312)

42. Fischer et al[49] reported that the concentration of asbestos fibres in lung tissue is the result of:

- the intensity and duration of exposure;
- geometry, type, and bio persistence of fibres;
- inhalation, elimination, and retention of fibres; and
- individual factors such as existing lesions, smoking, breathing frequency and volume, and work environment.

Light or electron microscopical lung-dust analysis with counts of asbestos bodies or asbestos fibres, are often conducted in order to quantify the actual pulmonary burden.[50]

43. The concentrations of asbestos fibres in the air are often reported as fibres in a millilitre (mL) i.e. fibre/mL. Inhalation exposure is generally regarded as cumulative and is expressed as concentration of fibres over time i.e. fibre/mL-years, noting that some asbestos fibres may be removed by mucociliary clearance or macrophages, while others may be retained in the lungs for extended periods.[51] Therefore, to

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47 Anonymous (International Expert Meeting). Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health. 1997;23(4):311-16. (RMA ID: 026517).

48 Anonymous (International Expert Meeting). 1997. (RMA ID: 026517). *ibid.*

49 Fischer M, Günther S, Müller KM. Fibre-years, pulmonary asbestos burden and asbestosis. Int J Hyg Environ Health. 2002;205(3):245-48. (RMA ID: 026590).

50 Fischer et al. 2002. (RMA ID: 026590). *ibid.*

51 Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for asbestos. 035227. Atlanta, GA: U.S. Department of Health and Human Services. Public Health Service - ATSDR, 2001. (RMA: 035227).

calculate asbestos exposure as cumulative fibre exposure of fibre/mL-year, duration of exposure (measured in years) is multiplied by the average air concentration for the period of exposure (measured in fibre/mL).[52]

44. The Agency for Toxic Substances and Disease Registry (ATSDR)[53] reported that observations of asbestos-related diseases in groups of workers with cumulative exposures ranging from 5 to 1200 fibre/mL-years, resulting from 40 years of occupational exposure to air concentrations of 0.125 to 30 fibre/mL, have been shown. For the presence of pleural plaques, the ATSDR[54] reported that an increased incidence of pleural plaques has been noted at relatively low cumulative exposure levels of approximately 0.12 fibre/mL-years.

45. Ehrlich et al[55] studied the long-term radiological effects of short-term exposure to amosite[56] asbestos among a subset of 386 factory workers from an original cohort of 820 (median exposure = 6 months; range = 0.01-13) and long-term follow-up (median exposure = 25 years). Asbestos dust concentrations were high (mean (*M*) = 51.8 fibre/mL, (standard deviation (*SD)* = 25)) and the asbestos exposure was accumulated rapidly, over months in most cases rather than years (median = 25.1 fibre/mL-years, range = 0.1-720). With as little as one month or less of employment, approximately 20% of the X-rays showed parenchymal abnormality (ILO profusion ≥1/0) and about a third showed pleural abnormality (any pleural thickening of the chest wall or diaphragm). Those in the lowest cumulative exposure group (≤5 fibre/mL-years) were found to have high rates of parenchymal and pleural abnormalities. The prevalence rate of parenchymal abnormality increased with increasing cumulative exposure; however, for pleural abnormalities there was no clear trend with increasing cumulative exposure.

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52 Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for asbestos. 035227. Atlanta, GA: U.S. Department of Health and Human Services. Public Health Service - ATSDR, 2001. (RMA: 035227).

53 Agency for Toxic Substances and Disease Registry (ATSDR). 2001. (RMA: 035227). *ibid.*

54 Agency for Toxic Substances and Disease Registry (ATSDR). 2001. (RMA: 035227). *ibid.*

55 Ehrlich R, Lilis R, Chan E, Nicholson WJ, Selikoff IJ. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

56 Amosite (sometimes referred to as brown asbestos) is amphibole asbestos. Amphibole asbestos has crystalline fibres that are long, thin, straight and generally brittle. Cited by: Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for asbestos. 035227. Atlanta, GA: U.S. Department of Health and Human Services. Public Health Service - ATSDR, 2001. (RMA: 035227).

46. The authors[57] concluded that no cumulative exposure threshold for parenchymal and pleural abnormalities was detectable, and progression was still evident 20 years or more after the end of exposure. This finding showed that radiological abnormalities indicative of interstitial pulmonary fibrosis and pleural thickening can develop with as little as one month's exposure to high concentrations of amosite asbestos fibre. The authors stated “that under certain circumstances, a high dose of asbestos fibre inhaled in a short period of time is more potent than the same cumulative exposure inhaled over a longer interval”.[58(p.274)]

47. Shepherd et al[59] examined progression of pleural and parenchymal disease on chest X-rays of 887 workers exposed to amosite asbestos. The exposure was short-term (*M* = 8.0 months, *SD* = 14.9), but high (*M* = 638.9 fibre/mL-months, *SD* = 1460 *i.e. M* = 53.3 fibre/mL-years). The study showed parenchymal disease doubled over approximately 10 years between X-rays with only slightly less progression for pleural disease, and that an intense although short exposure to amosite asbestos can produce pleural and parenchymal changes on chest radiographs.

48. Koskinen et al[60] screened 18 943 Finnish construction (*n* = 17 937), shipyard (*n* = 456), and asbestos industry (*n* = 550) workers for asbestos-related radiographic abnormalities. Of the screened workers, 4133 (22%) had asbestos-related radiographic abnormalities (22% construction; 16% shipyards; 24% asbestos industry). For all workers combined, the average duration of employment was 26 years, and the average duration of exposure was 9.0 years (construction *M* = 9.0 years; shipyards *M* = 7.8 years; and asbestos industry *M* = 9.7 years). The prevalence of positive findings increased with the duration of exposure (18% = <10 years; 27% = 10-30 years; and 43% = >30 years).

49. Of the 4133 workers with asbestos-related radiographic abnormalities present, 96% had abnormalities in the pleura and 4% had asbestosis. Pleural plaques were diagnosed in two-thirds of the workers with parenchymal fibrosis and they were observed in less than half of workers with small irregular opacities and less than 15

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57 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

58 Ehrlich et al. 1992. (RMA ID: 034901). *ibid.*

59 Shepherd JR, Hillerdal G, McLarty J. Progression of pleural and parenchymal disease on chest radiographs of workers exposed to amosite asbestos. J Occup Environ Med. 1997; 54(6):410-15. (RMA ID: 035757).

60 Koskinen K, Zitting A, Tossavainen A, Rinne JP, Roto P, Kivekäs J, et al. Radiographic abnormalities among Finnish construction, shipyard and asbestos industry workers. Scand J Work Environ Health. 1998;24(2):109-17. (RMA ID: 026513).

years of exposure. Additionally, after a period of exposure of more than 30 years, pleural plaques were present in 80% of cases. This study showed that parenchymal fibrosis developed after shorter and possibly greater asbestos exposure, whereas pleural plaques require a longer period to become detectable by chest X-ray.[61]

50. Schaeffner et al[62] assessed the association between asbestos exposure and pleural or parenchymal abnormalities on chest X-rays or CT scans of 103 asbestos-exposed patients with known lung cancer. Asbestos exposure was assessed using an asbestos exposure index that integrated time and intensity of reported exposure using a weighting score; low asbestos exposure index score = ≤10 and a high asbestos exposure index score = >10. There were 34 patients with a low exposure index score (≤10) and 69 had high exposure index score (>10) and a statistically significant correlation between exposure and chest CT scan changes in detecting asbestos-related abnormalities was shown. The group with the higher asbestos exposure index score were more likely to develop both pleural and parenchymal abnormalities (Odds ratio (OR) = 4.93; 95% confidence interval (CI): 1.05, 23.12). This study confirmed that chest CT scans were more sensitive in detecting pleural or parenchymal abnormalities than were standard chest X-rays and there was a significant correlation between higher asbestos exposure index scores and abnormalities on CT scans.

51. Rohs et al[63] conducted a 25 year follow-up study of low-level fibre-induced radiographic changes caused by Libby Vermiculite[64] of 280 participants. The average cumulative amphibole[65] exposure was low (M = 2.48 fibre/mL-years (SD = 4.19);

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61 Koskinen et al. Radiographic abnormalities among Finnish construction, shipyard and asbestos industry workers. Scand J Work Environ Health. 1998;24(2):109-17. (RMA ID: 026513).

62 Schaeffner ES, Miller DP, Wain JC, Christiani DC. Use of an asbestos exposure score and the presence of pleural and parenchymal abnormalities in a lung cancer case series. Int J Occup Med Environ Health. 2001; 7(1):14-18. (RMA ID: 026602).

63 Rohs AM, Lockey JE, Dunning KK, Shukla R, Fan H, Hilbert T, et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008; 177(6):630-37. (RMA ID: 067995).

64 Raw vermiculite is a mica-like mineral that expands rapidly upon heating, producing a lightweight, bulky material that is used in fireproofing, insulation, packaging, and in horticultural/agricultural products (as a soil conditioner, fertilizer carrier). Vermiculite can contain large amounts of tremolite, which is amphibole asbestos. One of the largest vermiculite deposits in the United States is in Libby, Montana, where raw vermiculite was mined and milled from 1923 until 1990. Cited by: Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for asbestos. 035227. Atlanta, GA: U.S. Department of Health and Human Services. Public Health Service - ATSDR, 2001. (RMA: 035227).

65 There are two groups of asbestos minerals; serpentine asbestos (chrysotile) and amphibole asbestos (amosite, crocidolite, and fibrous forms of tremolite, anthophyllite, and actinolite). Amphiboles are cleared less readily and are more persistent in the lungs than chrysotile serpentine asbestos. Cited by: Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for asbestos. 035227. Atlanta, GA: U.S. Department of Health and Human Services. Public Health Service - ATSDR, 2001. (RMA: 035227).

range = 0.01-19.03). A total of 80 participants had pleural changes (64 = localised pleural thickening only – pleural plaques; 10 = diffuse pleural thickening only; and 6 = both pleural thickening (4 = localised and 2 = diffuse) with interstitial changes) and 8 had interstitial changes (irregular opacities, profusion ≥1/0). Participants with any pleural changes (n = 80) had significantly greater cumulative fibre exposure compared with the 200 participants without pleural changes (M = 4.77 fibre/mL-years (SD = 5.72) and M = 1.56 (SD = 2.94); p< 0.001 respectively). Pleural changes were directly related to cumulative fibre exposure, with the greatest prevalence (54.3%) in the highest exposure quartile (range = 2.21-19.03 fibre/mL-years) of participants. The prevalence of pleural changes increased with age, including those workers with a lifetime cumulative fibre exposure of <1 fibre/mL-year. A significant increase in pleural changes overtime from 2.0% in the 1980 study to 28.7% in 2005 was shown.

52. In this study, interstitial changes (irregular opacities) were demonstrated in 2.9% of participants and were significantly related to cumulative fibre exposure. Participants with interstitial changes (n = 8) had significantly greater cumulative fibre exposure compared with the 198 participants without pleural changes (M = 11.86 fibre/mL-years (SD = 6.46); p< 0.001) and the 64 with only localised pleural changes (p< 0.001). A significant increase in interstitial changes over time from 0.2% in the 1980 study to 2.9% in 2005 was shown. This study demonstrated that exposure to asbestos fibres among users of Libby vermiculite ore caused pleural thickening and interstitial changes at low lifetime cumulative fibre exposure levels of <2.21 and 11.37 fibre/mL-years respectively. Rohs et al[66] reported that a level of <2.21 fibre/mL-years is below the lifetime cumulative fibre exposure for a worker exposed to the current Occupational Safety and Health Administration permissible exposure level standard of 0.1 fibre/mL for regulated asbestos in general industry over a 45-year working life (4.5 fibre/mL-years).

53. Kishimoto et al[67] examined the prevalence of pleural plaques and/or pulmonary changes, on chest X-rays and confirmed by chest CT, among construction workers in Japan. Among 2951 workers, 168 (5.7%) had significant findings for pleural plaques or pulmonary changes on chest X-ray, (74 = both pleural plaques and asbestosis; 85 = pleural plaques alone; and 9 = asbestosis alone). Pleural plaques were present in 5.4%, and fibrosis suggesting pulmonary asbestosis in 2.8% of chest CT images.

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66 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008; 177(6):630-37. (RMA ID: 067995).

67 Kishimoto T, Morinaga K, Kira S. The prevalence of pleural plagues and/or pulmonary changes among construction workers in Okayama, Japan. Am J Ind Med. 2000;37(3):291-95. (RMA ID: 026494).

Workers with pulmonary asbestosis accompanied by pleural plaques accounted for a considerably high percentage; there was no correlation between the lesions. More advanced age and a longer duration of asbestos exposure appeared to be associated with a higher percentage of workers with pulmonary and pleural lesions. The study demonstrated that pleural plaques develop even after exposure to a small amount of asbestos, but often do not develop until 20 years after the initial exposure.

Summary of Council's View

54. On the basis of its evaluation of the studies discussed above, the Council considered that the presence of either asbestos bodies or pleural plaques (and diffuse pleural thickening) were indicative of prior asbestos exposure. However, the Council did not find evidence that the presence of either of these markers alone, without a stated asbestos exposure level and duration, and suitable lag period, would indicate that the disease asbestosis was also present.

55. The Council recommends that the RMA considers whether diffuse pleural thickening should be specifically included in the SoPs regime as a compensable disease, possibly by recognising it as a separate disease.

New Information on the Relationship between Pleural Plaques and / or Asbestos Bodies and Fibrosing Interstitial Lung Disease and Asbestosis

56. The Council noted there is new information available on the relationship between pleural plaques, asbestos bodies, and asbestosis (see **Appendix A. Table A4**) and in relation to diffuse pleural thickening (see **Appendix A. Table A5**).

Comparison of the Statements of Principles, where Duration of Exposure is used as a Measure of Dose, with the Literature on a Threshold of Asbestos Concentrations

57. In previous RMA reviews in 1996[68, 69] and 2005[70] the required level of asbestos exposure associated with producing FILD or Asbestosis expressed as days was reduced, and exposure expressed as days changed to hours of exposure.

a. In the SoPs for Asbestosis for 1996[71] the minimum period of exposure to asbestos fibres was changed from 500 days (RH and BoP) to 200 days (RH) and 365 days (BoP). The rationale for the change was the 500 days minimum period of exposure was derived from occupational studies (15 studies/reviews,[72] particularly studies of Wittenoom mine workers and insulators by de Klerk et al[73] and Cookson et al)[74] and the dose was lowered to account for naval personnel who are often on board ship for 24 hours per day, seven days per week.[75]

b. In the SoPs for Asbestosis for 2005[76] the minimum period of exposure was changed from 200 days (RH) and 365 days (BoP) to 1000 hours (RH) and 1500 hours (BoP) in an enclosed environment and 3000 hours (RH) and 5000 hours (BoP) in an open environment. In making these changes

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68 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 11 and 12 of 1996 for Asbestosis. Brisbane, Australia: 1996.

69 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 138 and 139 of 1996 for Asbestosis. Brisbane, Australia: 1996.

70 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 23 and 24 of 2005 for Asbestosis. Brisbane, Australia: 2005.

71 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 138 and 139 for Asbestosis. Brisbane, Australia: 1996.

72 See Appendix A. Table A3 for 1996: Speizer 1994 [2005] (RMA ID: 46975); Roggli [1992] 1998 (RMA ID: 033719); Rom [1992] 1998 (RMA ID: 33719); Becklake & Case 1994 (RMA ID: 430); Churg & Vedal 1994 (RMA ID: 431); Murai et al 1994 (RMA ID: 437); Klass 1993 (RMA ID: 435); Gaensler 1992 (RMA ID: 434); Becklake 1991 (RMA ID: 429); de Klerk et al 1991 (RMA ID: 432); Sluis-Cremer 1991 (RMA ID: 440); Sluis-Cremer & Hnizdo 1989 (RMA ID: 035796); Cookson et al 1986 (RMA ID: 455); Finkelstein & Vingilis 1984 (RMA ID: 454); and Murphy et al 1971 Cited by: Gaensler 1992 (RMA ID: 434).

73 de Klerk NH, Musk AW, Armstrong BK, Hobbs MS. Smoking, exposure to crocidolite, and the incidence of lung cancer and asbestosis. Br J Ind Med. 1991;48(6):412-17. (RMA ID: 000432).

74 Cookson WO, de Klerk N, Musk W, Glancy JJ, Armstrong B, Hobbs M. The Natural history of asbestosis in former crocidolite workers in Wittenoom Gorge. Am Rev Respir Dis. 1986; 133(6):994-98. (RMA ID: 000445).

75 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 23 and 24 of 2005 for Asbestosis. Brisbane, Australia: 2005.

76 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 23 and 24 of 2005 for Asbestosis. Brisbane, Australia: 2005.

the RMA evaluated its previous evidence,[77] further relevant SMSE[78] and responded to submissions contending:

i. an exposure of 200 days was excessive for naval personal exposed 24 hours, 7 days a week when at sea; and
ii. exposure should be expressed in hours rather than days as is the case with passive smoking.

c. The RMA 2005 SoPs[79] changed the minimum period of exposure from days to hours as an attempt " to make up a time based dose "in a vacuum" as the actual or potential levels of exposure experienced by personnel is open to conjecture".[80(p.44)] The RMA took into account the previous 1996 studies and in 2005 an additional 13 studies/reviews - three review studies, seven cohort studies, two case series, and one case report, particularly studies by Wright et al[81] and Fischer et al,[82] a book chapter by Roggli,[83] and reviews by ATSDR[84] and Henderson et al.[85]

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77 See Appendix A. Table A3 for 1996: Speizer 1994 [2005] (RMA ID: 46975); Roggli [1992] 1998 (RMA ID: 033719); Rom [1992] 1998 (RMA ID: 33719); Becklake & Case 1994 (RMA ID: 430); Churg & Vedal 1994 (RMA ID: 431); Murai et al 1994 (RMA ID: 437); Klass 1993 (RMA ID: 435); Gaensler 1992 (RMA ID: 434); Becklake 1991 (RMA ID: 429); de Klerk et al 1991 (RMA ID: 432); Sluis-Cremer 1991 (RMA ID: 440); Sluis-Cremer & Hnizdo 1989 (RMA ID: 035796); Cookson et al 1986 (RMA ID: 455); Finkelstein & Vingilis 1984 (RMA ID: 454); and Murphy et al 1971 Cited by: Gaensler 1992 (RMA ID: 434).

78 See Appendix A. Table A3 for 2005: Henderson et al 2004 (RMA ID: 035222); Burdorf et al 2003 (RMA ID: 035752); Fischer et al 2002 (RMA ID: 026590); Szeszenia-Dabrowska et al 2002 (RMA ID: 028160); Wright et al 2002 (RMA ID: 026507); ATSDR 2001 (RMA ID: 035227); Schaeffner et al. 2001 (RMA ID: 026602); Kurumatani et al 1999 (RMA ID: 026552); Boffetta 1998 (RMA ID: 026600); Levin et al 1998 (RMA ID: 026470); Shepherd et al 1997 (RMA ID: 035757); Ehrlich et al 1992 (RMA ID: 034901); and Selikoff et al 1990 (RMA ID: 035792).

79 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 23 and 24 of 2005 for Asbestosis. Brisbane, Australia: 2005.

80 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 23 and 24 of 2005 for Asbestosis. Brisbane, Australia: 2005.

81 Wright RS, Abraham J, Harber P, Burnett BR, Morris P, West P. Fatal asbestosis 50 years after brief high intensity exposure in a Vermiculite expansion plant. Am J Respir Crit Care Medi. 2002;165(8):1145-49. (RMA ID: 026507).

82 Fischer et al. Fibre-years, pulmonary asbestos burden and asbestosis. Int J Hyg Environ Health. 2002; 205(3):245-48. (RMA ID: 026590).

83 Roggli VL. Fiber Analysis. In: Rom WN, ed. Environmental & Occupational Medicine. 3rd ed. Philadelphia: Lippincott-Raven; 1998. p. 335-45. (RMA ID: 033719).

84 Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for asbestos. 035227. Atlanta, GA: U.S. Department of Health and Human Services. Public Health Service -ATSDR, 2001. (RMA: 035227).

85 Henderson et al. The diagnosis and attribution of asbestos-related diseases in an Australian context. Report of the Adelaide Workshop on Asbestos-related Diseases October 6-7, 2000. Int J Occup Med Environ Health. 2004;10(1):40-6. (RMA ID: 035222).

d. The 2013 SoPs for Asbestosis[86] retained the minimum period of exposure set out in the 2005 SoPs,[87] after the RMA reviewed the 1996, 2005 studies and an additional 7 reviews/books and 14 studies.[88]

58. In the SMSE, a cumulative asbestos exposure of 25 fibre/mL-years is commonly cited as a level of exposure associated with the induction of asbestosis. The RMA has also noted this threshold level in briefing papers (see [62]) used to determine asbestos factors in SoPs in 2005 and 2013.

59. Boffetta[89] attributes a 25 fibre/mL-years exposure threshold to Doll & Peto.[90] Boffetta[91] states that:

> In case of exposure to a low level of asbestos, radiological, pathological and clinical evidence of lung fibrosis is generally absent. This feature has suggested the existence of a threshold for lung fibrosis: a value commonly proposed for chronic cumulative exposure is 25 fibres/mL-years (fb/ml-yrs)…It has however been suggested that short exposure to very high levels of amphibole asbestos, resulting in a cumulative value below 25 fb/ml-yrs, might induce radiological asbestosis.[92(p.472)]

60. In 1997, an expert group, which represented substantial consensus worldwide[93] developed the 'Helsinki diagnostic criteria'. These criteria have been reviewed in

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86 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013.

