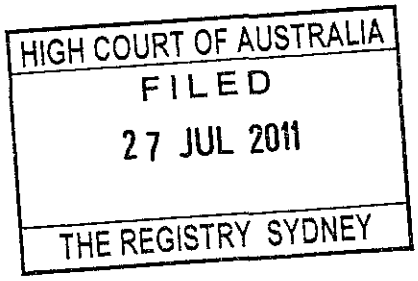
IN THE HIGH COURT OF AUSTRALIA
SYDNEY REGISTRY

No. S219 of 2011

BETWEEN:

AMACA PTY LMITED (ACN 000 035 512)
(UNDER NSW ADMINISTERED WINDING UP)
Appellant



and

JOHN WILLIAM BOOTH
First Respondent

AMABA PTY LIMITED (ACN 000 387 342)
(UNDER NSW ADMINISTERED WINDING UP)
Second