87 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 23 and 24 of 2005 for Asbestosis. Brisbane, Australia: 2005.

88 See Appendix A. Table A3 for 2013: Balmes & Speizer 2013 (RMA ID: 67565); Popper 2013 (RMA ID: 67504); Varkey et al [2012] 2013 (RMA ID: 73946); Webb & Higgins 2011 (RMA ID: 68770); Larson et al. 2010 (RMA ID: 67598); Roggli et al 2010 (RMA ID: 67548); and Cited by: Roggli 2010 (RMA ID: 67548) - Browne (2001), Browne (1996), Burdorf & Swuste (1999), Churg (1998), Dement et al. (1983), Fischer et al. (2002), Gibbs et al. (1998), Green et al. (1997), and Sluis-Cremer (1991); Paris et al. 2009 (RMA ID: 67601); Rohs et al. 2008 (RMA ID: 67995); U.S. Department of Health and Human Services, ATSDR, Disease Registry Division of Health Assessment & Consultation 2008 (RMA ID: 67997); Sullivan 2007 (RMA ID: 45641); American Thoracic Society 2004 (RMA ID: 67594); and Wright et al 2002 (RMA ID: 26507).

89 Boffetta P. Health effects of asbestos exposure in humans: a quantitative assessment. Medicina del Lavoro. 1998;89(6):471-80. (RMA ID: 026600).

90 Doll R, Peto J. Asbestos. Effects on health of exposure to asbestos. London: Health & Safety Commission: H.M.S.O; 1985. Cited by: Boffetta P. Health effects of asbestos exposure in humans: a quantitative assessment. Medicina del Lavoro. 1998;89(6):471-80. (RMA ID: 026600).

91 Boffetta P. 1998. (RMA ID: 026600). *ibid.*

92 Boffetta P. 1998. (RMA ID: 026600). *ibid.*

93 Anonymous (International Expert Meeting). Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health. 1997;23(4):311-16. (RMA ID: 026517).

ways that do not affect the matters contended in this review. [94] The 1997 paper states:

> …a cumulative exposure of 25 fibre-years is estimated to increase the risk of lung cancer 2-fold. Clinical cases of asbestosis may occur at comparable cumulative exposures…Cumulative exposures below 25 fibre-years are also associated with an increased risk of lung cancer, but to a less extent. [95(p.314)]

61. The 'Helsinki diagnostic criteria[96] have gained wide acceptance. To assess diagnosis and attribution of asbestos-related diseases in an Australian context, a number of Australian and International experts met in 2000. The Henderson et al[97] report of the expert meeting provided the explanation for the 'Helsinki diagnostic criteria'[98] cumulative exposure of 25 fibre/mL-years associated with asbestosis. The report states:

> …the 25 fibers/mL-year requirement in the Helsinki Criteria is based upon correlative multidisciplinary studies that include fiber burden studies of lung tissue, cohort studies, and case–referent studies, some of which include individualized estimates of exposures and which also include studies of those exposed at points of end-use of asbestos mixtures (representing the most common pattern of exposure)—for which cohort studies are unrealistic—and which may be more representative of the overall risk of asbestos-related lung cancer for an industrialized society than cohort studies restricted to special industries. The requirement of 25 fibers/mL-year of cumulative exposure set forth in the Helsinki Criteria represents a fair and reasonable approach for the attribution of lung cancer to asbestos for amphibole-only exposures (except for Wittenoom), mixed exposures, and asbestos textile exposures; the Helsinki Criteria did not specifically distinguish between fiber types of exposure for the purpose of attribution. Given an adequate period of latency, this cumulative exposure level has been widely used as a cumulative exposure index that makes it more likely than not that asbestos contributed to a particular lung cancer.[99(p.44)]

94 Finnish Institute of Occupational Health. Asbestos, Asbestosis, and Cancer. Helsinki Criteria for Diagnosis and Attribution 2014. Helsinki. (New Information).

95 Anonymous (International Expert Meeting). Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health. 1997;23(4):311-16. (RMA ID: 026517).

96 Anonymous (International Expert Meeting). 1997. (RMA ID: 026517). *ibid.*

97 Henderson et al. The diagnosis and attribution of asbestos-related diseases in an Australian context. Report of the Adelaide Workshop on Asbestos-related Diseases October 6-7, 2000. Int J Occup Med Environ Health. 2004;10(1):40-6. (RMA ID: 035222).

98 Anonymous (International Expert Meeting). 1997. (RMA ID: 026517). *ibid.*

99 Henderson et al. 2004. (RMA ID: 035222). *ibid.*

> A cumulative exposure of 25 fibers/mL-year delineates exposure of a character and magnitude sufficient to induce clinical/radiologic asbestosis in some individuals so exposed, whereas one study carried out on the South Carolina chrysotile textile cohort found that histologic evidence of asbestosis was usually present when cumulative exposures were in excess of 20 fibers/mL-year, but a few cases were encountered with exposures in the range of 10–20 fibers/mL-year.[100(p. 44)]

62. The 2005 RMA briefing paper for Asbestosis[101] cited the cumulative exposure level of 25 fibre/mL-years stated by Henderson et al[102] and acknowledged lower cumulative exposure levels for inducing clinical/radiologic asbestosis in some individuals as reported by Henderson et al[103] and Fischer et al.[104] The RMA reported that:

> …This would support an option to focus dose on the levels used in assessments of occupational exposures, eg an assessment of fibers/mL-year (perhaps 25 fibers/mL-year cumulative exposure to asbestos fibres for BoP and 15 fibers/mL-year for Reasonable Hypothesis).[105(p.11)]

63. The RMA's methodology[106] for the calculation of inhaling respirable asbestos fibres for a cumulative period of hours when making both sets of SoPs (for FILD[107] and Asbestosis[108]) in 2005 and 2013 is set out below:

> The current (1996) RH asbestos factor required a minimum exposure for 200 days (about the number of working days in a calendar year after weekends, holidays and public holidays removed) which if working days is an exposure of 1600 hours. There are some cases which have low but intense exposures and the dose could be say 1000 hours (125 working/8 hour days) for the enclosed space exposures with some

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100 Henderson et al. The diagnosis and attribution of asbestos-related diseases in an Australian context. Report of the Adelaide Workshop on Asbestos-related Diseases October 6-7, 2000. Int J Occup Med Environ Health. 2004;10(1):40-6. (RMA ID: 035222).

101 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 23 and 24 of 2005 for Asbestosis. Brisbane, Australia: 2005.

102 Henderson et al. 2004. (RMA ID: 035222). *ibid*.

103 Henderson et al. 2004. (RMA ID: 035222). *ibid*.

104 Fischer et al. Fibre-years, pulmonary asbestos burden and asbestosis. Int J Hyg Environ Health. 2002; 205(3):245-48. (RMA ID: 026590).

105 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013.

106 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013.

107 RMA. Statements of Principles No. 53 and 54 of 2013 for Fibrosing Interstitial Lung Disease. Brisbane, Australia: 2013. Available from: www.rma.gov.au

108 RMA. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013. Available from: www.rma.gov.au

level of justification from the literature. Though the vast majority of those with asbestosis have had a history of heavy asbestos exposures. [109(p.11)]

For exposures in an open space (drawing on evidence form shipyard and construction workers) exposures may still be significant and contribute to asbestosis. …Dose is again a low end estimate and should probably equate to several years working in that environment. For that I would suggest 3000 hours (approximately two working years). [110(p.11)]

64. The cumulative periods of hours of exposure for both sets of SoPs[111, 112] are shown in Table 1.

TABLE 1. ASBESTOS FACTORS IN THE CURRENT STATEMENTS OF PRINCIPLES FOR FIBROSING INTERSTITIAL LUNG DISEASE AND ASBESTOSIS

Reasonable Hypothesis - for FILD No. 53 of 2013 and for Asbestosis No. 55 of 2013	
(b)&(a)	inhaling respirable asbestos fibres in an **enclosed space**: (i) for a cumulative period of at least **1000 hours** before the clinical onset of asbestosis; and (ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (iii) the first inhalation of asbestos fibres commenced at least **five years** before the clinical onset of asbestosis; or
Balance of Probabilities - for FILD No. 54 of 2013 and for Asbestosis No. 56 of 2013	
(b)&(a)	inhaling respirable asbestos fibres in an **enclosed space**: (i) for a cumulative period of at least **1500 hours** before the clinical onset of asbestosis; and (ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (iii) the first inhalation of asbestos fibres commenced at least **ten years** before the clinical onset of asbestosis; or
Reasonable Hypothesis - for FILD No. 53 of 2013 and for Asbestosis No. 55 of 2013	
(c)&(b)	inhaling respirable asbestos fibres in an **open environment**: (i) for a cumulative period of at least **3000 hours** before the clinical onset of asbestosis; and (ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (iii) the first inhalation of asbestos fibres commenced at least **five years** before the clinical onset of asbestosis;
Balance of Probabilities - for FILD No. 54 of 2013 and for Asbestosis No. 56 of 2013	
(c)&(b)	inhaling respirable asbestos fibres in an **open environment**:

109 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013.

110 RMA Secretariat. Briefing paper - Investigation into Asbestosis. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013.

111 RMA. Statements of Principles No. 53 and 54 of 2013 for Fibrosing Interstitial Lung Disease. Brisbane, Australia: 2013. Available from: www.rma.gov.au

112 RMA. Statements of Principles No. 55 and 56 of 2013 for Asbestosis. Brisbane, Australia: 2013. Available from: www.rma.gov.au

	(i) for a cumulative period of at least **5000 hours** before the clinical onset of asbestosis; and (ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (iii) the first inhalation of asbestos fibres commenced at least **ten years** before the clinical onset of asbestosis;
Reasonable Hypothesis - for Asbestosis No. 55 of 2013 only	
(c)	inhaling respirable asbestos fibres: (i) for a cumulative period of at least **1000 hours** before the clinical worsening of asbestosis; and (ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (iii) within the **two years** before the **clinical worsening** of asbestosis
Balance of Probabilities - for Asbestosis No. 56 of 2013 only	
(c)	inhaling respirable asbestos fibres: (i) for a cumulative period of at least **1500 hours** before the clinical worsening of asbestosis; and (ii) at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled; and (iii) within the **two years** before the **clinical worsening** of asbestosis

65. In relation to the existing asbestos factors above, the Council noted that assessment of the actual dose of asbestos that has been inhaled to produce asbestos-related diseases can only be, at best, a very rough estimate. Days of exposure give a limited evaluation of risk if concentration is not known. The cumulative periods indicated in the SoPs do not take account of other important determinants for the occurrence of disease, such as the type of asbestos fibres, ventilation, the presence of other dusts, smoking, and individual susceptibility.

66. In 2005, the RMA considered using a 25 fibre/mL-year cumulative exposure to asbestos fibres at the BoP level (see [62]), however, it decided to continue to use duration of exposure in the SoPs (1500 hours for an enclosed space and 5000 for an open environment at the BoP level). The medical science basis used to determine the level of cumulative exposure of asbestos in the SoPs was not totally clear to the Council.

The Levels of Asbestos Exposure Associated with Asbestosis in the Sound Medical-Scientific Evidence

67. Forty eight studies were considered relevant SMSE to this review, of which twelve studies reported asbestos cumulative exposure levels significantly associated with producing asbestosis (see **Appendix A. Table A1**). The other thirty six studies (and

an erratum) reported the relationship between the exposure to asbestos and asbestosis but did not report a cumulative fibre/mL-year exposure level or a significant association (see **Appendix A. Table A2**).

68. The twelve studies listed in **Appendix A. Table A1** reported asbestos cumulative exposure levels significantly associated with producing asbestosis and included analysis of asbestosis incidence and mortality. The methods for the determining and classifying asbestosis varied between the studies. In the five morbidity studies[113, 114, 115, 116, 117] the ILO classification of Radiographs of Pneumoconioses[118] was used. In the seven mortality studies [119, 120, 121, 122, 123, 124, 125] asbestosis (or pneumoconiosis or non-malignant respiratory disease) was identified and determined using death certificates/databases, autopsy reports and/or recorded International Coding of Disease (ICD) codes, and in some an analysis of asbestos bodies and lung histology.

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113 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

114 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

115 Paris C, Benichou J, Raffaelli C, Genevois A, Fournier L, Menard C, et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3):206-14. (RMA ID: 067600).

116 Berry G, Gilson JC, Holmes S, Lewinsohn HC, Roach SA. Asbestosis: a study of dose-response relationships in an asbestos textile factory. Br J Ind Med. 1979;36(2):98-112. Cited by: Becklake MR. Asbestos and other fiber-related diseases of the lungs and pleura. Distribution and determinants in exposed populations. Chest. 1991;100(1):248-54. (RMA ID: 000429).

117 Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

118 International Labour Office. Guidelines for the use of the ILO International Classification of Radiographs of Pneumoconioses. Occupational Safety and Health Series, No 22 Revised edition 2000. Cited by: American Thoracic Society Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715 (RMA ID: 067594).

119 Sluis-Cremer GK, Hnizdo E, du Toit RS. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

120 Hein MJ, Stayner LT, Lehman E, Dement JM. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

121 Larson TC, Antao VC, Bove FJ. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

122 Green FH, Harley R, Vallyathan V, Althouse R, Fick G, Dement J, et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

123 McDonald JC, Harris J, Armstrong B. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012;18(2):161-67. (RMA ID: 068460).

124 Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect. 2007;115(4): 579-85. (RMA ID: 045641).

125 Seidman H, Selikoff IJ, Gelb SK. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposures. Am J Ind Med. 1986;10(5-6):479-514. (RMA ID: 020152).

69. The twelve studies listed in **Appendix A. Table A1** reported exposure data from South Africa,[126] USA,[127, 128, 129, 130, 131, 132, 133] France,[134] the UK,[135] and Canada.[136, 137] The data included studies of workers exposed to: amphibole asbestos and serpentine asbestos from an amphibole mine;[138] chrysotile asbestos textile plant;[139, 140] amosite asbestos factory;[141, 142] vermiculite mine/mill;[143, 144, 145, 146] asbestos textile and friction material factory, shipyards, fossil fuel power stations, and industrial insulation;[147] chrysotile and crocidolite asbestos textile factory;[148] and a chrysotile

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126 Sluis-Cremer et al. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

127 Hein et al. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

128 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

129 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

130 McDonald et al. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012;18(2):161-67. (RMA ID: 068460).

131 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

132 Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect. 2007;115(4):579-85. (RMA ID: 045641).

133 Seidman et al. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposures. Am J Ind Med. 1986;10(5-6):479-514. (RMA ID: 020152).

134 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3):206-14. (RMA ID: 067600).

135 Berry et al. Asbestosis: a study of dose-response relationships in an asbestos textile factory. Br J Ind Med. 1979;36(2):98-112. Cited by: Becklake MR. Asbestos and other fiber-related diseases of the lungs and pleura. Distribution and determinants in exposed populations. Chest. 1991;100(1):248-54. (RMA ID: 000429).

136 Green et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

137 Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

138 Sluis-Cremer et al. 1990. Cited by: Sluis-Cremer GK. 1991. (RMA ID: 000440). *ibid*.

139 Hein et al. 2007. (RMA ID: 067597). *ibid*.

140 Green et al. 1997. (RMA ID: 026469). *ibid*.

141 Ehrlich et al. 1992. (RMA ID: 034901). *ibid*.

142 Seidman et al. 1986. (RMA ID: 020152). *ibid*.

143 Larson et al. 2010. (RMA ID: 067598). *ibid*.

144 McDonald et al. 2004. Cited by: Antao et al. 2012. (RMA ID: 068460). *ibid*.

145 Rohs et al. 2008. (RMA ID: 067995). *ibid*.

146 Sullivan PA. 2007. (RMA ID: 045641). *ibid*.

147 Paris et al. 2004. (RMA ID: 067600). *ibid*.

148 Berry et al. 1979. Cited by: Becklake MR.1991. (RMA ID: 000429). *ibid*.

and crocidolite asbestos, silica, and cement factory.[149] There were no studies reporting asbestos exposure data in the military or naval setting.

70. The twelve studies reported the collection of fibre concentrations using various air concentration estimation methods; konimeters; thermal precipitation; Optical Phase Contrast Microscopy; or the midget impinger collection method. From 1968 and later, seven studies[150, 151, 152, 153, 154, 155, 156] reported the use of personal monitoring using membrane filter methods. While two studies[157, 158] reported the use of estimations of fibre concentrations extrapolated from fibre concentrations measured in other factories than those studied.

149 Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

150 Sluis-Cremer et al. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

151 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

152 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

153 McDonald et al. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012;18(2):161-67. (RMA ID: 068460).

154 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

155 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3):206-14. (RMA ID: 067600).

156 Finkelstein MM, Vingilis JJ.1984. (RMA ID: 000454). *ibid*.

157 Seidman et al. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposures. Am J Ind Med. 1986;10(5-6):479-514. (RMA ID: 020152).

158 Ehrlich et al. 1992. (RMA ID: 034901). *ibid*.

71. Eight studies[159, 160, 161, 162, 163, 164, 165, 166] calculated cumulative exposure using varied methods of individual's duration of employment, weighing exposure by occupation using job matrices. Larson et al[167] and Rohs et al[168] both reported weighting the occupational exposure using the 8-hour time weighted average (TWA), multiplied by the number of years, summed over the total years. Seidman et al[169] and Ehrlich et al[170] calculated the average fibre counts for each job title estimated from measurements carried out at two other plants operated by the company and Green et al[171] did not have individual exposure data available for each worker, therefore estimates of exposure by job category at the plant were conducted. Finkelstein and Vingilis[172, 173] calculated their cumulative exposure using a model that extrapolated measurements made by the personal membrane filter (a method that came into use 21 years after the plant was open) and individual work histories.

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159 Sluis-Cremer et al. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

160 Hein et al. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

161 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

162 McDonald et al. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012;18(2):161-67. (RMA ID: 068460).

163 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

164 Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect. 2007;115(4):579-85. (RMA ID: 045641).

165 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3): 206-14. (RMA ID: 067600).

166 Berry et al. Asbestosis: a study of dose-response relationships in an asbestos textile factory. Br J Ind Med. 1979;36(2):98-112. Cited by: Becklake MR. Asbestos and other fiber-related diseases of the lungs and pleura. Distribution and determinants in exposed populations. Chest. 1991;100(1):248-54. (RMA ID: 000429).

167 Larson et al. 2010. (RMA ID: 067598). *ibid.*

168 Rohs et al. 2008. (RMA ID: 067995). *ibid.*

169 Seidman et al. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposures. Am J Ind Med. 1986;10(5-6):479-514. (RMA ID: 020152).

170 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

171 Green et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

172 Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

173 Finkelstein MM. Asbestosis in long-term employees of an Ontario asbestos-cement factory. Am Rev Respir Dis. 1982;125(5):496-501. Cited by: Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

72. Duration of exposure was reported in the twelve studies with large variability, ranging from 1 day to 30 years, with the studies reporting duration exposure levels as either mean,[174, 175, 176, 177, 178] median,[179, 180, 181, 182] range,[183, 184] not stated,[185] or exact duration figures were not reported.[186] The duration of exposure was most often calculated from the duration of employment: M = 4.0 (range = 1 day-43.1) years;[187] M = 9 years;[188] M = 20.1 years;[189] M = 24.9 (SD = 9.1) years;[190] M = 30.4 (SD = 14.0) years;[191] median = 0.5; 6 months (range = 0.1-13) years;[192] median = 0.8 (25th-75th

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174 Sluis-Cremer et al. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

175 McDonald et al. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012;18(2):161-67. (RMA ID: 068460).

176 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3):206-14. (RMA ID: 067600).

177 Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect. 2007;115(4):579-85. (RMA ID: 045641).

178 Berry et al. Asbestosis: a study of dose-response relationships in an asbestos textile factory. Br J Ind Med. 1979;36(2):98-112. Cited by: Becklake MR. Asbestos and other fiber-related diseases of the lungs and pleura. Distribution and determinants in exposed populations. Chest. 1991;100(1):248-54. (RMA ID: 000429).

179 Hein et al. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

180 Hein et al. 2007. (RMA ID: 067597). *ibid*.

181 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

182 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

183 Green et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

184 Seidman et al. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposures. Am J Ind Med. 1986;10(5-6):479-514. (RMA ID: 020152).

185 Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

186 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

187 Sullivan PA. 2007. (RMA ID: 045641). *ibid*.

188 McDonald et al. 2004. Cited by: Antao et al. 2012. (RMA ID: 068460). *ibid*.

189 Berry et al. 1979. Cited by: Becklake MR.1991. (RMA ID: 000429). *ibid*.

190 Paris et al. 2004. (RMA ID: 067600). *ibid*.

191 Sluis-Cremer et al. 1990. Cited by: Sluis-Cremer GK.1991. (RMA ID: 000440). *ibid*.

192 Ehrlich et al. 1992. (RMA ID: 034901). *ibid*.

percentile 0.1-4.1 years);[193] median = 1.1 (range = 0.1-46.8) years;[194] range = <1 month to 2-14 years;[195] and 0.1- >27.3 years;[196] and 1 year.[197]

73. Of the five morbidity studies, three studies[198, 199, 200] demonstrated cumulative asbestos exposure levels of 25 fibre/mL-years or lower (at 5; 11.86; and 25 fibre/mL-years). Both Ehrlich et al[201] and Rohs et al[202] showed a significant association between cumulative asbestos exposure and asbestosis (ILO profusion ≥1/0) at <5 fibre/mL-years (*n* = 10) and 11.86 fibre/mL-years (*n* = 8) respectively. Paris et al[203] showed a significant association between cumulative asbestos exposure and asbestosis (ILO profusion ≥1/1, HRCT grade 2 or 3) at 25 fibre/mL-years (*n* = 51). There were two studies[204, 205] that demonstrated cumulative asbestos levels greater than 25 fibre/mL-years, at 84 and 150 fibre/mL-years respectively.

193 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

194 Hein et al. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

195 Seidman et al. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposures. Am J Ind Med. 1986;10(5-6):479-514. (RMA ID: 020152).

196 Green et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

197 Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

198 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

199 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

200 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3):206-14. (RMA ID: 067600).

201 Ehrlich et al. 1992. (RMA ID: 034901). *ibid*.

202 Rohs et al. 2008. (RMA ID: 067995). *ibid*.

203 Paris et al. 2004. (RMA ID: 067600). *ibid.*

204 Berry et al. Asbestosis: a study of dose-response relationships in an asbestos textile factory. Br J Ind Med. 1979;36(2):98-112. Cited by: Becklake MR. Asbestos and other fiber-related diseases of the lungs and pleura. Distribution and determinants in exposed populations. Chest. 1991;100(1):248-54. (RMA ID: 000429).

205 Finkelstein MM, Vingilis JJ. 1984. (RMA ID: 000454). *ibid*.

74. Of the seven mortality studies, five studies[206, 207, 208, 209, 210] demonstrated cumulative asbestos exposure levels below 25 fibre/mL-years (at 2; 3; 8.6; 10; and 11.7 fibre/mL-years). Sluis-Cremer et al[211] found an increased risk of death from asbestosis at cumulative exposures of >2-5 fibre/mL-years (*n* = 4), Hein et al[212] at 3-<16 fibre/mL-years (*n* = 42), Larson et al[213] at 8.6 to <44.0 fibre/mL-years (*n* = 25), Green et al[214] at 10-20 fibre/mL-years (*n* = 3), and McDonald et al[215] found an increased risk of death from non-malignant respiratory disease at 11.7 fibre/mL-years (*n* = 51). There were two studies[216, 217] that reported levels above 25 fibre/mL-years at 50 and 100 fibre/mL-years respectively.

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206 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

207 Hein et al. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

208 Green et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

209 McDonald et al. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012; 18(2):161-67. (RMA ID: 068460).

210 Sluis- Cremer et al. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

211 Sluis-Cremer et al.1990. Cited by: Sluis-Cremer GK. 1991. (RMA ID: 000440). *ibid*.

212 Hein et al. 2007. (RMA ID: 067597). *ibid*.

213 Larson et al. 2010. (RMA ID: 067598). *ibid*.

214 Green et al. 1997. (RMA ID: 026469). *ibid*.

215 McDonald et al. 2004. Cited by: Antao et al. 2012. (RMA ID: 068460). *ibid*.

216 Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect. 2007;115(4): 579-85. (RMA ID: 045641).

217 Seidman et al. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposures. Am J Ind Med. 1986;10(5-6):479-514. (RMA ID: 020152).

75. There were eight [218, 219, 220, 221, 222, 223, 224, 225] of the twelve studies that provided results of an association between a cumulative exposure between 2-25 fibre/mL-years and a diagnosis of asbestosis. The eight studies had cumulative exposure levels equivalent to or lower than the Helsinki diagnostic criterion[226] of 25 fibre/mL-years associated with producing asbestosis.

76. It has been reported that asbestosis is more prevalent and more advanced for a given duration of exposure in cigarette smokers,[227] however, only half of the twelve

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218 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

219 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

220 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3):206-14. (RMA ID: 067600).

221 Sluis-Cremer et al. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

222 Green et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

223 Hein et al. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

224 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

225 McDonald et al. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012; 18(2):161-67. (RMA ID: 068460).

226 Anonymous (International Expert Meeting). Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. Scand J Work Environ Health. 1997;23(4):311-16. (RMA ID: 026517).

227 Barnhart S, Thornquist M, Omenn GS, Goodman G, Feigl P, Rosen-stock LT. The degree of roentgenographic parenchymal opacities attributable to smoking among asbestos-exposed subjects. Am Rev Respir Dis. 1990;141:1102-06. Cited by: American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

studies documented smoking histories[228, 229, 230, 231, 232, 233, 234] while the other studies did not report or adjust for individuals smoking history.[235, 236, 237, 238, 239, 240, 241]

77. Of the twelve studies, there were no data from Australia reporting cumulative exposure levels significantly associated with asbestosis. However, of the 36 studies there are data from Australia (see **Appendix A. Table A2**). The Wittenoom[242] studies by Cookson et al[243, 244] and Reid et al[245] (see [80]) were included in the review. These

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228 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

229 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

230 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3): 206-14. (RMA ID: 067600).

231 Berry et al. Asbestosis: a study of dose-response relationships in an asbestos textile factory. Br J Ind Med. 1979;36(2):98-112. Cited by: Becklake MR. Asbestos and other fiber-related diseases of the lungs and pleura. Distribution and determinants in exposed populations. Chest. 1991;100(1):248-54. (RMA ID: 000429).

232 Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

233 Seidman et al. Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short-term work exposures. Am J Ind Med. 1986;10(5-6):479-514. (RMA ID: 020152).

234 Green et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

235 Sluis-Cremer et al. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

236 Hein et al. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

237 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

238 Green et al. 1997. (RMA ID: 026469). *ibid*.

239 McDonald et al. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012; 18(2):161-67. (RMA ID: 068460).

240 Seidman et al. 1986. (RMA ID: 020152). *ibid*.

241 Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a cohort mortality study. Environ Health Perspect. 2007;115(4):579-85. (RMA ID: 045641).

242 A single company operated the Wittenoom crocidolite mine and mill in the Pilbara region of Western Australia between 1943 and 1966. The mine and mill at Wittenoom exposed workers to high concentrations of asbestos fibres in inhaled air and the employment records of the company identifying nearly 7000 workers have been retained and form the basis of a continuing study of the workforce. Cited by: Cookson et al. Prevalence of radiographic asbestosis in crocidolite miners and millers at Wittenoom, Western Australia. Br J Ind Med. 1986;43(7):450-57. (RMA ID: 000444).

243 Cookson WO, De Klerk NH, Musk AW, Armstrong BK, Glancy JJ, Hobbs MS. Prevalence of radiographic asbestosis in crocidolite miners and millers at Wittenoom, Western Australia. Br J Ind Med. 1986;43(7):450-57. (RMA ID: 000444).

244 Cookson et al. The Natural history of asbestosis in former crocidolite workers in Wittenoom Gorge. Am Rev Respir Dis. 1986; 133(6):994-98. (RMA ID: 000445).

245 Reid A, de Klerk N, Ambrosini GL, Olsen N, Pang SC, Berry G, et al. The effect of asbestosis on lung cancer risk beyond the dose related effect of asbestos alone. J Occup Environ Med. 2005;62(12):885-89. (RMA ID: 037507).

studies did include cumulative exposure data but the conditions of exposure were very different from those of the Australian military and showed also there was a significant incidence of asbestosis in non-cancer cases and at lower exposures than 25 fibre/mL-years. To the Council's knowledge, there have been no reports regarding exposure levels in the Australian military. The Council acknowledges however that there are some reports from the military setting in the international literature. The Council noted that using data from the mining and manufacturing settings as exposure surrogates may not be appropriate for informing on military exposure levels.

78. The Council considered that the more reliable papers reporting asbestos exposure and threshold for development of asbestosis were the morbidity studies, while accepting that the radiological criteria for asbestosis are imperfect. The morbidity studies used a radiological definition for defining incidence of asbestosis rather than mortality studies that used autopsy reports and death certificates and/or databases. Morbidity studies reporting incidence analysis allow a more complete analysis of disease incidence in a non-fatal disease, which is more relevant to the Applicants contention.

79. The Council considered that studies using the membrane filter method for fibre collection and analysis are the most scientifically robust studies.[246, 247, 248, 249, 250, 251, 252] Collection taking place in the job environment as close as possible to the area of respiration, at multiple collection points, and with data available across a number of years provide the most reliable exposure levels. All twelve studies reported various

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246 Sluis-Cremer et al. Evidence for an amphibole asbestos threshold exposure for asbestosis assessed by autopsy in South African asbestos miners. Ann Occup Hyg. 1990;34(5):443-51. Cited by: Sluis-Cremer GK. Asbestos disease at low exposures after long residence times. Ann N Y Acad Sci. 1991;643:182-93. (RMA ID: 000440).

247 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

248 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

249 McDonald et al. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. J Occup Environ Med. 2004;61(4):363-66. Cited by: Antao et al. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 2012; 18(2):161-67. (RMA ID: 068460).

250 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

251 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3): 206-14. (RMA ID: 067600).

252 Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

methods to calculate fibre concentration estimates, and many of the studies used exposure estimates derived from average airborne fibre concentrations that did not represent an assessment of individualised exposures. There were inconsistencies in measurement strategies and sampling techniques, which was further complicated by the use of retrospective exposure data. Retrospective data was often incomplete rendering the accuracy of the exposure, exposed mining and manufacturing workers prone to underestimation. However, the Council acknowledges the difficulties in collection of such data and the limitations inherent in assessing past exposures.

80. The Council considered that the most informative papers into lower dose exposure of the twelve studies that reported cumulative exposure levels were those of Rohs et al[253] and Paris et al.[254] The Council also acknowledged the Wittenoom studies conducted by Cookson et al[255, 256] and Reid et al[257] reporting asbestos exposure in the Australia mining industry.

a. Rohs et al conducted a 25-year follow-up study of 280 Libby Vermiculite mine workers of the original 513 cohort, analysing incidence of asbestosis. There were eight cases of interstitial changes (ILO profusion of ≥1/0). From 1972, membrane filters method was used and prior to that air sampling was conducted by industrial hygienists who followed a worker with a sampling device. Each department was assigned two values of exposure (fibre exposures ≤1973 and >1973). Individuals were assigned a cumulative fibre exposure value, which was the summation of estimated fibre exposure by department, based on the years employed between 1963 and 1980. Cumulative fibre exposure was calculated by multiplying the 8-hour TWA exposure by the number of years. The authors demonstrated interstitial changes were significantly related to a mean cumulative fibre exposure of 11.37 fibre/mL-years. A limitation of the study is the potential misclassification of exposure due to limited industrial hygiene data at the facility and by not accounting for workers overtime. A second limitation is the potential for participation bias, as 30.9 % of cases

253 Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

254 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3): 206-14. (RMA ID: 067600).

255 Cookson et al. Prevalence of radiographic asbestosis in crocidolite miners and millers at Wittenoom, Western Australia. Br J Ind Med. 1986;43(7):450-57. (RMA ID: 000444).

256 Cookson et al. The Natural history of asbestosis in former crocidolite workers in Wittenoom Gorge. Am Rev Respir Dis. 1986; 133(6):994-98. (RMA ID: 000445).

257 Reid et al. The effect of asbestosis on lung cancer risk beyond the dose related effect of asbestos alone. J Occup Environ Med. 2005;62(12):885-89. (RMA ID: 037507).

were lost to follow-up, therefore several of those with advanced disease or illness may have been missed.

b. Paris et al[258] screened 706 retired workers in the textile and friction material factory, shipyards, fossil fuel power stations, and industrial insulation industries for the incidence of early-stage asbestosis. There were 51 cases of pulmonary fibrosis diagnosed by HRCT (grade 2 or 3), of which only 38 could be diagnosed via chest X-ray (ILO profusion ≥1/1). A specific job-exposure matrix was used, based upon air-borne samples collected annually between 1959 and 1999 in the various workshops of the plant. From 1973 the membrane filter method was used. A cumulative-exposure index was calculated by summing the values for all job positions held, with reference to the occupational calendar established in the interview, the products of the job exposure level (in fibre/mL) by job duration (in years).The mean duration of exposure was 24.9 years. A significant association between HRCT fibrosis and cumulative asbestos exposure was apparent, with a clear dose-response relationship seen at 25-99.9 fibre/mL-years (OR = 3.4; 95% CI: 0.8, 15.2; $p>0.05$) compared with an exposure of 25 fibre/mL-years or less. A limitation of the study was the potential for participation bias, as sufferers of established asbestosis were excluded from entry. This study demonstrated asbestosis was significantly associated with cumulative exposure >25 fibre/mL-years, and that the majority of cases occurred at much higher levels than 25 fibre/mL-years.

c. Cookson et al[259] examined the prevalence of unrecognised pneumoconiosis in former crocidolite asbestos workers of Wittenoom who had never entered a compensation claim. Chest X-rays were available for 859 subjects, of which 541 had radiographs taken after the date of first employment, 52% of the original sample had post-employment radiographs that could be used to draw conclusions about the current prevalence of radiographic abnormality. The other 318 subjects with radiographs formed a comparison group with no known exposure to crocidolite at Wittenoom at the time of their radiographs. Prevalence of parenchymal abnormality (ILO profusion ≥1/0) of nearly 20% was calculated after adjusting for age, time since first exposure, and cumulative exposure level. A random selection of 74 men had a new radiographic examination, with 10 cases of parenchymal abnormality, and the

258 Paris et al. Factors associated with early-stage pulmonary fibrosis as determined by high-resolution computed tomography among persons occupationally exposed to asbestos. Scand J Work Environ Health. 2004;30(3): 206-14. (RMA ID: 067600).

259 Cookson et al. Prevalence of radiographic asbestosis in crocidolite miners and millers at Wittenoom, Western Australia. Br J Ind Med. 1986;43(7):450-57. (RMA ID: 000444).

predicted prevalence was confirmed. The total average cumulative exposure for 541 men was 9.6 fibre/mL-years; 318 men was 5.3 fibre/mL-years; and for 74 men followed-up was 8.1 fibre/mL-years. The average duration of exposure was less than 4 months. This study showed an increased relative risk for those workers exposed to <20 fibre/mL-years (RR = 1.227; 95%CI: 1.079, 1.396); 20-54.5 fibre/mL-years (RR = 1.289; 95%CI: 1.115, 1.49); and ≥55 fibre/mL-years (RR = 1.387; 95%CI: 1.20, 1.603). The results showed a prevalence of uncompensated radiographic abnormality consistent with pneumoconiosis in at least 16% of former Wittenoom workers and the data are consistent with there being no threshold dose of crocidolite exposure for the development of radiographic abnormality in this group. However, the authors were cautious in interpreting the results, stating that the study related only to the finding of radiographic changes of small irregular or small rounded opacities, and that the radiographic changes were not diagnostic of radiographic asbestosis and not necessarily indicative of clinical asbestosis in the participants.

d. Cookson et al [260] examined 354 former crocidolite asbestos workers of Wittenoom who had applied for compensation for asbestosis. Chest X-rays were available for 280 men. Subjects were excluded if there was no definite radiographic evidence of pneumoconiosis, if they had a known other disease accounting for radiographic abnormality, or more than 5 years exposure to silica elsewhere in the mining industry. There were two radiograph readers, Reader 1 identified 136 subjects and Reader 2 identified 139 subjects who met the inclusion criteria. Subjects had an average of 10 or more radiographs each to document the onset and progression of asbestosis. For Reader 1, for the 136 men onset of definite radiographic asbestosis was observed in radiographs taken between 2 and 34 years after commencing employment (median = 14 years). No subjects developed asbestosis during the period of employment. The median cumulative exposure was 91 fibre/mL-years and median duration of exposure was 37 months. For Reader 2, for the 139 men there was a range between 1 to 33 years (median = 13 years) from the time of commencing employment to observed development of radiographic asbestosis. There were seven men diagnosed with asbestosis while they were still employed. The median cumulative exposure was 77 fibre/mL-years and median duration of exposure to onset of asbestosis was 33 months. Only the results of the analysis from Reader 1 were presented in the paper. Onset of asbestosis was most frequent between 10 and 20 year from first exposure, with no increased relative risk of

[260] Cookson et al. The Natural history of asbestosis in former crocidolite workers in Wittenoom Gorge. Am Rev Respir Dis. 1986; 133(6):994-98. (RMA ID: 000445).

progression for cumulative exposure of 0-54 fibre/mL-years. An increased relative rate of progression was seen for cumulative exposure of 55-148 fibre/mL-years (ILO category 1 to 2: RR = 1.6; 95%CI: 1.1, 2.3; ILO category 2 to 3: RR = 2.7, 95%CI: 0.9, 8.2) and cumulative exposure of >148 fibre/mL-years (ILO category 1 to 2: RR = 2.5; 95%CI: 1.2, 5.4; ILO category 2 to 3: RR = 7.1, 95%CI: 0.8, 66.5). Both onset of asbestosis and progression from ILO category 1 to 2 continued to occur throughout the follow-up period in workers exposed to crocidolite. There was no evidence of an effect of cumulative exposure to crocidolite on the time to onset of pneumoconiosis. However, there was some evidence of an effect of cumulative exposure for progression of pneumoconiosis. This study showed an increased relative rate of progression for cumulative exposure of ≥55 fibre/mL-years (ILO category 1 to 2 and 2 to 3). The authors concluded the rate of radiographic progression of established asbestosis increases with the accumulated exposure to crocidolite, and decreases with time from initial crocidolite exposure to the onset of definite radiographic abnormality.

e. Reid et al[261] studied 1988 former workers and residents of Wittenoom with known crocidolite asbestos exposure participating in a cancer prevention program, to determine if the presence of asbestosis is a prerequisite for lung cancer. Over a 12 year period there were 58 cases of lung cancer, the cases had a greater intensity of asbestos exposure and greater cumulative exposure than the remaining cohort. For the 58 cases, 21 (36%) had radiographic evidence of asbestosis on chest X-rays (ILO profusion ≥1/0) compared to 220 (11%) non-cases (*n* = 1930), and 12% of total study participants (*n* = 1988). For the 58 cases the average cumulative exposure was 11 fibre/mL-years (*SD* = 2.6-46.5) with a duration of exposure of 330 days (interquartile range (IQR) = 118-1048). For the 1930 non-cases the average cumulative exposure was 5 fibre/mL-years (*SD* = 1.84-13.5) with a duration of exposure of 285 days (IQR = 106-868). Both radiographic asbestosis (OR = 1.94; 95%CI: 1.09, 3.46) and asbestos exposure (OR = 1.21 per fibre/mL-years; 95%CI: 1.02, 1.42) were significantly associated with an increased risk of lung cancer. There was an increased risk of lung cancer with increasing exposure in those without asbestosis. The authors concluded that the relative risk of lung cancer was higher in former Wittenoom workers and ex-residents with asbestosis than those without asbestosis, even after adjusting for the amount of asbestos exposure. This study showed the presence of asbestosis is not a necessary a precursor for lung cancer,

261 Reid et al. The effect of asbestosis on lung cancer risk beyond the dose related effect of asbestos alone. J Occup Environ Med. 2005;62(12):885-89. (RMA ID: 037507).

and the presence of asbestosis is also associated with an increased risk of lung cancer which may be due to some action of the fibrosis itself, but in this study this excess risk may also be due to an underestimate of asbestos exposure.

New Information on the Levels of Asbestos Exposure Associated with Asbestosis in the Sound Medical-Scientific Evidence

81. The Council noted there is new information available on the levels of asbestos exposure associated with asbestosis.

82. The Council considered that some of the most important relevant new studies are by Harries[262, 263] and van Oyen et al.[264] An example of actual military data are studies by Harries[265, 266] which were conducted in the British Naval Dockyards and provide information regarding asbestos fibre concentrations and exposure of naval personnel in the UK. There are others from, for example, the USA.[267] Furthermore, the study by van Oyen et al[268] is an example of a job-exposure matrix using TWA for occupational asbestos exposure in Australia (see New Information section from [93] and **Appendix A. Table A4**).

Summary of Council's View

83. The Council's attempt to compare a cumulative period of hours (from the SoPs) with fibre/mL-years (from the literature) did not reveal any relevant SMSE which could allow definite conclusions about the asbestos duration of exposure factors in Australian military personnel.

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262 Harries PG. A comparison of mass and fibre concentrations of asbestos dust in shipyards insulation processes. Ann Occup Hyg. 1971;14(3):235-40. (New Information).

263 Harries PG. Asbestos dust concentrations in ship repairing: a practical approach to improving asbestos hygiene in naval dockyards. Ann Occup Hyg. 1971:14(3):241-54. (New Information).

264 Van Oyen et al. Development of a job matrix (AsbJEM) to estimate occupational exposure to asbestos in Australia. Ann Ocup Hyg. 2015;59(6):737-48 and supplement tables S1 & S2 pp1-9. (New Information).

265 Harries PG. A comparison of mass and fibre concentrations of asbestos dust in shipyards insulation processes. Ann Occup Hyg. 1971;14(3):235-40. (New Information).

266 Harries PG. Asbestos dust concentrations in ship repairing: a practical approach to improving asbestos hygiene in naval dockyards. Ann Occup Hyg. 1971:14(3):241-54. (New Information).

267 Murbach DM, Madi AK, Unice KM, Knutsen JS, Chapman PS, Brown JL, et al. Airborne concentrations of asbestos onboard maritime shipping vessels (1978-1992). Ann Occup Hyg. 2008;52(4):267-79. (New Information).

268 Van Oyen et al. 2015. (New Information). *ibid.*

84. The relevant SMSE in the information available to the Council was sufficient to show a dose of 25 fibre/mL-years does cause FILD and Asbestosis [67]-[80]. However, there were also studies that demonstrated the development of asbestosis occurring below this level at between 2-15 fibre/mL-years. [269, 270, 271, 272] Additionally, the Council notes that fibre type should be considered along with estimates of the concentration of fibres, as there are data suggesting differences in rates of development of asbestosis depending on fibre type.

85. The Council believes that the SoPs should ideally use historical measurements from military work environments to determine the relevant dose as well as a cumulative period of time. If these data are not available, then those doses from comparable environments (for example the building of Royal Australian Navy ships) in the published literature should be used. If no data currently exist, the Council recommends that such asbestos exposures should be scientifically assessed as far as is practicable using optimal methods, before all naval vessels containing asbestos are retired. It concedes, that the measure of a cumulative period of hours may be the only practical measure currently available to the RMA to characterise dose, but considers this to be incomplete.

86. The assessment of exposures to asbestos experienced in the past is likely to continue to be estimated using the proxy measure of duration of employment in the SoPs until better methods become available.

87. The Council also considered those studies,[273, 274] that used accurate work histories to calculate a TWA of exposure for particular work roles, and concluded that these methods are likely to provide the most accurate representation of exposure data. Current methods using, for example, a job exposure matrix and an 8-hour TWA

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[269] Hein et al. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. J Occup Environ Med. 2007;64:616-25. (RMA ID: 067597).

[270] Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

[271] Green et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. J Occup Environ Med. 1997;54(8):549-59. (RMA ID: 026469).

[272] Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

[273] Larson et al. 2010. (RMA ID: 067598). *ibid.*

[274] Rohs et al. Low-level fiber-induced radiographic changes caused by Libby Vermiculite. A 25-year follow-up study. Am J Respir Crit Care Med. 2008;177(6):630-37. (RMA ID: 067995).

(rather than extrapolated exposure measurements or estimates of exposure by job category and time), are likely to be more accurate.

88. The Council noted a cumulative exposure involves the assumption that duration and intensity are equally important in determining the effective dose. If exposure estimates are inaccurate or inconsistently measured, a reported statistically significant association between cumulative exposure and asbestosis can misrepresent the actual exposure – response relationship.

89. The Council noted that the 12 studies reporting duration of exposure ranging from 1 day to 30 years (see [72]) and that eight of the twelve studies (see **Appendix A. Table A1**) demonstrated a significant association between lower cumulative asbestos exposure than 25 fibre/mL-years (see [75]) and asbestosis.

90. It is difficult to assess the applicability of the reported studies to the Australian military environment. The wide range (2-150 fibre/ml-years) of relevant asbestos exposure data in the 12 studies evaluated by Council (at [67] to [80]), and the imprecision of the measurement of dose in those studies and in practice generally, does not allow the Council to specify with confidence a duration of exposure that causes asbestosis.

91. Therefore, the Council was unable to recommend changes to the duration of employment as used for asbestos exposure in the SoPs, but suggests that the RMA considers the SMSE pertinent to current methods of optimally assessing occupational exposure to asbestos.

92. The Council discussed the possibility of obtaining concentrations of asbestos fibres in the air specific to Australian military work environments. Obtaining actual asbestos fibre measurements from past and current naval vessels and other military work environments may allow these measurements to be used in the future to build a job matrix specific to Australian conditions.

COUNCIL'S CONCLUSIONS ON THE SOUND MEDICAL-SCIENTIFIC EVIDENCE RELEVANT TO THE APPLICANT'S CONTENTIONS

93. While the Council evaluated the SMSE relevant to the Applicant's contentions. The Council placed particular weight on the articles discussed above (see **Appendix A. Table A1 and Table A2**) that provided specific results data for:

a. Pleural Plaques and Asbestos bodies;

b. The duration of asbestos exposure; and

c. The completeness of the information the RMA sent to the Council.

Pleural Plaques and Asbestos Bodies

94. The Council's summary of its consideration of relevant SMSE concerning pleural plaques and asbestos bodies is at [54]. Asbestos bodies or pleural plaques are markers of asbestos exposure, but do not equate to a diagnosis of asbestosis.

95. The Council considered all of the SMSE and its view was that the relevant SMSE fell short of indicating the existence of a reasonable hypothesis regarding the contended exposure to asbestos and the presence of pleural plaques and asbestos bodies alone as definite evidence of FILD or Asbestosis. That is, the SMSE was insufficient to justify an amendment to either of the reasonable hypothesis SoPs.

96. Accordingly, the Council's view was that the SMSE was also insufficient to justify an amendment to either of the SoPs on the balance of probabilities.

The Duration of Asbestos Exposure in the Statements of Principles

97. The Council's summary of its consideration of the relevant SMSE concerning the duration of asbestos exposure is at [83] to [96].

98. A cumulative period of hours is not used in current SMSE as a measure of dose. In the absence of a job exposure matrix for military personnel it is difficult to accurately estimate cumulative fibre exposure. The Council recommends that a job exposure matrix for military personnel would be useful, based upon measured time weighted averages of exposure in actual settings, and accurate military records of jobs and tasks and their durations.

99. The Council, having considered all of the relevant SMSE formed the view that changing the duration of exposure to asbestos as expressed in the SoPs, to an alternative scientific measure or in any other way, was not justifiable for either the RH or BoP tests. That is, the SMSE was insufficient to justify an amendment to either of

the reasonable hypothesis or balance of probabilities SoPs. Hence the Council made the declaration at [1] to [4].

Council's View on Whether the Information Gathered by the Repatriation Medical Authority was Complete

100. The Council's summary of its consideration of the relevant SMSE concerning new information in relation to:

i. pleural plaques is at [54] to [56];

ii. diffuse pleural thickening is at [54] to [56]; and

iii. duration of exposure is at [65] to [66].

and can be found in the following 'New Information'.

New Information

101. The status of the information discussed below is 'new information', that is, information that was not available to (not before) the RMA. Accordingly, it the Council did not take it into account for the purposes of the review.

102. Rather, the Council has considered the new information to determine whether, in the Council's view, it warrants the Council making any directions or recommendations to the RMA.

103. In the Council's view any such direction or recommendation should only be made by the Council if it formed the view that the new information:

- comprised sound medical-scientific evidence as defined in section 5AB(2) of the VEA being information which:
    - epidemiologists would consider appropriate to take into account; and
    - in the Council's view, 'touches on' (is relevant to) any contended factor and has been evaluated by the Council according to epidemiological criteria, including the Bradford Hill criteria; and

- could potentially satisfy the reasonable hypothesis and/or balance of probabilities tests.

New Information Submitted by the Applicant

104. The Council noted the Applicant's references to and submissions concerning information not available to (not before) the RMA (see **Appendix A: Table A4**). The Council considered this information carefully. While some of the information may be considered relevant SMSE, this information did not allow a complete evaluation of the contended matters nor allow a practical method for amendment for the current SoPs on:

   a. Pleural Plaques and Asbestos bodies being sufficient evidence of FILD or Asbestosis; and

   b. Asbestos exposure duration in the current factors.

   The Council noted and acknowledges that a number of the new information articles submitted by the Applicant concerned the process of inflammation and or wound healing as part of pulmonary healing and the development of fibrosis. The Council considered these papers, but they did not change its view about the role of pleural plaques and asbestos bodies, or asbestos exposure duration.

105. While the Council did not undertake a comprehensive analysis of any of the new information it noted the following study which was relied on by the Applicant.

   a. Gaensler et al [275] studied lung tissue samples of 176 people exposed to asbestos. Nine had the clinical features of asbestosis but histologic sections failed to demonstrate the presence of asbestos bodies. These nine were compared by analytic electron microscopy with nine persons with idiopathic pulmonary fibrosis (Group 2), and with nine persons with all the criteria of asbestosis (Group 3). The authors concluded that it is sometimes difficult to define American Thoracic Society criterion of a reliable history of asbestos exposure. The authors also stated that:

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275 Gaensler EA, Jederlinic PJ, Churg A. Idiopathic pulmonary fibrosis in asbestos-exposed workers. Am Rev Respir Dis 1991;144(3 Pt 1):689-96. Abstract. (New Information).

> … asbestos bodies are seen in tissue sections only when exposure has been reasonably high, and given the proper clinical setting, the presence of diffuse fibrosis and asbestos bodies in tissue sections are sensitive and specific criteria for a diagnosis of asbestosis.(at abstract)

The Council acknowledges the low sensitivity of asbestos bodies in histological sections of lung when examined by light microscopy. The finding of asbestos bodies alone is not sufficient for a histologic diagnosis of asbestosis and the Asbestos Committee recommends that such a diagnosis should only be made when there is an average rate of $2/cm^2$ of lung in conjunction with an acceptable pattern of alveolar septal fibrosis.[276] Electron microscopic studies are reported to be more sensitive in this regard and also allow differentiation of fibre type. However, lung tissue samples are rarely available and the Council would not recommend that these be obtained solely for the purposes of compensation. High resolution CT scanning is a practical surrogate, and if performed using the recommended technique,[277] is an adequate the recommended method for the diagnosis of asbestosis when taken in conjunction with the occupational exposure history.

New Information Identified by the Council

106. The Council identified information not available to (not before) the RMA, which it considered was likely to be relevant SMSE (see **Appendix A: Table A4**).

107. While the Council did not undertake a comprehensive analysis of any of the new information it noted the following:

Diffuse Pleural Thickening

a. Selikoff et al[278] examined insulation workers, where much of their work was in open air aboard ship, in tight quarters with poor ventilation. The study found 48.5% had asbestosis seen on X-ray, and a correlation between years

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276 Roggli et al. Pathology of asbestosis - An update of the diagnostic criteria: Report of the asbestosis committee of the College of American Pathologists and Pulmonary Pathology Society. Arch Pathol & Lab Med. 2010;134(3):462-80. (RMA ID: 067548).

277 Tossavainen A. Consensus report. International expert meeting on new advances in the radiology and screening of asbestos-related diseases. Scand J Work Environ Health. 2000;26(5):449-54. (New Information).

278 Selikoff IJ, Churg J, Hammond EC. The occurrence of asbestosis among insulation workers in the United State. Ann N Y Acad Sci.1965;132(1):139-55. (New Information).

of onset of exposure and increase in the development of asbestosis. Examination of lung tissue from 45 asbestos insulation workers, there were no case that the authors fail to find typical pulmonary asbestosis, with fibrosis and asbestos bodies present.

b. Miles et al[279] conducted a comprehensive review of the clinical consequences of asbestos-related diffuse pleural thickening. This review has been used in the body of this document (see [39]).

Pleural Plaques

c. IACC Position Paper 23[280] reported the evidence of pleural plaques, including prevalence, occupational causation, likelihood of disability, and if it should be a compensated disease. Based on the evidence, the IACC recommended against including pleural plaques as a compensated disease under the Industrial Injuries Disablement Benefit Scheme.

Asbestosis

d. Park et al[281] studied lung function measurements in men with asbestosis; diffuse pleural thickening; asbestosis and diffuse pleural thickening; pleural plaques only; and healthy individuals with a history of asbestos exposure. The study showed the presence of asbestos-related diseases lowered lung function measurements compared to healthy individuals exposed to asbestos, with significant differences between asbestos-related diseases.

e. Harries[282] conducted a comparison of mass and fibre concentrations of asbestos dust produced during shipbuilding and refitting in the British Naval Dockyards. This study shows that there is some correlation between mass and fibre concentrations in stripping processes with higher dust levels, but that there is no correlation between the dust indices at lower concentrations. Comparison of the results of measuring total dust, 'respirable' dust, and fibre concentration show that gravimetric measurements although simpler and quicker are not suitable for environmental monitoring in Naval Dockyards and

279 Miles et al. Clinical consequences of asbestos-related diffuse pleural thickening: A review. Journal of Occupational Medicine and Toxicology. 2008;3:20. (New Information).

280 IIAC: The Industrial Injuries Advisory Council. Pleural plaques: IIAC Position Paper 23. 1-58. 2009. (New Information).

281 Park EK, Yates DH, Wilson, D. Lung function profiles among subjects with non-malignant asbestos-related disorders. Saf Health Work. 2014 Dec;5(4):234-37. (New Information).

282 Harries PG. A comparison of mass and fibre concentrations of asbestos dust in shipyards insulation processes. Ann Occup Hyg. 1971;14(3):235-40. (New Information).

the membrane filter technique is recommended for estimating fibre concentration. This study provided fibre concentration measurements and showed high concentration of asbestos fibre levels associated with removal of sprayed crocidolite asbestos, removal of pipe lagging in particular in brick stowage space, and the application of pipe lagging-boiler rooms in the Naval Dockyards. The fibre concentrations measured in this study are likely not relevant to current conditions including those in the Australian military setting.

f. Harries[283] conducted a survey of asbestos fibres associated with working with asbestos insulating materials in ships being refitted in British Naval Dockyards. The membrane filter method of sampling and evaluation was used, and samples were also taken in other areas of the Dockyards. Very high concentrations of asbestos were recorded. This study showed there was widespread dispersion of high concentrations of asbestos dust throughout the ship, in particular, in areas of the ship which were not considered to be affected, unknowingly exposing men without respiratory protection to high concentrations of asbestos. The authors concluded that all processes involving work with asbestos insulating materials in Naval Dockyards give rise to asbestos dust concentrations of >2 fibre/mL, with many processes with dust concentrations of ≥50 fibre/mL. This study demonstrate that naval personnel working on the Dockyards were exposed to high concentration of asbestos, and in particular men working in small enclosed spaces were exposed to high levels of asbestos fibres.

Fibrosing Interstitial Lung Disease

g. Kawabata et al[284] retrospectively examined 1718 cases (1202 men) who underwent lobectomy for resection of pleuro pulmonary tumours. The presence of malignant pleural mesothelioma, pleural plaques, and asbestos bodies in the histological specimen were used as objective markers for asbestos exposure. Two groups were studied. Group 1 = 183 cases with asbestos exposure had higher rates of positive occupational history and histological usual interstitial pneumonia (31%) than the remaining 1535, and a small numbers of asbestos bodies were found in histological specimens of

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283 Harries PG. Asbestos dust concentrations in ship repairing: a practical approach to improving asbestos hygiene in naval dockyards. Ann Occup Hyg. 1971:14(3):241-54. (New Information).

284 Kawabata Y, Shimizu Y, Hoshi E, Murai K, Kanauchi T, Kurashima K, et al. Asbestos exposure increases the incidence of histologically confirmed usual interstitial pneumonia. Histopathology. 2015; Published Online 22 July 2015. (New Information).

21 cases of histological usual interstitial pneumonia. Group 2 = 239 with histological usual interstitial pneumonia, pleural plaques and asbestos bodies were more frequent than the remaining 1479 of cases without histological usual interstitial pneumonia. The authors concluded asbestos exposure causes asbestosis and increases the incidence of histological usual interstitial pneumonia.

h. Copley et al[285] studied the differences in thin-section CT features between asbestosis and idiopathic pulmonary fibrosis and tested the findings in a subset of histopathologically proved cases of usual interstitial pneumonia and nonspecific interstitial pneumonia. Consecutive patients with a diagnosis of idiopathic pulmonary fibrosis (n = 212) or asbestosis (n = 74) were included. The relationships derived from the initial comparison were tested in a separate group of biopsy-proved usual interstitial pneumonia (n = 30) and nonspecific interstitial pneumonia (n = 23) cases. The study showed those for the 212 people with idiopathic pulmonary fibrosis, fibrosis was present in all cases (n = 212); emphysema in 76 cases (36%); diffuse pleural thickening in 4 cases (2%) and pleural plaques were absent in all cases. For the 74 asbestosis cases, fibrosis was present in all cases (n = 74); emphysema in 25 cases (34%); pleural plaques in 58 cases (78%); and diffuse pleural thickening in 61 cases (82%). Both pleural thickening and pleural plaques were present for 49 cases (n = 49/74; 66%) and four patients (5%) had had no pleural disease. Fibrosis was more extensive in the idiopathic pulmonary fibrosis group (55%; range 5%–96%) than in the asbestosis group (26%; range 0.5%–90.5%) (p<.001). The study findings indicate that asbestosis closely resembles usual interstitial pneumonia but is strikingly different from nonspecific interstitial pneumonia. As patients with asbestosis had a coarser pattern of fibrosis than did the subgroup of patients with histopathologically proved nonspecific interstitial pneumonia. There was no statistically significant difference in coarseness between the asbestosis and biopsy-proved usual interstitial pneumonia groups.

[285] Copley SJ, Wells AU, Sivakumaran P, Rubens MB, Lee YC, Desai SR, et al. Asbestosis and idiopathic pulmonary fibrosis: comparison of thin-section CT feature. Radiol. 2003;229(3):731-36. (New Information).

Job Matrix

i. van Oyen et al[286] developed a job-exposure matrix (JEM) to estimate occupational asbestos exposure levels in Australia (other than Wittenoom mine) since 1943 using all relevant and available data. The data included exposure measurements from 57 industries and 89 occupations from 1937 to 2007 in Australia, as well as records from overseas. Exposure measurements also included occupations within the armed forces.[287] Intensity of exposure was assigned in five categories, based on the mid-point of five exposure ranges according to the Australian Standard for asbestos exposure 0.1 fibre/mL and expressed as a TWA for an 8 hour working day. Frequency of exposure was assigned over a working year of 240 days assuming 4 weeks holidays. A total of 537 combinations of 224 occupations and 60 industries with potential exposure to asbestos (mostly a mixture of asbestos varieties) were identified. The authors reported that asbestos workers, insulators, waterside workers (including shipyard, wharf, navy, and marine), carpenters/joiners, boilermakers / welders, power supply, and railway workers were estimated to have experienced the highest levels of occupational asbestos exposure. The JEM can provide a quantified estimate of asbestos exposure for any individual with a history of having worked in Australia, based on knowledge of the person's occupation, the industry or location, year started working, and time spent (in months or years) or year finished. Thereby summing the exposure estimates for each year, an estimate of the cumulative asbestos exposure that an individual has experienced over their working life in Australia is calculated. Ideally, direct comparison of JEMs with lung asbestos fibre counts is required for validation of this matrix.

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286 Van Oyen et al. Development of a job matrix (AsbJEM) to estimate occupational exposure to asbestos in Australia. Ann Ocup Hyg. 2015;59(6):737-48 and supplement tables S1 & S2 pp1-9. (New Information).

287 Occupations in the armed forces included driver; police officer; rigger; shipwright; welder; pilot navy; aircraft handler; communications officer; cook (army/air force); engineer (aircraft/civil/mechanical); engineer (military)/sapper; machine operator; officer; steward; storeman (non-marine); weapons technician (air force); cook (navy); coxswain; officer (navy); petty officer radio operator; radar operator; seaman; steward (navy); fireman; weapons technician (army); (ship's refitting, weapons technician (navy) electrical maintenance; electrician; engineer (navy);demolition (building) mechanic builder; fitter, shipyard labourer/worker); carpenter/joiner) boiler; and engine driver; boiler attendant; blacksmith (navy); shipwright (asbestos exposed); artificer; engine room attendant, marine engineer, stoker; insulation/lagging.

j. Hyland et al [288] developed an asbestos specific task exposure matrix (ASTEM) based on quantitative measurements from historical workplace reports in NSW, using asbestos dust exposure measurements (fibre/mL) taken during workplace inspections in the 1970s and 1980s. Exposure was calculated by using the proportion of time spent on each task multiplied by the ASTEM value for each task and product combination. All resulting values were added to provide a cumulative asbestos exposure for a worker in fibre/mL-years. An asbestos task exposure matrix was created using 19 task-product combinations chosen for the matrix, the matrix was task-based rather than full shift TWA measurements primarily due to a lack of TWA data.

k. Macfarlane et al[289] provided an overview of asbestos exposure in the Australian population, and described how the OccIDEAS exposure assessment tool is being adapted for use in the Australian Mesothelioma Registry to retrospectively assess asbestos exposure among mesothelioma cases. Within OccIDEAS a participant's full job history is entered based on his job history, a job-specific questionnaires (known as job specific module) to jobs which potentially involve exposure to asbestos is assigned. A structured interview (usually by telephone) is conducted with the participant, and the interviewer records the answers to the job specific module questions in OccIDEAS. Twelve job specific modules have been developed specifically for assessing asbestos exposure, and have been developed to cover all job categories likely to be reported and which have possible asbestos exposure in the Australian context.

108. The Council's view is that the above new information is likely to be relevant, should the RMA decide to conduct any future investigation concerning FILD and Asbestosis. The Council is of the view that the new information, particularly the job matrix papers by van Oyen et al, [290] Hyland et al, [291] and Macfarlane et al[292] are valuable papers,

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288 Hyland RA, Yates DH, Benke G, Sim M, Johnson AR. Occupational exposure to asbestos in New South Wales, Australia (1970-1989): development of an asbestos task exposure matrix. Occup Environ Med. 2010;67(3):201-6. (New Information).

289 Macfarlane E, Benke G, Sim MR, Fritschi L. OccIDEAS: An Innovative Tool to Assess Past Asbestos Exposure in the Australian Mesothelioma Registry. Saf Health Work. 2012;3(1):71-6. (New Information).

290 Van Oyen et al. Development of a job matrix (AsbJEM) to estimate occupational exposure to asbestos in Australia. Ann Occup Hyg. 2015;59(6):737-48 and supplement tables S1 & S2 pp1-9. (New Information).

291 Hyland et al. 2010. (New Information). *ibid.*

292 Macfarlane et al. 2012. (New Information). *ibid.*

especially as they refer specifically to the Australian context. Hence the Council made the declaration at [5].

Specialist Medical Review Council

Declaration and Reasons for Decisions

Section 196W

Veterans' Entitlements Act 1986

Re: Statements of Principles Nos. 53 and 54 of 2013 concerning fibrosing interstitial lung disease and Statements of Principles Nos. 55 and 56 of 2013 concerning asbestosis

Request for Review Declaration Nos. 27 and 28

APPENDICES

Appendix A	Tables Outlining the Sound Medical Scientific Evidence Table A1. Overview of 12 Papers Reporting Asbestos Cumulative Exposure Levels Table A2. Overview of 36 Papers Relevant to Low Level Exposure to Asbestos for Asbestosis Table A3. List of Medical Science Cited in the RMA Briefing Papers Table A4. New Information Identified by the Council and/ or the Applicant Table A5. New Information Identified by the Council on Diffuse Pleural Thickening
Appendix B	The constituted council and legislative framework of the review
Appendix C	Written and oral submissions
Appendix D	The Available Information, sent to the Council by the RMA under section 196K.

APPENDIX A: TABLES OUTLINING THE SOUND MEDICAL EVIDENCE

Table A1. Overview of 12 Papers Reporting Asbestos Cumulative Exposure Levels Associated with Producing Asbestosis (Ordered from Lowest to Highest Exposure Level)

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
Sluis-Cremer (1990)[1]	Cited by: Sluis-Cremer (1991)[2] RMA ID: 000440	Evidence for an Amphibole Asbestos Threshold Exposure for Asbestosis Assessed by Autopsy in South African Asbestos Miners	807 miners (100% men) who had autopsies to determine presence of pneumoconiosis 411= asbestosis deaths No smoking history	Amphibole mine (crocidolite; chrysotile workers excluded) Also exposure to silica and gold in mines Also likely environmental exposures near mills and mine dumps	Cumulative exposure: *N*=807; *M*=176.5 (*SD*=307.8) fibre/mL-year ≤1 (*n*=33) >1-2 (*n*=29) >2-5 (*n*=64) >5-10 (*n*=73) >10-20 (*n*=68) >20-50 (*n*=105) >50-200 (*n*=125) >200-300 (*n*=175) >300 (*n*=135) fibre/mL-year	Fibre concentration methods used: 1940-1965 Konimeters 1965-1975 Thermal Precipitator 1975-current Membrane Filter Method. Different fibre lengths and ratios for different time periods Chronological exposure was calculated using mean mine graph for time exposed and weighing	Duration of exposure: *N*=807; *M*=30.4 (*SD*=14.0) years ≤15 years (*n*=111) 16-30 years (*n*=338) 31-45 years (*n*=232) 45+ years (*n*=126)	**Post-mortem study reporting asbestos exposure producing asbestosis** Risk of asbestosis at cumulative exposures >2-5 fibre/mL-years (1 death at ≤15 years and 3 deaths at 16-30 years of exposure). Asbestosis increased with cumulative exposures fibre/mL-year and number of years since the onset of exposure. Cumulative exposure >300 fibre/mL-years and ≥45 years since the onset of exposure 82% (*n*=64/78) of the workers had asbestosis Author's conclusion: incidence of asbestosis at increasing average fibre levels and increasing duration of	Asbestos exposure producing asbestosis seen at >2-5 fibre/mL-years

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						the exposure by occupation		exposure times. When exposure was expressed as fibre/mL-years no asbestosis was found at autopsy when exposure was <2 fibre/mL-years, even after 31-45 years of residence time. In the group exposed to >2-5 fibre/mL-years asbestosis was found (4 deaths). If a threshold dose for asbestosis does exist it is at approximately 2 fibre/mL if off-shift exposure is ignored. No threshold demonstrated.	
Hein et al (2007)[3]	RMA ID: 067597	Follow-up Study of Chrysotile Textile Workers: Cohort Mortality and Exposure-Response	3072 textile plant workers (59% men) 36=asbestosis deaths (1960-2001) No smoking history	South Carolina chrysotile asbestos textile plant Small amounts of crocidolite from South Africa	Cumulative exposure: *N*=3072; Median=5.5 (range=0.1-699.8) fibre/mL-year	Fibre concentration method used: statistical modelling of nearly 6000 industrial hygiene samples taken over the period 1930-1975 and analysed using Phase	Duration of employment: *N*=3072; median=1.1 (range=0.1–46.8) years	**Mortality study of chrysotile exposure and cause of death as indicated within the National Death Index** 36 asbestosis deaths (US expected deaths = 0.15; SMR=232.5; 95%CI: 162.8, 321.9; *p*<0.01) (South Carolina expected deaths =0.33; SMR=108.2, 95%CI:	Asbestos exposure producing pneumoconiosis seen at 3-<16 fibre/mL-years

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						Contrast Microscopy. Specific job matrix used to calculate job exposure. Each day in the work history was assigned an exposure level based on the job exposure matrix and cumulative exposure was defined as the sum of the assigned exposure concentrations over all days worked.		75.8, 149.8). Mortality analysis (observed pneumoconiosis and other respiratory diseases deaths=85, asbestosis=42) showed increasing trends in pneumoconiosis SMRs with increasing cumulative exposure were observed overall from 3-<16 fibre/mL-years (SMR 2.43; (1.33-4.08). Asbestosis mortality (observed deaths=62) showed statistically significant interaction between cumulative exposure and age category was observed (*p*=0.026). Author's conclusion: association between estimated chrysotile exposure and mortality from asbestosis observed in previous updates of this cohort were confirmed with the addition of 11 years of follow-up. Cumulative exposure was positively and	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
								significantly associated with asbestosis mortality. Modelled exposure-response relationship showed no threshold for development of asbestosis.	
Ehrlich et al (1992)[4]	RMA ID: 034901	Long Term Radiological Effects of Short Term Exposure to Amosite Asbestos Among Factory Workers	Subset of 386 factory workers (100% men) with less than one year of exposure and low cumulative exposure (<25 fibre/mL-year) 238 had serial X-rays Smoking history available	Amosite asbestos factory (Imported from South Africa; very small amounts of chrysotile used at one time) Also residential exposure	5 -120 fibre/mL (>5μm long) Cumulative exposure: *n*=386; Median=25.1 (range=0.1 - 720) fibre/mL-year Exposure concentration: *M*=51.8 (*SD*=25) fibre/mL	Fibre concentration method used: Membrane Filter Method (1968 and 1971). Average fibre counts for each job title were estimated from measurements carried out at two other plants operated by the company after 1954; one in Tyler, Texas and another in Port Allegheny, Pennsylvania.	Duration of exposure: Median=0.5;6 months (range=0.1-13) years	**Radiological study reporting effect of short term amosite exposure producing parenchymal and pleural abnormalities.** Parenchymal abnormality (profusion >1/0) >20 years from first employment by cumulative exposure (fibre/mL-year): <5 fibre/mL-year: *n*=85,10(12%)= abnormal 5.1-25 fibre/mL-year: *n*=87, 12(14%)= abnormal 25.1-125 fibre/mL-year: *n*=119, 30(25%)= abnormal >125 fibre/mL-year: *n*=49,29(59%)= abnormal	Asbestos exposure producing parenchymal abnormality seen at <5 fibre/mL-years

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
								Pleural abnormality (any pleural thickening of the cheat wall or diaphragm): Time from first employment (decade) by R1: OR=1.67 95%CI: 1.15, 2.42; *p*=0.007. R2: OR=1.85 95%CI: 1.24, 2.76; *p*=0.002 Cumulative exposure (≤ 5 fibre/mL-years) showed a significant rate of abnormality (R1: 23%; R2:17%) and the prevalence of abnormality increased with increasing cumulative exposure Risk of developing small parenchymal opacities for an increment in cumulative exposure of 10 fibre/mL-years (R1: OR:1.06; 95% CI: 1 04, 1.09; *p*=0.000 and R2: OR:1 07; 95% CI: 1 04, 1.10; *p*=0.000) Author’s conclusion: exposure to high concentrations of amosite with follow up	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
								for ≥20 years, no cumulative exposure threshold for parenchymal and pleural fibrosis detected, and parenchymal and pleural progression were still detectable >20 years after the end of exposure. Exposure for ≥1 month was sufficient to produce radiological signs of parenchymal and pleural fibrosis. Those in the lowest cumulative exposure stratum <5 fibre/mL-years were found to have high rates of abnormality. Increase in risk of parenchymal abnormality of 6-7% for each fibre/ml-years.	
Larson et al (2010)[5]	RMA ID: 067598	Vermiculite Worker Mortality: Estimated Effects of Occupational Exposure	1862 mine and process workers 69=asbestosis deaths	Vermiculite mining and processing operation (tremolite and actinolite,	Cumulative exposure: *N*=1862; Median=4.3 (range=0.8-22.5) fibre/mL-year	Fibre concentration method used: Historical air sampling data were used to estimate the 8-hour time-	Duration of exposure: *N*=1862; Median=0.8 (25^{th}-75^{th} percentile 0.1-4.1 years)	**Retrospective cohort mortality study** Significantly elevated SMR for deaths from asbestosis =69 (expected=0.5; SMR=142.8; 95%CI:111.1,180.8)	Asbestos exposure producing asbestosis seen at 8.6 to <44.0 fibre/mL- years

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
		to Libby Amphibole	No smoking history	winchite and richterite)	Cumulative exposure - asbestosis: *n*=69; Median=39.0 (25th-75th percentile =14.6-283.2) fibre/mL-year	weighted average (TWA) fibre exposure. The proportion of each day spent at each location was calculated for each job title, and an 8-hour TWA exposure was estimated for each job at a given time. The lifetime cumulative fibre exposure for each worker was obtained by summing the cumulative fibre exposure for each job that worker held. Before 1967 samples collected using midget impinger and later samples using a membrane	Duration of exposure - asbestosis: *n*=69; Median=10.4 (25th-75th percentile 1.2–19.5) years	Workers who died from asbestosis had a much greater median lifetime cumulative fibre exposure (39.0 fibre/mL-year compared with 2.4 fibre/mL-year for censored (or alive) workers) and median length of employment (10.4 years compared to 0.8 years for censored (alive) workers) Cox models for death from asbestosis were statistically significant (with no lag and 20-year lag) for increase in the hazard of mortality, indicating <1% increase in the hazard of mortality with each additional fibre/mL-years of exposure (i.e. 1 fibre/mL-years) Relative risks (RRs) showed an increased RR with cumulative exposure, which did not become statistically significant for asbestosis until the	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						filter. Conversion factors used to convert between the two and 8 hr TWAs calculated.		3rd quartile. Cumulative fibre exposure (20 year lag) showed statistically significant risk from 8.6 to <44.0 fibre/mL-year (*n*=25; RR=8.0, 95%CI: 3.2, 19.5) and ≥44.0 fibre/mL-year (*n*= 32, RR= 11.8, 95%CI: 4.9, 28.7) Authors conclusion: statistically significant association from 8.6 fibre/mL- year for deaths from asbestosis with significantly elevated SMR=142.8 for asbestosis.	
Green et al (1997)[6]	RMA ID: 026469	Exposure and Mineralogical Correlates of Pulmonary Fibrosis in Chrysotile Asbestos Workers	54 asbestos workers necropsy reports (lung tissue for 39 workers) (44=men, 10=women) 34 controls necropsy reports (lung tissue for 31 workers)	Chrysotile asbestos textile plant Raw asbestos predominantly from Quebec with small amounts from BC and Zimbabwe; small amounts of crocidolite	Cumulative exposure: *N*=54; Median=30.2 (range=0.1-370.0) fibre/mL-year Men had larger cumulative exposures than women 34.6 and 25.6	Exposure assessment method used (published previously): Linear statistical models for reconstructing historic dust exposure levels, taking into account textile	Duration of employment: *N*=54; range=0.1->27.3 years Latency: Median=30.0 (range=17-44) years	**Case- control study examining relationship between chrysotile exposure and pulmonary fibrosis in a South Carolina textile plant** Asbestosis was usually present in asbestos textile workers with >20 fibre/mL-year cumulative exposure	Asbestos exposure producing asbestosis seen at 10-20 fibre/mL-years

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
			(22=men, 12=women) Fibre counts performed on 39 former textile workers and 31 controls. NB: small numbers Incomplete smoking history available	yarn woven but never carded, spun or twisted.	median fibre/mL-year respectively	processes, dust control measures, and job were developed. Estimates used 5,952 industrial hygiene samples from 1930-1975. Individual exposure data were not available for each worker in the cohort; estimates of exposure by job category at the plant were required. Both the exposure zone concept and the uniform task concept were used to develop exposure models which used historic plant production, control, and		Median fibrosis scores were greater in the asbestos workers than in the control population for **all** levels of exposure significant (*p*< 0.001) for exposure 7.2-36.5 fibre/mL-year- Chrysotile median 37.6 & tremolite median 5.0 asbestos fibre x10^6 dry lung *p*<0.05 36.6-109.5 fibre/mL-year Chrysotile median 30.8 & Tremolite median 6.4 asbestos fibre x10^6 dry lung *p*<0.05 109.5-370 fibre/mL-year Chrysotile median 104.5 & Tremolite median 27.8 asbestos fibre x10^6 dry lung *p*<0.05 Not significant for 0.1-7.1 fibre-years. Author's conclusion: Both cumulative exposures to asbestos and lung fibre burden were strongly correlated with	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						exposure data to estimate exposure by job and calendar period. Cumulative lifetime TWA exposure for each worker was calculated by multiplying estimates of exposure for each job held by the time spent in each job and was expressed as fibre-year.		severity of asbestosis. Lack of statistical power in the study was largely due to the small number of cases with low and intermediate levels of exposure, making it not possible to detect a threshold dose for the development of asbestosis. Asbestosis of grade 2 severity or more was consistently found in workers with lifetime cumulative exposures in excess of 20 fibre/mL-year and in three cases with exposures ranging from 10-20 fibre/mL-year Authors' conclusion: histologic asbestosis was usually detectable with exposures of >20 fibre/mL-year and a few cases were found with estimated cumulative exposures of 10-20 fibre/mL-year.	
McDonald et al (2004)[7]	Cited by: Antao et al	Mortality in a Cohort of Vermiculite	406 mine workers (100% men) with net	Vermiculite mine/mill	Exposure Concentration: *M*=18 fibre/mL	Fibre concentration method used:	Duration of exposure: *M*=9 years	**Cohort mortality study reporting deaths in vermiculite**	Asbestos exposure producing non-

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
	(2012)[8] RMA ID: 068460	Miners Exposed to Fibrous Amphibole in Libby, Montana	service of at least 1 year before 1.1.63 285=deaths 51=non-malignant respiratory disease deaths No smoking history			Standard Optical Microscope Measurements were scanty and, until about 1970, by Midget Impinger method only. After 1970 Membrane Filter Method was used. Estimating cumulative exposures was to take the maximum advantage of existing air measurements and to concentrate efforts on the past dry mill period (before 1975) when substantial exposures occurred. Altogether, results of 1363 air measurements		**miners from Libby, Montana** Non-malignant respiratory disease associated with cumulative exposure: SMR=3.09 (95% CI: 2.30, 4.06). Non-malignant respiratory disease (*n*=51): Cumulative exposure of 11.7- (16.7) fibre/mL-year (deaths=13, expected death =3.7; RR=2.53; 95%CI: 0.88, 7.24) Cumulative exposure at 25.2- (53.2) fibre/mL-year (deaths=14, expected deaths=3.8; RR=2.62 (95%CI: 0.93, 7.27). Cumulative exposure at 113.8- (393.8) fibre/mL-year (deaths=19, expected deaths=4.1; RR=3.11; 95% CI: 1.15, 8.44). Linear model (per 100 fibre/mL-year) RR=0.38 (95%CI: 0.12, 0.96; *p*=0.0001) Author's conclusion: a statistically significant	malignant respiratory disease seen at 11.7 fibre/mL-year

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						were available for the period before1975. Used job descriptions to estimate for each job the duration and the number of hours spent by workers in different operation locations; the cumulative exposure was then calculated		linear trend between exposure and deaths from non-malignant respiratory disease. The all-cause linear model would imply a 14% increase in mortality for mine workers exposed occupationally to 100 fibre/mL-year or about 3.2% for a general population exposed for 50 years to an ambient concentration of 0.1 fibre/mL – not insignificant impact.	
Rohs et al (2008)[9]	RMA ID: 067995	Low-Level Fiber-Induced Radiographic Changes Caused by Libby Vermiculite. A 25-Year Follow-up Study (Chest radiographic changes only)	280 mine workers of the original 513 cohort (94.3% men) 80=pleural changes (68=localised pleural plaques 12=diffuse pleural thickening, 28.7%) 8=Interstitial changes	Vermiculite mine/mill	Cumulative exposure: *N*=280 *M*=2.48 (*SD*=4.19; range=0.01-19.03) fibre/mL-year Cumulative exposure - Interstitial changes (*n*=8): *M*= 11.86	Fibre concentration method used: Membrane filters method since 1972. Cumulative fibre exposure was calculated using the 8-hour TWA by the number of years at the TWA summed overall years according to	Duration of exposure: N=280 based on hire date Hired on or before 1973 *n* =186 Hired after 1973 *n*= 94	**Radiographic study reporting vermiculite - induced interstitial change and pleural changes in a cohort of vermiculite workers** Significant increase seen in the follow-up in pleural changes: 2.0% in the 1980 study to 28.7% in 2005. Interstitial changes: 0.2% in 1980 to 2.9% in 2005	Asbestos exposure producing interstitial changes seen at 11.37 fibre/mL-year

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
			(irregular opacities, profusion of ≥1/0; 2.9%) NB. Small numbers of workers with interstitial changes (*n*=8) Smoking history available		(*SD*=6.46) fibre/mL-year All 8 with interstitial changes occurred at the 72% or greater maximum cumulative fibre exposure range, 6 occurring at the 90% or greater range.	department. Due to new environmental controls, there was a decrease in fibre exposure after 1973. Therefore, each department was assigned two values, fibre exposures through 1973 and exposures after 1973. Individuals were assigned a cumulative fibre exposure value, which was the summation of estimated fibre exposure by department, based on the years employed between 1963 and 1980.		Pleural changes directly related to cumulative fibre exposure, greatest prevalence (54.3%) in the highest exposure quartile (range=2.21-19.03 fibre/mL-year) of workers Pleural changes can occur at low lifetime cumulative fibre exposure levels as demonstrated by the 12% prevalence within participants and living non participants with <1.92 fibre/ mL-years cumulative fibre exposure, and 20% prevalence within participants with <2.21 fibre/mL-year cumulative exposure 2.9% of participants had interstitial changes and were significantly related to cumulative fibre exposure mean exposure of 11.37 fibre/mL-year Author's conclusion: exposures to asbestos	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
								among users of Libby vermiculite ore within an industrial process cause pleural thickening at low lifetime cumulative fibre exposure levels of <2.21 fibre/mL-year.	
Paris et al (2004)[10]	RMA ID: 067600	Factors Associated with Early-Stage Pulmonary Fibrosis as Determined by High-Resolution Computed Tomography Among Persons Occupationally Exposed to Asbestos	706 retired workers exposed to asbestos (89% men) 51=pulmonary fibrosis (grade 2- bilateral interstitial abnormalities limited in extent but consistent with asbestos-related pulmonary fibrosis or grade 3-profuse bilateral interstitial abnormalities visible on several slices) diagnosed by HRCT (only 38 diagnosed asbestosis -	Asbestos textile and friction material factory, shipyards, fossil fuel power stations, and industrial insulation	Cumulative exposure: *N=706; M*=137.9 (*SD* =136.4) fibre/mL-year 112 participants (16%)=<25 fibre/mL-year Only 2 subjects with HRCT pulmonary fibrosis exposed to <25 fibre/mL-year (9.3 and 15 fibre/mL-year respectively) 52 participants with <25 fibre/mL-year had no symptoms or	Fibre concentration method used: For the participants who had worked in the asbestos textile and friction material plant a specific job-exposure matrix determined from air-borne samples collected annually between 1959 and 1999 in the various workshops of the plant. For all the other	Duration of exposure: *N=706; M*=24.9 (*SD*=9.1) years	**Radiological study reporting relationship between asbestos exposure and HRCT findings** There were 51 pulmonary fibrosis (grade 2 or 3; different scoring system to ILO) diagnosed by HRCT. Only 38 diagnosed with pulmonary fibrosis according to American Thoracic Society criteria[b], 9 which were diagnosed by HRCT For 112 participants the cumulative exposure level was <25 fibre/mL-year of which 2 subjects had HRCT asbestosis Significant association between HRCT fibrosis and cumulative asbestos exposure,	Asbestos exposure producing asbestosis seen at 25-99.9 fibre/mL-year

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
			small opacity profusion of ≥1/1 according to American Thoracic Society criteria[b]) Excluded those with known asbestos-related diseases Aims of study were to clarify group suitable for screening by HRCT Variable asbestos exposure although the main exposure types unclear Smoking history available		abnormal examinations no case of HRCT asbestosis detected. 60 participants with <25 fibre/mL-year, 2 cases were detected, both having basilar crackles and one having small irregular opacities on X-ray	participants, the asbestos exposure level associated with each job was assessed using published airborne measurements available in the French database Cumulative-exposure index was then calculated by summing the values for all job positions held, with reference to the occupational calendar established in the interview, the products of the job exposure level (in fibre/mL) by job duration (in years).		with a clear dose-response relationship for 25-99.9 fibre/mL-year (OR=3.4; 95% CI: 0.8, 15.2; *p*>0.05) and≥100 fibre/mL-year (OR=6.1; 95% CI: 1.5, 25.9) in comparison with ≤25 fibre/mL-year (Trend test *p* =0.002) but not with duration of exposure (Trend test *p*=0.791). Author's conclusions: significant association between HRCT fibrosis and cumulative asbestos exposure, with a clear dose-response relationship. Only 2 cases of HRCT pulmonary fibrosis (2%) among the 112 patients with a cumulative fibre exposure of <25 fibre/mL -years. HRCT can detect early-stage asbestosis in people who have been highly exposed to asbestos and whose X-ray can be considered normal. HRCT screening does not seem warranted	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						Dust measurements performed in numerous points of the workshops: Membrane Filter Method since 1973: phase-contrast light microscopy counts of fibres performed by an accredited laboratory between 1960–1974, samples were collected on Soluble Filters by an AvyRaille`re-Martin (ARM) type of apparatus, and the light microscopy fibre counts Measurements were performed by a specialised independent		for people with low occupational exposure such as cumulative exposure level <25 fibre/mL-year, especially in the absence of other asbestosis-related alterations.	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						laboratory as no atmospheric measurements were available for the period before 1960.			
Sullivan (2007)[11]	RMA ID: 045641	Vermiculite, Respiratory Disease, and Asbestos Exposure In Libby, Montana: Update of a Cohort Mortality Study	1672 mine and mill workers (all men) 40=asbestosis deaths 22=asbestosis deaths Followed May 1982-Dec 2002 No smoking history	Vermiculite mine/mill Miners, millers and processors	Cumulative exposure: *N*=1672; Median=8.7 fibre/mL-year All deaths: *n*=767; Median= 21.0 fibre/mL-year Asbestosis: *n*=40; Median=228.4 fibre/mL-year (worked <1year =36.2 fibre/mL-year and worked ≥1 year =244.8 fibre/mL-year	Fibre concentration method used: Job-exposure matrix exposure index available for this and previous studies of Libby workers is based on fibre count data obtained using Optical Phase Contrast Microscopy of all asbestiform fibres, of which 6% were estimated to be tremolite	Duration of exposure: *N*=1672; *M*=4.0 (range=1day -43.1) years Asbestosis deaths: *n*=40 (5.3% of cohort); mean duration of employment in those with asbestosis *M*=14.6 years	**Historical cohort mortality study reporting vermiculite exposure** Based on 22 asbestosis deaths SMR increased with increasing cumulative exposure: <0-49.9 fibre/mL-year exposure (SMR=37.3; 95% CI:7.5, 122.3); SMR for non-malignant respiratory death was 1.9 for CFE < 3.5 fibre/mL-year 50.0-249.9 fibre/mL-year (SMR =212.6; 95%CI: 91.6, 433.2) and ≥250 fibre/mL-year (SMR=749.1; 95% CI: 373.0, 1367.8) allowing for a 15-year exposure lag More likely than expected to die from	Asbestos exposure producing asbestosis seen at ≥50 fibre/mL-year

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
								asbestosis with employment duration <15 months (SMR=38.2; 95% CI: 7.7, 125.1), 15 months to 9.9 years (SMR=236.0; 95%CI: 107.8, 461.1) and employment duration ≥10 years (SMR=628.6; 95%CI: 301.1, 1185.1) Author's conclusion: SRR was statistically significant asbestosis increased across categories of exposure duration (≥15 months) and over increasing categories of cumulative exposure from ≥50 fibre/mL-year asbestosis. Note levels of likely exposure were high in this mill.	
Berry et al (1979)[12]	Cited by: Becklake (1991)[13] RMA ID: 000429	Asbestosis: a study of dose-response relationships in an asbestos textile factory	379 textile factory workers (100% men) with >10 years' service 58 =possible asbestosis (34 =	Chrysotile and crocidolite asbestos textile factory	Group first employed after 1950 cumulative exposure: (*n*=197) *M*=84 fibre/mL-year	Fibre concentration method used: For each job description a dust level was calculated for each year by	Duration of exposure*:* *M*=20.1 years Group first employed after 1950 (*n*=197) the average	**Morbidity study reporting asbestos exposure producing asbestosis and crepitation's** For possible and certified asbestosis the 1% prevalence are	Asbestos exposure producing asbestosis seen 84 fibre/mL-year

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
			certified as asbestosis) Diagnosis and examination by Pneumoconiosis Medical Panel 82=crepitation's Smoking history available		Concentration of exposure: *M*=5.0 fibre/mL	taking the average of the levels measured at the static dust sampling locations in the area where the job was carried out. Fibre counts were not available for 1951-60, but Thermal Precipitator particle counts were available for 1952 and 1960. The fibre counts for 1951-1955 were taken as those of 1961 multiplied by the ratio of the 1952 to the 1960 thermal precipitator measurement. The counts for 1956-1960 were taken to	follow-up since first exposure: *M*=16 years	estimated at 55 fibre/mL-year and 72 fibre/mL-year. Prevalence of small irregular opacities graded 1/0 or more = 21.2% pleural changes = 7%. 4/36 subjects with exposures < 50 fibre/mL-year had signs of asbestosis. Author's conclusion: For possible and certified asbestosis the 1% prevalence's estimated at 55 and 72 fibre/mL-year respectively. The most reliable data relate to men first employed after 1950 the average cumulative exposure was 84 fibre/mL-year, the average follow-up since first exposure was 16 years, and the prevalence of possible asbestosis was 6.6 %. Higher prevalence of crepitation's at any dose than previously observed. No room for complacency regarding the 2 fibre/cm^3 standard of	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						be the same as 1961-1965. Before 1950 no dust measurements For 1933-45 the concentrations were taken to be 1.5 times those in 1951 and for 1946-1950 as 1-25 times the 1951 values.		the British Occupational Hygiene Society.	
Seidman et al (1986)[14]	RMA ID: 020152	Mortality Experience of Amosite Asbestos Factory Workers: Dose-Response Relationships 5 to 40 Years After Onset of Short-Term Work Exposures	820 asbestos factory workers (100% men) 31=asbestosis deaths No smoking history	Amosite asbestos factory	Concentration of exposure: Median=50 (range=5-120) fibre/mL	Fibre concentration method used: There were no direct fibre count measurements or exposures in this plant. Average fibre counts for each job title were estimated from measurements carried out at two other plants operated by	Duration of exposure: *N*=820; range=<1 month to 2-14 years *n*=384:<6 *n*=241:6-24 *n*=195:24+ months	**Mortality study after short-term work exposure** The lower the cumulative exposure dose, the longer it took for adverse mortality experience to become evident and also the smaller the magnitude of that adverse mortality. Workers exposure to <25 fibre/mL-year cumulative exposure observed and expected deaths from 5 to 40 elapsed years since onset work were:	Asbestos exposure producing asbestosis seen at 100 fibre/mL-year

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
						the company after 1954; one in Tyler, Texas and another in Port Allegheny, Pennsylvania.		109 men exposed to 6.0 -11.9 fibre/mL-year (asbestosis deaths=1; seen at 5-24 years since onset of work, SMR not reported if both observed and expected deaths are <5) 139 men exposed to 12.0-24.9 fibre/mL-year (asbestosis deaths=1; see at 5-19 years since onset of work, SMR not reported) 57 men exposed to 100.0-149.9 fibre/mL-year (asbestosis deaths=5; significant increase seen from 5-24 years (*n*=4) since onset of work, SMR = 15.63; *p*<0.001) 58 men exposed to 150.0-249.9 fibre/mL-year (asbestosis deaths=7; significant increase seen from 5-29 years (*n*=6) since onset of work, SMR = 13.04; *p*<0.001) 53 men exposed to ≥250 fibre/mL-year	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
								(asbestosis deaths=13; significant increase seen from 5-19 years (*n*=6) since onset of work, SMR = 36.84; *p*<0.001) Dose response relationship observed at all durations of employment Authors conclusion: with lighter (and/or shorter) direct exposure, as time goes on, the longer the time after onset of work, the more pronounced the excesses in mortality	
Finkelstein & Vingilis (1984)[15]	RMA ID: 000454	Radiographic Abnormalities Among Asbestos-Cement Workers: an Exposure-Response Study	181 asbestos cement workers (100% men) 51(28%)=small irregular opacities 1/1 or more 40 (22%)= pleural thickening of category A or more	Chrysotile and crocidolite asbestos, silica, and cement factory	18 year cumulative exposures: *N*=181; *n* =32:0-49.9 *n* =68: 50-99.9 *n* =41: 100-149.9 *n* =25: 150-199.9 *n* =15: >200 fibre/mL-year	Fibre concentration method used: Cumulative exposures to asbestos were calculated using a model that extrapolated measurements made by the personal Membrane Filter.	Duration of exposure: ≥1 year	**Morbidity study reporting asbestos exposure producing asbestosis and pleural plaques** Risk of small irregular opacities category >1/0 was seen at >200 fibre/mL-year (All men *n*=60; RR=2.24;Trend test (X^2) =27.9; *p*<0.001) Risk of small irregular opacities category	Asbestos exposure producing small irregular opacities 1/1 or more seen at 150-199.9 fibre/mL-year

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
			Excluded 5 men who had died of unstated cause. Smoking history available			Exposures were estimated to be accurate to a factor of 3-5. Eighteen-year cumulative exposures were calculated by summing annual exposures during the 18 year from first exposure Silica exposure not assessed (also can produce interstitial opacities on chest radiographs)		>1/1 was seen at >200 fibre/mL-year (All men *n*=50; RR=2.57;Trend test (X^2) =24.4; *p*<0.001) Risk of small irregular opacities category >1/2 was seen 150-199.9 fibre/mL-year (All men *n*=11; RR=2.19;Trend test (X^2) =18.0; *p*<0.001) Risk of bilateral pleural thickening (grade A) was seen at >200 fibre/mL-year (All men *n* =40; RR=1.44;Trend test (X^2) =4.34; *p*=0.04) Risk of bilateral pleural thickening (grade B) was seen at >200 fibre/mL-year (All men *n* =23; RR=1.38;Trend test (X^2) =2.36; *p*=0.12) Author's conclusion: 20 years from 1st exposure mortality rates were elevated. Men leaving asbestos exposure had a risk of radiographic progression similar to	

Original Study Author (RMA ID) *Or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL-year)	Exposure Assessment Method	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure producing Asbestosis; then Other Studies & Reviews	Comparison of the Studies Dose to that of the Asbestosis SoPs
								men continuing in exposure and men with abnormal radiographs had a higher mortality rate than men with normal chest X-rays. Increased risk of cancer with interstitial opacities and bilateral pleural changes.	

[a] Participants with small opacity ILO profusion of grade 1/1 on X-rays were considered to have asbestosis according to the American Thoracic Society criteria, whether or not they were found to have inspiratory crackles, restrictive pattern impairment, or a low T_{LCO}. Pleural abnormalities were defined by the presence of the ILO- criteria for circumscribed pleural plaques or diffuse pleural thickening. Abbreviations: TWA= time weighted average; SMR= standardised mortality rate; SRR = standardised rate ratio; *M* = mean; *SD* = standard deviation; OR = odds ratio; RR = relative risk; NS = non-significant

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4 Ehrlich et al. Long term radiological effects of short term exposure to amosite asbestos among factory workers. Br J Ind Med. 1992;49(4):268-72. (RMA ID: 034901).

5 Larson et al. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 2010;52(5):555-60. (RMA ID: 067598).

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[15] Finkelstein MM, Vingilis JJ. Radiographic abnormalities among asbestos-cement workers: an exposure-response study. Am Rev Respir Dis. 1984;129(1):17-22. (RMA ID: 000454).

Table A2. Overview of 36 Papers Relevant to Low Level Exposure to Asbestos for Asbestosis (Ordered by Study Type and Date)

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
Mortality Studies							
Paris et al (2009)[1]	RMA ID: 067601	Pleural Plaques and Asbestosis: Dose- and Time-Response Relationships Based on HRCT Data	5545 retired workers exposure to asbestos (100% men) 882=pleural plaques 375=asbestosis	Metal, construction and repairs industry	Cumulative Exposure Index[a]: *M*=49.6 (Estimated 80% had high level of exposure) Concentration of exposure (fibre/mL) not reported	Duration of exposure: *M*=27.4 (*SD*=11.1) years Time since 1st exposure: *M*=41.9 (*SD*=8.1) years	Morbidity study reporting asbestos exposure producing asbestosis and pleural plaques. Pleural plaques: Overall (*n*=882) Trend test *p*<0.0001. High exposure (*n*=777; 17.5%) OR significant (OR=2.89; 95%CI: 1.51, 5.52; *p*<0.0001). Asbestosis: Overall (*n*=375) Trend test *p*=0.01. High exposure (*n*=322; 7.3%) OR significant (OR=1.20; 95%CI: 0.62, 2.30; *p*=0.02). Only cumulative exposure (*p*<0.0001) or level of exposure (*p*=0.02) were significantly associated with asbestosis. Both time and dose-response relationships were demonstrated for pleural plaques. Only dose-response relationships were shown for asbestosis. Author's conclusion: Both time-response and dose-response relationships were demonstrated for pleural plaques, while only dose-response relationships were demonstrated for asbestosis.
Reid et al (2005)[2]	RMA ID: 037507	The Effect of Asbestosis on Lung Cancer Risk Beyond the Dose Related Effect of Asbestos Alone	1988 (1196 former Wittenoom workers; 792 residents of Wittenoom) cancer prevention program participants 58=lung cancer cases(21= asbestosis)	Crocidolite mine/mill	Cumulative exposure: Cases (*n*=58 lung cancer): *M*=11 (range=2.6-46.5) fibre/mL-year Controls (*n*=1930 non-cases): *M*=5	Duration of exposure (days): Cases (*n*=58 lung cancer): *M*=330 (range=118-1048) days Controls(*n*=1930 non-cases): *M*=285	Morbidity study reporting asbestos exposure producing asbestosis in lung cancer patients. 58 lung cancer cases 21; 36% had radiographic asbestosis compared to 220 (11%) of participants. Radiographic asbestosis (OR=1.94, 95% CI: 1.09, 3.46) and asbestos exposure (OR=1.21 per fibre/mL-year, 95% CI: 1.02, 1.42) significantly

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
			1930=non-cases (220=asbestosis)		(range=1.8-13.5) fibre/mL-year Concentration of exposure: Cases (*n*=58 lung cancer): *M*=12 (range=6.2-40.3) fibre/mL Controls: (*n*=1930 non-cases): *M*=6 (range=2.1-20.3) fibre/mL	(range=106-854) days	associated with an increased risk of lung cancer. Intensity of exposure and cumulative exposure were both statistically significant. The interaction between cumulative asbestos exposure and asbestosis was not significant (Hazard Ratio = 0.82, 95% CI: 0.59, 1.13, *p* = 0.23). Author's conclusion: asbestos fibres cause lung cancer in persons exposed to asbestos. The presence of asbestosis is also associated with an increased risk of lung cancer which may be due to some action of the fibrosis itself, but the excess risk may also be due to an underestimate of asbestos exposure.
Peipins et al (2003)[3] RMA ID: 046972	RMA ID: 046972	Radiographic Abnormalities and Exposure to Asbestos-Contaminated Vermiculite in the Community of Libby, Montana, USA	7307 participants of a Community based medical screening program (6668 chest X-rays) Nearly 18%= radiographic pleural abnormalities and asbestos exposure 1%= interstitial abnormalities (opacities profusion ≥1/0)	Occupational and/or community exposure from Libby, Montana	Not reported	Duration of exposure: ≥6 months	Morbidity study reporting asbestos exposure producing asbestosis, pleural changes and pleural thickening. There were 29 occupational, recreational, household, and other exposure pathway examined and the prevalence of pleural abnormalities increased with the increasing number of exposures. Ranging from 6.7% for those without exposure to 34.6% for those ≥12 pathways. Pleural abnormalities in 51% of the 365 and interstitial abnormalities in 3.8% workers at the mine and associated facilities. Authors conclusion: the factors most strongly associated with pleural abnormalities were being; a former Libby mine

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							worker; older, a household contact of a Libby mine worker; and male
Fischer et al (2002)[4]	RMA ID: 026590	Fibre-Years, Pulmonary Asbestos Burden and Asbestosis	366 patients on the German Mesothelioma Register 64=asbestosis cases 193=without elevated pulmonary burden	German Industry exposure	Exposure: range=0.094-314 fibre/mL-year	Not reported	Morbidity study reporting asbestos exposure producing asbestosis. Without elevated pulmonary burden (*n*=193): 19.6%=cumulative asbestos exposure of ≥25 fibre/mL-year. Asbestosis cases (*n*=64): 58% = cumulative asbestos exposure of ≥25 fibre/mL-year and 42% = cumulative asbestos exposure of <25 fibre-year. In contrast 24% of the patients with >25 fibre-year showed no elevated levels of pulmonary asbestos burden and no asbestos-associated lung fibroses. Using the criterion of asbestos fibre concentration of 25 fibre/mL-year showed 42% false-negative and 24% false-positive result. Author conclusion: the criterion of 25 fibre/mL-year alone is an insufficient parameter for the industrial disease adjudication procedure in the sector of asbestos associated lung fibroses.
Koskinen et al (1998)[5]	RMA ID: 026513	Radiographic Abnormalities Among Finnish Construction, Shipyard and Asbestos Industry Workers	18 943 workers screened (96%=men) 4133 (22%)= positive radiological finding (96% abnormalities in the pleura) 4%=asbestosis	Finnish construction, shipyard, and asbestos industry workers	Not reported	Duration exposure: Construction: *M*=9.0 years Shipyards: *M*= 7.8 years Asbestos industry: *M*= 9.7 years	Morbidity study reporting asbestos exposure producing asbestosis and pleural plaques. Pleural plaques were diagnosed for two-thirds of the workers with small irregular lung opacities (at least ILO 1/0), 52% were found to have bilateral pleural plaques, 20% unilateral plaques, and 28% no plaque change. Pleural plaques were observed in < half of workers with small irregular opacities and <15 years

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
							of exposure, but after a long period of exposure (>30 years) pleural plaques were present in 80% of cases. Prevalence of positive findings increased with duration of exposure, 18% among those who had been exposed to asbestos for <10 years (*n*=12 507), 27% among those exposed for 10-30 years (*n*=5542), and 43% among those exposed for > 30 years (*n*=914). Author's conclusion: parenchymal fibrosis may have developed after shorter and possibly larger asbestos exposure, whereas pleural plaques require a longer period to become detectable by chest X-ray.
Klaas (1993)[6]	RMA ID: 000435	A Diagnostic Approach to Asbestosis, Utilizing Clinical Criteria, High Resolution Computed Tomography (HRCT), and Gallium Scanning	75 previous workers (100% men) 16=asbestosis cases	Shipyard welders, riggers, pipe fitters, longshoremen boilermakers, machinists, janitors insulation strippers, and labourers	Not reported	Duration of exposure: *N*=75;*M*=22.7 (range=2-42) years Asbestosis cases: *n*=16; *M*=23.3 (latency *M*=35.9) years	Morbidity study reporting asbestos exposure producing asbestosis and pleural plaques. Of 75 men, 16 (21%) met the clinical asbestosis diagnosis criteria, 59 (75%) had both a positive HRCT and a positive gallium scan for asbestosis. The 16 men with asbestosis, 14 (87%) had asbestosis and 10 (62%) had pleural plaques on chest X-ray. Control group (*n*=59): 43 (73%) had pleural plaques on chest X-ray. Author's conclusions: 21% of the subjects satisfied commonly accepted criteria for the diagnosis of asbestosis, 75% had evidence of disease by both HRCT and gallium scanning. Therefore the current six-criterion approach may underestimate the presence of asbestos-related pulmonary disease in asbestos

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
							workers. HRCT and gallium scanning detect lesser degrees of asbestos-related pulmonary disease.
Jarvholm (1992)[7]	RMA ID: 035335	Pleural Plaques and Exposure to Asbestos: A Mathematical Model	2423 Swedish shipyard workers (100% men)	Shipyard workers exposed to asbestos Göteborg, Sweden	Not reported	Not reported	Morbidity study reporting asbestos exposure producing pleural plaques. Increase in prevalence beginning at about 10-14 years after onset of exposure. Incidence of pleural plaques had a latency period around 13 years. Authors conclusion: the incidence of pleural plaques attributable to asbestos exposure in humans can be modelled as: $1(t) = K(t-w)^a$ *for* $t>w$ where *K* is a constant depending on intensity of asbestos exposure, *a* constant around 0.4 and *w* a minimum latency period of around 13 years.
Lilis et al (1991)[8]	RMA ID: 046916	Radiographic Abnormalities in Asbestos Insulators: Effects of Duration from Onset of Exposure and Smoking. Relationships of Dyspnea with Parenchymal and Pleural Fibrosis	2790 asbestos insulators 439 (15.7%)= normal chest X-ray 668 (23.9%)= pleural changes 325 (11.6%)= parenchymal changes 1358 (48.7%)= both parenchymal and pleural changes	Asbestos insulation workers	Not reported	Duration of exposure: *n*=368;<30 years *n*=1712;30-39 years *n*=710;≥40 years	Morbidity study reporting asbestos exposure producing parenchymal and pleural changes. Prevalence of radiographic parenchymal abnormalities changes increased significantly (p<0.001) from 38.6% duration from onset of exposure (<30 years) to 69.7% (≥40 years). Pleural abnormalities changes the comparative prevalence's were 55.4% and 82.4%.Author conclusion: each of the three categories of duration from onset of exposure prevalence of interstitial pulmonary fibrosis was consistently higher in persons with a history of cigarette smoking than workers who had never smoked.

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
Selikoff & Lilis (1991)[9]	RMA ID: 035756	Radiological Abnormalities Among Sheet-Metal Workers in the Construction Industry in the United States and Canada: Relationship to Asbestos Exposure	1330 sheet metal workers (100% men)	Sheet metal	Not reported	Duration from onset of exposure: *M*=39.51 (*SD*=7.41) years For 76.4% (1016/1330) men employed as sheet-metal workers for ≥35 years	Morbidity study reporting asbestos exposure producing asbestosis and pleural plaques. As the number of years exposed increased, so did the prevalence of radiologic abnormalities and breathing difficulties. Radiologic changes were seen in 58.9% of the 1330 men. These changes occurred more frequently in the subgroup of 1016 workers who had been exposed ≥35 years (63.3%). Author's conclusion: majority of individuals who have been employed ≥30 years now have abnormal chest X-rays that resulted from exposure, and the less experienced men face the likelihood of developing similar changes over time.
Selikoff et al (1990)[10]	RMA ID: 035792	Asbestotic Radiological Abnormalities Among United States Merchant Marine Seamen	3324 seamen (100% men) 1157 (34.8%)= pneumoconiosis 329 (9.9%) (profusion ≥1/0) 227 (6.8%) (pleural thickening, pleural plaques, with or without calcification) 601 (18.1%) (only radiological change)	Merchant marine seamen	Not reported	Duration of exposure: *M*=>20 years	Morbidity study reporting asbestos exposure producing pneumoconiosis and pleural plaques. Prevalence rate for all radiological asbestos related abnormalities (pleural or parenchymal or both) highest level (38.5%) >40 years from onset of exposure. Authors conclusion: prevalence of asbestotic changes was greater among seamen who had served in the engine department (391/420; 425%) compared with seamen in other departments, including deck (301/820; 36.6%), steward (278/ 981; 28.4%), or with service in multiple departments (167/541; 30.9%)

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
Sluis-Cremer (1989)[11]	RMA ID: 035796	Progression of Irregular Opacities in Asbestos Miners	1454 South African asbestos mine and mill workers (100% men) (661 discontinued work; 793 continued work)	Amphibole mine/mill	*Mining exposure up to 1st X-ray:* exposure after 1st X-Ray: Yes=793; *M*=38.4 fibre/mL-year No=661; *M*=23.2 fibre/mL-year *Mining exposure between the 1st and 2nd X-ray:* exposure after 1st X-Ray Yes=793;*M*=6.2 fibre/mL-year	*Mining exposure up to* 1st *X-ray:* exposure after 1st X-Ray Yes=793; *M*=6.7 years No=661; *M*=4.2 years *Mining exposure between the* 1st *and 2nd X-ray:* exposure after 1st X-Ray Yes=793; *M*=4.5 years	Morbidity study reporting asbestos exposure producing asbestosis and pleural plaques. No difference in the frequency of disease incidence or rate of progression between those who discontinued work (discontinued asbestos exposure) and continued work (asbestos exposure) <5 years of asbestos exposure and in those with <5 fibre/mL-year cumulative exposure a progression of irregular opacities. Authors conclusion: relatively short period of follow up means that the group who continued exposure had only a mean of 4.5 years of exposure and 6.2 fibre-years after the first radiograph and that the time may have been too short for the additional exposure to take effect and the dose too low. Non-significant.
Cookson et al (1986)[12]	RMA ID: 000444	Prevalence of Radiographic Asbestosis in Crocidolite Miners and Millers at Wittenoom, Western Australia	859 mine and mill workers (100% men) 541 = X-ray from 1st date of employment 318 = comparison group with no known exposure to crocidolite at the time of X-ray 74 = men randomly selected from those known to be alive for a new radiographic	Crocidolite mine/mill	Cumulative exposure: 541 men=9.6 fibre/mL-year 318 men=5.3 fibre/mL-year 74 men followed up: 8.1 fibre/mL-year	Total days worked:541 men=126 days 318 men=65 days 74 men=101 days Average duration of stay = <4 months	Morbidity study reporting asbestos exposure producing radiographic changes of small irregular or small rounded opacities. 299 men = no exposure, 5 = abnormal X-ray and 432 men =exposed had 45 abnormal X-ray. Increases in prevalence of radiographic abnormality both with age at the time of the radiograph from (0% at age 15-24 to 22.2% at age ≥65) and years since first exposed (from 1.6% with no exposure to 26.6% more than 20 years after first exposure. Linear increase in log odds of the log of days (364 days) since starting work after the

Original Study Author (RMA ID) *or* Review of that Study	**RMA ID *Or* Review Author (RMA ID)**	**Title**	**Subjects**	**Type of Exposure**	**Level of Exposure (fibre/mL)**	**Duration of Exposure (year)**	**Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews**
			examination (10 abnormal)				first year for each of the three defined cumulative exposure groups:<20 fibre/mL-year (RR=1.227; 95%CI: 1.079, 1.396). 20-54.5 fibre/mL-year (RR=1.289; 95%CI: 1.115, 1.492) ≥55 fibre/mL-year (RR=1.387; 95%CI: 1.20, 1.603) (significance was not reported). Even with short periods of work, 5% of the workforce were exposed to at least 100 fibre/mL-year and 11% of the workforce to at least 50 fibre/mL-year. For 74 men there was 10 agreed "cases" in the current radiographs total estimated exposure ranged from 0.5-120 fibre-mL-year, with 3 men having total exposures <5 fibre-mL-year. Author's conclusion: study relates only to the finding of radiographic changes of small irregular or small rounded opacities (ILO classification of the pneumoconiosis). These changes are not diagnostic of radiographic asbestosis and not necessarily indicative of clinical asbestosis in the participants. The results clearly show a prevalence of uncompensated radiographic abnormality consistent with pneumoconiosis in at least 16% of former Wittenoom workers. Data are consistent with there being no threshold dose of crocidolite exposure for the development of radiographic abnormality.

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
Cookson et al (1986)[13]	RMA ID: 000445	The Natural History of Asbestosis in Former Crocidolite Workers of Wittenoom Gorge	136 mine and mill workers (100% men) (R1: radiographic evidence of pneumoconiosis) 139 subjects (R2: radiographic evidence of pneumoconiosis)	Crocidolite mine/mill	(R1) Onset of asbestosis 2-34 years after starting work (median=14 years) Cumulative exposure=91 fibre/mL-year (R2) Onset of asbestosis 1-33 years after starting work (median=13 years) Cumulative exposure: Median=77 fibre/mL-year	(R1) duration of exposure: median=3 years approx. (37 months) (R2) duration of exposure: median = 2.75 years approx. (33 months)	Morbidity study reporting asbestos exposure producing pneumoconiosis. In 136 men had a median exposure duration of 37 months, radiographic asbestosis appeared between 1 and 34 years after initial exposure and then progressed continuously. Onset of asbestosis was most frequent between 10 and 20 year from first exposure, No increased RR of progression for cumulative exposure of 0-54 fibre/mL-year. Increase RR of progression was seen for cumulative exposure of 55-148 fibre/mL-year (ILO category 1 to 2: RR=1.6; 95%CI: 1.1, 2.3; category 2 to 3: RR=2.7, 95%CI: 0.9, 8.2) and cumulative exposure of >148 fibre/mL-year (category 1 to 2: RR=2.5; 95%CI: 1.2, 5.4; category 2 to 3: RR=7.1, 95%CI: 0.8, 66.5) *p* values were not provided. Both onset of asbestosis and progression from Category 1 to Category 2 continued to occur throughout the follow-up period in workers exposed to crocidolite. No evidence of an effect of cumulative exposure to crocidolite on the time to onset of pneumoconiosis. However, progression of pneumoconiosis there was some evidence of effect of cumulative exposure. Authors conclusion: rate of radiographic progression of established asbestosis increases with the accumulated exposure to crocidolite and decreases with time from initial crocidolite

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
							exposure to the onset of definite radiographic abnormality
Irwig et al (1979)[14,15]	Cited by Becklake (1991)[16] RMA ID: 000429	Risk of Asbestosis in Crocidolite and Amosite Mines in South Africa. (And an erratum)	1692 mine and mill workers (100% men) 76=pleural thickening 123 =small opacities (profusion of ≥1/0)	Crocidolite and Amosite mines/mills	Concentration exposure ≤20 fibre/mL *n*=668 20.01-50 fibre/mL *n*=521 >50 fibre/mL *n*=274	Duration of exposure: *N*=1692 ≤ 1 year: *n*=569 1.01-3 year: *n*=348 3.01-7 years: *n*=348 7.01-15 years: *n*=281 >15 years: *n*=146	Morbidity study reporting asbestos exposure producing irregular opacities and pleural thickening. Prevalence small irregular opacities = 7.3%; and any pleural changes = 7.6%. Prevalence of irregular opacities increased from 2.3% in men exposure for ≤1 year to 26.7% in men >15 years of exposure. Authors conclusion: prevalence of pleural abnormality (pleural thickening, calcification, or obliteration of the costophrenic angle) were related to both age and the duration of asbestos exposure. Pleural and parenchymal abnormalities significantly predicted by fibre concentration, after adjusting for age and duration of exposure
Mortality Studies							
Deng et al (2012)[17]	RMA ID: 067596	Exposure-Response Relationship Between Chrysotile Exposure and Mortality from Lung Cancer and Asbestosis	586 Chinese textile factory workers 226= deaths 37=asbestosis deaths	Asbestos chrysotile textile factory	Cumulative exposure: *N*=586; *M*=126.1 (*SD* =181.1) fibre/mL-year Concentration of exposure: *M*=13.8 (*SD*=17.3) fibre/mL	Duration of exposure: *N*=586; *M*=25.4 (*SD*=8.3) years	Mortality study reporting asbestos exposure producing asbestosis. 37=asbestosis deaths 2.39 per 1000 person-years. Cumulative asbestos exposure was significantly associated with mortality from asbestosis in all models (*p*<0.001), except for the additive relative risk model. High estimated cumulative exposures were due to high dust/fibre concentrations in the workplace and the longer duration of employment. Estimated risk of asbestosis mortality increased with

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
							asbestos cumulative exposure in a convex curve, which tended to be steeper at a low exposure level (<50 fibre/mL-year) than a higher exposure level (>50 fibre/mL-year). Risk of asbestosis mortality rose more sharply than lung cancer mortality at higher exposure levels (>150 fibre/mL-year). Author's conclusion: study confirmed strong associations between exposure to chrysotile asbestos and lung cancer and asbestosis, in which clear exposure-response relationships were observed.
Moolgavkar et al (2010)[18]	Cited by Antao et al (2012)[19] RMA ID 068460	Potency Factors for Risk Assessment at Libby, Montana	1662 Libby vermiculite workers (100% men) 117=non-malignant respiratory disease	Vermiculite mine/mill	Cumulative exposure: *N*=1662 *M*=91.4 fibre/mL-year; Worked <1 year: *M*=8.5 fibre/mL-year Worked ≥1 year: *M*=178 fibre/mL-year 117 non-malignant respiratory disease cumulative exposure: *M*=270 fibre/mL-year; Worked <1 year: *M*=16.1 fibre/mL-year Worked ≥1 year:	Employment duration: *N*=1662 *M*= 3.6 years; Worked <1 year(*n*=850): 0.27 years. Worked ≥1 year (*n*=812): *M*=7.1 years 117 non-malignant respiratory disease employment duration: 91.4 *M*= 5.9 years; Worked <1 year (*n*=48): 0.30 years Worked ≥1 year (*n*=69): *M*=9.8 years	Mortality study reporting asbestos exposure producing non-malignant respiratory disease. Risk for non-malignant respiratory disease cumulative exposure of 100 fibre/mL-year: RR=1.14 (95% CI: 1.09, 1.18). Non-malignant respiratory disease (*n*=117): SMR=2.29 (95% Cl: 1.89, 2.74). Non-malignant respiratory disease for those worked ≥1 year (*n*=69): SMR=2.62 (95% Cl: 2.04, 3.31). Author's conclusion: SMRs for non-malignant respiratory disease are significantly elevated in the entire cohort and also in the sub-cohort of workers employed for ≥ 1 year. Detailed comparison by exposure categories was not possible for non-malignant respiratory disease.

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					M=447 fibre/mL-year		
Agency for Toxic Substances and Disease Registry (ATSDR) (2002)[20]	Cited by US Department of Health & Human Services, ATSDR & Disease Registry Division of Health Assessment & Consultation (2008)[21] RMA ID:067997	Health consultation: mortality in Libby, Montana (1979-1998)	542 subjects 12=asbestosis deaths (1=household contact, 11=former mine employees)	Libby, Montana community	Not reported	Duration of exposure: Former employees died from asbestosis: *n*=11; *M*=24.5 (*SD*=10.7; range=3-38) years	Mortality due to asbestosis was significantly elevated over the 20-year period. All asbestosis deaths were associated with the mining and milling facility through either previous employment or as a household contact of a former worker. Author's conclusion: SMR were all statistically significant for asbestosis in all areas of analysis. A mortality for the Libby community revealed significantly elevated SMR for asbestosis (40 to 80 times higher than expected) and lung cancer (20% to 30% higher than expected)
Liddell et al (1997)[22]	RMA ID: 026465	The 1891-1920 Birth Cohort of Quebec Chrysotile Miners and Millers: Development from 1904 and Mortality to 1992	9780 workers (100% men) 8009=deaths 108= pneumoconiosis deaths	Chrysotile mine/mill and asbestos factory	Level of exposure: Exposure 300 million particles per cubic foot (mppcf) British Occupational Hygiene Society [23] (1968) suggested an asbestos air concentration of 1 mppcf = 3 fibre/ml (detected by phase contrast	Duration of employment: >1 month (0.1 years)	Mortality study reporting asbestos exposure producing pneumoconiosis. Pneumoconiosis death rates per 100 000 subject-years clearly associated with exposure at the asbestos mine/mill (23=deaths) and Thetford mine-company 3 (53=deaths). Men whose exposures were <300mpcf.y accounted for 30 pneumoconiosis deaths. Exposure <300 mpcf.y has been essentially mild, although there was a small risk of pneumoconiosis mortality. Author's conclusion: Higher exposures have, led to excesses, increasing with degree of exposure, of mortality from

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
					microscopy). Therefor the concentration of exposure approximately 300 mppcf = 900 fibre/mL		all causes, but such exposures, of at least 300 mpcf.y.
Albin et al (1994)[24]	RMA ID: 034864	Retention patterns of asbestos fibres in lung tissue among asbestos cement workers	165 Lung tissue analysis (69 Swedish asbestos-cement workers and 96 controls) 2=pulmonary fibrosis deaths	Asbestos-cement chrysotile, tremolite, and crocidolite	Concentration of exposure: M=1.7 (median=1.2, range=00-7-3) fibre/mL. For workers performing milling, mixing, sawing, and polishing operations, or sweeping asbestos barn M=>2 fibre/mL	Duration of employment: M=18 (median=15) years	Mortality study reporting asbestos exposure producing pulmonary fibrosis. Chrysotile relatively rapid removal in lungs, whereas amphiboles (tremolite and crocidolite) displayed a slower removal patterns. Author's conclusion: Chrysotile retention may be dependent on dose rate, chrysotile and crocidolite retention may be increased by smoking, and chrysotile and tremolite retention may be enhanced by the presence of lung fibrosis.
Amandus et al (1987)[25]	Antao et al [26]						Study reporting asbestos exposure producing non-malignant respiratory disease. Non-malignant respiratory disease (n=20): SMR=2.43 (95% CI: 1.48, 3.75; p<0.05). Non-malignant respiratory disease: <50 fibre/mL-year (n=8; SMR=2.20; p<0.05). 50-99 fibre/mL-year (n=2; SMR=1.70; NS)100-399 fibre/mL-year (n=3; SMR=1.79;NS) >399 fibre/mL-year (n=7; SMR=2.20; p<0.01). Author's conclusion: an association between cumulative fibre exposure and non-malignant respiratory disease mortality

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
							was not found, and conclusions as to the exposure-response association are unclear. For >20 years latency, an inverse relationship is suggested. There were also too few deaths due to asbestos-related respiratory disease, 10 of 20 non-malignant respiratory disease.
McDonald et al (1986)[27]	Cited by: Antao et al [28] RMA ID: 068460	Cohort Study of Mortality of Vermiculite Miners Exposed to Tremolite	406 Libby vermiculite workers employed (100% men) 165=total deaths 21=non-malignant respiratory disease (8=pneumoconiosis)				Mortality study reporting asbestos exposure producing pneumoconiosis. Non-malignant respiratory disease: SMR=2.55 (95% CI not presented) for among mine workers employed for at least 1 year. Non-malignant respiratory disease associated with exposure: SMR=3.36 (10-19 years) and SMR=5.30 (>20 years) since first employment. Pneumoconiosis: linear increase in relative risk is estimated at 0.3% per fibre year (95% CI: 0.0, 4.1; NS). Author's conclusion: although positive relations with cumulative exposure were observed with pneumoconiosis numbers were small, confidence intervals wide, and conventional levels of statistical significance were not reached.
Review/Reports							
Antao et al[29]	RMA ID: 068460	Libby Vermiculite Exposure & Risk of Developing Asbestos	Not reported	Vermiculite mine/mill	Not reported	Not reported	Review**.** Author's conclusion: excessive mortality attributed to non-malignant respiratory disease among sub-cohorts of Libby mine and mill workers. Association of asbestosis

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
		Related Lung & Pleural Diseases					mortality with levels of cumulative exposure and duration of exposure
Mossman et al (2011)[30]	RMA ID: 067547	Pulmonary Endpoints (Lung Carcinomas and Asbestosis) Following Inhalation Exposure to Asbestos	Not reported	Not reported	Not reported	Not reported	Review. Authors conclusion: Fibre toxicity studies suggests that human exposure to respirable fibre that are bio persistent in the lung may induce significant and persistent pulmonary inflammation, cell proliferation, or fibrosis, and inhalation exposure to such fibre needs to be viewed with concern. Despite the fact that most chrysotile fibre are cleared rapidly, some small proportion of these fibre remains in the lung, and all of the asbestos types were noted in animal models and in epidemiological studies to induce asbestosis and lung cancer, especially in smokers.
Roggli et al (2010)[31]	RMA ID: 067548	Pathology of Asbestosis - An Update of the Diagnostic Criteria.	Not reported	Not reported	Not reported	Not reported	Report Authors conclusion: Probability of exposure is evidence of direct exposure to asbestos ≥5.0 fibre/mL for >1 year is enough for "ascertainment" of asbestosis (i.e. >5.0 fibre/mL-year). Estimates of cumulative asbestos exposures required for induction of asbestosis have diminished during the years. The threshold cumulative dose of asbestos necessary for clinical manifestations of asbestosis 20-200 fibre/mL-year with a few cases of 2-20 fibre/mL-year.
US Department of Health and	RMA ID: 067997	Summary Report.	28 sites - 3 groups who experienced	sites that exfoliated	Not reported	Not reported	Report. Author's conclusion: Fibre levels inside the exfoliation facilities

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
Human Services, ATSDR & Disease Registry Division of Health Assessment & Consultation (2008)[32]		Exposure to Asbestos-Containing Vermiculite From Libby, Montana, at 28 Processing Sites in the United States	the most significant exposure to asbestos former employees at exfoliation facilities, household contacts of these former employees, and some community members	Vermiculite from Libby:			ranged from below detection levels - 139 fibre/mL. Before 1980, measured fibre levels ranged from 1-10 fibre/mL, which is above the current acceptable level of 0.1 fibre/mL for occupational exposure to asbestos. Libby indicated airborne fibre levels were as high as 245 fibre/mL in an unloading area. Short-duration sampling results indicated airborne fibre levels of 3.9-23.3 fibre/mL, with a corresponding 8-hour TWA calculated as 5.7 fibre/mL. Significantly elevated SMR for asbestosis in the Libby Montana community were 40 to 80 times higher than expected
American Thoracic Society (2004)[33]	RMA ID: 067594	Diagnosis & Initial Management of Non-malignant Diseases Related to Asbestos	Not reported	Asbestos	Not reported	Not reported	Report. Author's conclusion: asbestosis is commonly associated with prolonged exposure, usually over 10 to 20 years. Short, intense exposures to asbestos, from several months to ≥1 year, can be sufficient to cause asbestosis.
Henderson et al (2004)[34]	RMA ID: 035222	The Diagnosis and Attribution of Asbestos Related diseases in an Australian Context.	Not reported	Not reported	Not reported	Not reported	Report. Author's conclusion: cumulative exposure of 25 fibre/mL-year delineated exposure of a character and magnitude sufficient to induce asbestosis. In some individuals. Asbestosis in chrysotile workers with cumulative exposure of >20 fibre/mL-year and in some cases 10-20 fibre/mL-year.

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
Agency for Toxic (2001)[35]	RMA ID: 035227	Toxicological Profile for Asbestos	Not reported	Not reported	Level of exposure: 10 fibre are typically in a cubic metre (fibre/m3) of rural outdoor air. There is 1 million cm3 (or 1 million mL) in a cubic metre so there would be 0.00001 fibre/mL of asbestos in rural air. Air in the city is 10 fold higher. Close to asbestos mines or factories levels reach 10,000 fibre/m3 i.e. 0.01 fibre/mL.	Not reported	Report. Author's conclusion: asbestosis at prolonged exposures of 5-20 fibre/mL corresponds to cumulative exposures of 50-200 fibre/mL-year for a 10 year exposure. Chronic exposure significantly increases asbestosis mortality rates in exposed workers with cumulative exposure of 32-1271 fibre/mL-year. Cases of asbestosis with very high asbestos air fibre and shorter latent periods (5-6 years) compared with latency of 10-20 years from studies of workers more recently exposed to lower fibre concentrations. Exposure durations (median 6-12 months) at 5-100 fibre/mL associated with pulmonary fibrosis.
Burdorf & Swuste (1999)[36]	Roggli et al (2010)[37] RMA ID: 067548	An Expert System for the Evaluation Of Historical Asbestos Exposure as Diagnostic Criterion in Asbestos Related Diseases	Not reported	Not reported	Not reported	Not reported	Historical Review. Author's conclusion: a life time risk of asbestosis of 2 per 1000 cases at 4.5 fibre/mL-year. Evidence of direct exposure to asbestos amounting to, or in excess of 5.0 fibre/mL for more than 1 year is enough for "ascertainment" of asbestosis. The 1st step in the decision tree distinguishes between jobs in which it is most likely blue collar worker is exposed and jobs where asbestos exposure is limited to those workers who handle asbestos.

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
							For the latter group proof of asbestos exposure is required at individual level. The 2nd and 3rd steps provide the qualitative cut-off points for the decision as to the attribution of asbestosis to occupational exposure. The cut-off point roughly reflects the minimum cumulative exposure of 5 fibre/mL-year to induce asbestosis.
Roggli (1998)[38]	RMA ID: 033719	Fiber analysis	Not reported	Not reported	Fibre per gram of wet or dry lung or cubic centimetre of lung i.e. 1 fibre/g wet lung = 1 fibre/cm3 =10 fibre/g dry lung	Not reported	Review. Asbestos body counts exceed 1700 per gram of wet lung in 90% in a series of 148 asbestosis cases. 59 insulators workers (59 asbestosis cases) median 20 400 bodies/gram of wet lung. 60 shipyard workers (19 asbestosis cases) median 3600 asbestos bodies/gram wet lung. 10 rail workers (1 asbestosis case) median 55 bodies/ gram of wet lung. 8 brake line workers (no cases) 50 bodies/gram wet lung. 24 other asbestos workers (6 asbestosis cases) 2360 bodies/gram wet lung. 16 household content workers (2 asbestosis cases) 410 bodies/gram of wet lung. 4 building occupants workers (no cases) 1.9 bodies/gram wet lung.
Rom (1998)[39]	RMA ID: 033719	Asbestos related disease	Not reported	Not reported	Number of asbestos bodies per gram in general population ~>500 but twice as many are found in the	Exposure duration depends on intensity	Report. Author's conclusion: people with pleural plaques have 10000 to 20000 asbestos bodies per gram of dry lung compared to people with asbestosis have >100 000 and often > 1 million. Short term high exposure in contained area – short exposures of 1

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
					lungs of blue collar workers		to 2 years would be sufficient to induce asbestos-related disease.
Anonymous (1997)[40]	RMA ID: 026517	Asbestos, Asbestosis, and Cancer: the Helsinki Criteria for Diagnosis and Attribution	Not reported	Not reported	For clinical purposes, the following guide-lines are recommended to identify persons with a high probability of exposure to asbestos dust at work: > 0.1 million amphibole fibres (>5 μm) per gram of dry lung tissue or over I million amphibole fibres (>1 μm) per gram of dry lung tissue; or over 1000 asbestos bodies per gram of dry tissue (100 asbestos bodies per gram of wet tissue) or over 1 asbestos body per mL of bronchoalveolar lavage fluid, as measured by light microscopy	Not reported	Report.Author's conclusion: asbestosis generally associated high exposure levels with radiological signs of parenchymal fibrosis. Mild fibrosis may occur at lower exposure levels, and detectable parenchymal fibrosis may not been seen on X-rays. HRCT can confirm radiological findings of asbestosis and show early changes not seen on chest X-rays. Cumulative exposure, on a probability basis, should be considered the main criterion for the attribution of a substantial contribution by asbestos to lung cancer risk. RR is roughly doubled for cohorts exposed to asbestos fibres at a cumulative exposure of 25 fibre-years or with an equivalent occupational history, at which level asbestosis may or may not be present or detectable. Heavy exposure, in the absence of radiological diagnosed asbestosis, is sufficient to increase the risk of lung cancer. Cumulative exposures <25 fibre-years are also associated with an increased risk of lung cancer, but to a less extent. The relative risk of lung cancer is estimated to increase 0.5-4% for each fibre per cc per year (fibre-years) of cumulative exposure. With the use of the upper boundary of this range, a cumulative expo-sure of 25 fibre-years is estimated to increase the

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
							risk of lung cancer 2-fold. Clinical cases of asbestosis may occur at comparable cumulative exposures.
Becklake & Case (1994)[41]	RMA ID: 000430	Fiber Burden And Asbestos-Related Lung Disease: Determinants of Dose Response Relationships	Not reported	Mine/mill workers, insulators, & tradesmen	Not reported	Not reported	Editorial. Author's conclusion: There are major differences in the relationship of fibre concentrations and disease for chrysotile and tremolite, and amosite and crocidolite. Heaviest burdens for asbestosis and airway fibrosis with lower burdens for pleural plaques and lung cancer. Differences between fibre types and doses responses.
Churg (1994)[42]	Mossman et al (2011)[43] RMA ID: 067547	Deposition and Clearance of Chrysotile Asbestos	Lung tissue analysis of 94 chrysotile asbestos miners and millers from Thetford –Quebec Canada	Chrysotile asbestos mill/mine	Not reported	Not reported	Review. Author's conclusion: The retained chrysotile and exposure atmosphere contained small percentage of tremolite, yet the lungs contained more tremolite than chrysotile, and the tremolite content increased rapidly with the duration of exposure. Most inhaled chrysotile was rapidity cleared by the lungs a small fraction was retained indefinitely. After exposure ended there was little or no clearance of either fibres from the lung.
Becklake (1991)[44]	RMA ID: 000429	Asbestos and other fiber-related disease of the lungs and Pleura. Distribution & determinants	Not reported	Not reported	Not reported	Not reported	Review. Author conclusion: men exposure to low level of fibre/mL over a short duration (5 -20 years) had small irregular opacities and pleural changes

Original Study Author (RMA ID) *or* Review of that Study	RMA ID *Or* Review Author (RMA ID)	Title	Subjects	Type of Exposure	Level of Exposure (fibre/mL)	Duration of Exposure (year)	Outcome: Study/Review with asbestos exposure (but with no exposure data that could be compared to the levels in the SoPs), producing Asbestosis; then Other Studies & Reviews
		in exposed populations.					
Sluis-Cremer (1991)[45]	RMA ID: 000440	Asbestos Disease at Low Exposures After Long Residence Times	Not reported	Not reported	A risk of asbestosis at cumulative exposure of 2-5 fibre/mL-year	Not reported	Review. Author conclusion: No difference in the frequency of disease incidence or rate of progression of irregular opacities between workers with discontinued asbestos exposure compared to workers with continued asbestos exposure

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Table A3. List of Medical Science Cited in the RMA Briefing Papers for Determining the Asbestos Factors in 1996, 2005, and 2013

RMA ID	Title
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033719	Rom WN. Chapter 24: Asbestos related diseases. In: Rom WN, editor. Environmental & Occupational Medicine. 3rd ed. Philadelphia: Lippincott-Raven; 1998. p. 349-75.
000430	Becklake MR, Case BW. Fiber burden and asbestos-related lung disease: Determinants of dose-response relationships. Am J Respir Crit Care Med. 1994;150(6 Pt 1):1488-92.
000431	Churg A, Vedal S. Fiber burden and patterns of asbestos-related disease in workers with heavy mixed amosite and chrysotile exposure. Am J Respir Crit Care Med. 1994;150(3):663-739.
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000435	Klaas VE. A diagnostic approach to asbestosis, utilizing clinical criteria, high resolution computed tomography, and gallium scanning. Am J Ind Med. 1993;23(5):801-9.
000434	Gaensler EA. Asbestos exposure in buildings. Clin Chest Med. 1992;13(2):231-42.
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026590	Fischer M, Günther S, Müller KM. Fibre-years, pulmonary asbestos burden and asbestosis. Int J Hyg Environ Health. 2002;205(3):245-48.
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APPENDIX B: THE CONSTITUTED COUNCIL AND LEGISLATIVE FRAMEWORK OF THE REVIEW

The Specialist Medical Review Council

109. The composition of each Review Council changes from review to review depending on the issues relevant to the particular Statement/s of Principles under review. When a review is undertaken three to five Councillors selected by the Convener constitute the Council.

110. The Minister must appoint one of the Councillors to be the Convener. If the Council does not include the Convener, the Convener must appoint one of the Councillors selected for the review to preside at all meetings as Presiding Councillor.

111. Professor Charles Guest, Convener of the Council, was the Presiding Councillor for this review. He is a public health physician with ACT Health and the Australian National University, and a Past-President of the Australasian Faculty of Public Health Medicine.

112. The other members of the Council were:

– Associate Professor Deborah Yates, Senior Staff Specialist in Thoracic Medicine, St Vincent's Hospital, Sydney, and conjoint Associate Professor, UNSW. Former alternate Chairperson of Medical Authority, Dust Diseases Board of NSW, and of the Central Pneumoconiosis Panel, London, UK. Leads a research program into occupational and obstructive lung diseases, including mesothelioma, asbestos-related disease and occupational asthma. Current member of the National Centre for Asbestos Related Diseases (NCARD).

– Professor John Wilson, Respiratory Physician and Head, Cystic Fibrosis Service, Department of Allergy, Immunology & Respiratory Medicine, Alfred Hospital, Melbourne. Professor, Faculty of Medicine, Nursing and Health Sciences, Monash University.

– Associate Professor Ian Glaspole, Visiting Medical Officer, Department of Allergy, Immunology and Respiratory Medicine, Alfred Hospital, Melbourne. Head of Interstitial Lung Disease Clinic. Co-chair, Australian Idiopathic Pulmonary Fibrosis Registry. Adjunct Clinical Associate Professor, Monash University, Central and Eastern Clinical School, Alfred Hospital. The Legislation

113. The legislative scheme for the making of Statements of Principles is set out in Parts XIA and XIB of the VEA. Statements of Principles operate as templates. They are determined by the RMA, and set out those criteria (conditions or exposures), known as factors, that must as a minimum exist before it can be said that an injury, disease or death can be connected with service, on either or both of the two statutory tests, the reasonable hypothesis test [293] and the balance of probabilities test.[294] Statements of Principles are ultimately applied by decision-makers in determining individual claims for benefits under the VEA and the Military Rehabilitation and Compensation Act 2004 (the MRCA).[295]

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[293] The reasonable hypothesis test is set out in section 196B(2) of the VEA which provides;

If the Authority is of the view that there is sound medical-scientific evidence that indicates that a particular kind of injury, disease or death can be related to:

(a) operational service rendered by veterans; or

(b) peacekeeping service rendered by members of Peacekeeping Forces; or

(c) hazardous service rendered by members of the Forces; or

(caa) British nuclear test defence service rendered by members of the Forces; or

(ca) warlike or non-warlike service rendered by members;

the Authority must determine a Statement of Principles in respect of that kind of injury, disease or death setting out:

(d) the factors that must as a minimum exist; and

(e) which of those factors must be related to service rendered by a person;

before it can be said that a reasonable hypothesis has been raised connecting an injury, disease or death of that kind with the circumstances of that service.

[294] The balance of probabilities test is set out in section 196B(3) of the VEA which provides:

If the Authority is of the view that on the sound medical-scientific evidence available it is more probable than not that a particular kind of injury, disease or death can be related to:

(a) eligible war service (other than operational service) rendered by veterans; or

(b) defence service (other than hazardous service and British nuclear test defence service) rendered by members of the Forces; or

(ba) peacetime service rendered by members;

the Authority must determine a Statement of Principles in respect of that kind of injury, disease or death setting out:

(c) the factors that must exist; and

(d) which of those factors must be related to service rendered by a person;

before it can be said that, on the balance of probabilities, an injury, disease or death of that kind is connected with the circumstances of that service.

[295] See sections 120, 120A and 120B of the VEA and sections 335, 338 and 339 of the MRCA.

APPENDIX C: WRITTEN AND ORAL SUBMISSIONS

Applicant's Submissions

114. The Applicant, made written and oral submissions. In them he raised concerns with both the duration of exposure required in the SoPs, and the means of exposure. He contended that the medical consensus that conditions such as asbestosis are usually associated with heavy long term exposure appears to be shifting to the view that low levels of asbestos fibres in the air can also be dangerous.

115. The Applicant also contended that the American Thoracic Society has proposed that the presence of asbestos bodies or pleural plaques can be used as an "…alternative to duration of exposure in attributing non-malignant diseases to asbestos exposure".

116. The Applicant regarded the American Thoracic Society criteria and the parties that developed the Helsinki criteria as expert bodies.[296] He contended that the current requirement in the SoPs for exposure to be experienced, "at the time material containing respirable asbestos fibres was being applied, removed, dislodged, cut or drilled…", is not supported by the findings of these expert bodies.

117. The Applicant considered that the Greenberg article also supported his contentions that low exposures to asbestos can potentially create disease.

118. The Applicant proposed that what he referred to as the '…long exposure requirements and the heavy exposure requirement' in the current SoPs could be amended in accordance with the 2004 American Thoracic Society criteria.

Commissions Submission

119. The Commissions' representative made written and oral submissions. Essentially the Commissions contended that pleural plaques and asbestos bodies are markers of exposure at some time to asbestos, but not to how much asbestos exposure is required to develop asbestosis or pulmonary fibrosis.

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296 American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med. 2004;170(6):691-715. (RMA ID: 067594).

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Greenberg M. The British approach to asbestos standard setting: 1898-2000. Am J Indust Med. 2004;46(5):534-41. (RMA ID: 034865).

120. The Commissions contended that there was sound medical scientific evidence[297] from studies of people who had worked in asbestos handling industries, asbestos product manufacture and miners, shipbuilders that related to long term occupational exposure generally measured in years.

121. The Commissions also noted the evidence in non-occupationally exposed persons such as wives of shipbuilders or from people who lived in proximity to asbestos industrial plants, with quite low level exposures.

122. The Commissions contended there was other evidence where authors advocated exposures between 25 fibre/mL/years as a bench mark level sufficient to induce fibrosis, but that other evidence supported lower levels of around 5 fibre/mL-years, although there was also evidence that it was possible to have quite a high exposure and not get asbestosis.

123. In the Commissions' view the evidence in respect of Merchant Mariners (Selikoff et al[298]) relate to the sort of low exposures likely for Australian NAVY veterans, but asbestos exposure may have occurred in the other services for example related to mechanical situations such as with asbestos brake linings.

124. In conclusion the Commissions submitted that lower minimum exposure durations than contained in the existing SoP factors were not warranted by the available evidence. The Commissions re-affirmed their view that the existence of either pleural plaques or asbestos bodies were markers of asbestos exposure, but did not reduce the minimum amount of asbestos exposure necessary to cause asbestosis.

The Commissions' view was that amendments to factors in the SoP to lower minimum exposure durations were not warranted on the available evidence.

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297 The medical science articles cited by the Commissions - see **Appendix C**

298 Selikoff et al. Asbestotic radiological abnormalities among United States merchant marine seamen. British Journal of Industrial Medicine. 1990;47(5):292-297. (RMA ID: 035792).

APPENDIX D: INFORMATION BEFORE THE COUNCIL

The Available Information, sent to the SMRC by the RMA under section 196K.

Australian Government
Repatriation Medical Authority

ASBESTOSIS

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Appendix A2

Information received in relation to

investigation 048-02 concerning asbestosis as at 7 August 2013

1. Submissions

1.1 Repatriation Commission, 15 November 1995 'Asbestosis' (submission for consideration of the Authority prior to the initial determination of SOPs concerning asbestosis Numbers 11 and 12 of 1996, including draft SOPs and reference list) (1385760R);

1.2 [redacted] (1385759R);

1.3 RMA medical researcher letter, 26 August 1996 (1385758R);

1.4 [redacted] email, [redacted] (1385767R);

1.5 [redacted] (1385773R);

1.6 [redacted] (1385769R);

1.7 [redacted] (1385768R);

1.8 RMA medical researcher discussion paper 'Asbestosis', 6 February 2003 (143755R);

1.9 [redacted] (request for investigation) (143753R);

1.10 [redacted] (143753R);

1.11 [redacted] (143752R);

1.12 [redacted] (143754R)

1.13 RMA medical researcher letter, 16 May 2005 (13103785R);

1.14 RMA medical researcher briefing paper 'Asbestosis', May 2005 (13103804R);

1.15 Department of Veterans' Affairs (Decision Support Unit), 25 May 2005 (13103791R);

1.16 Department of Veterans' Affairs (Decision Support Unit), 21 June 2005 (13103790R);

1.17 Department of Veterans' Affairs (Decision Support Unit), 6 July 2005 (13103789R);

1.18 [redacted] (1385762R);

1.19 [redacted] (1385761R);

1.20 [redacted] (1385552R);

1.21 [redacted] (141487R);

1.22 [redacted] (141688R);

1.23 [redacted] (141486R).

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Appendix A1

Information received in relation to investigation 285-09

concerning fibrosing interstitial lung disease as at 7 August 2013

1. Submissions

1.24 [redacted] (request for investigation concerning crytogenic fibrosing alveolitis) (143709R);
1.25 Repatriation Commission, October 1997 (submission for consideration of the Authority prior to determination of SOPs concerning idiopathic fibrosing alveolitis, Numbers 15 and 16 of 1998, including reference list) (13114006R and 131140086R);
1.26 RMA medical researcher briefing paper, 19 January 1998 (143710R);
1.27 [redacted] (143713R);
1.28 [redacted] (143714R);
1.29 [redacted] (143712R);
1.30 [redacted] (143715R);
1.31 [redacted] (request for investigation) (143726R);
1.32 RMA medical researcher discussion paper 'Idiopathic fibrosing alveolitis and request for investigation regarding smoking', 24 June 2003 (143727R);
1.33 [redacted] (143721R);
1.34 [redacted] (143722R);
1.35 RMA medical researcher summary of email interactions with [redacted] (143723R);
1.36 RMA medical researcher summary of issues 'Fibrosing alveolitis', April 2009 (13105361R);
1.37 RMA medical researcher briefing paper 'Fibrosing alveolitis', Volumes 1-3 April 2009 (13105362R), (13105363R) & (13105364R);
1.38 RMA medical researcher additional briefing paper 'Fibrosing alveolitis', April 2009 (13105360R);
1.39 [redacted] (request for investigation) (143724R);
1.40 [redacted] (143725R);

1.41 RMA medical researcher summary of issues 'Fibrosing interstitial lung disease – focussed review concerning smoking and paraquat', June 2010 (13105412R);

1.42 RMA medical researcher briefing paper 'Fibrosing interstitial lung disease – focussed review concerning smoking and paraquat', June 2010 (13105413R);

1.43 RMA medical researcher discusion paper 'Diagnostic radiation', August 2010 (1396573R);

1.44 RMA medical researcher summary of issues 'Fibrosing interstitial lung disease and ionising radiation', April 2011 (13105447R);

1.45 RMA medical researcher briefing paper 'Fibrosing interstitial lung disease and ionising radiation', April 2011 (13105448R);

1.46 [redacted] (request for investigation) (143716R);

1.47 [redacted] (143711R);

1.48 [redacted] (request for investigation) (143718R);

1.49 [redacted] (143720R);

1.50 [redacted] (request for investigation) (143717R);

1.51 [redacted] (1385552R);

1.52 [redacted] (141487R);

1.53 [redacted] (141688R);

1.54 RMA medical researcher summary of issues 'Fibrosing interstitial lung disease', August 2012 (13105469R);

1.55 RMA medical researcher briefing paper 'Gastro-oesophageal reflux disease and smoking, and fibrosing interstitial lung disease', August 2012 (13105471R);

1.56 RMA medical researcher summary of studies table 'Fibrosing interstitial lung disease and smoking', August 2012 (13105470R);

1.57 [redacted] (141486R).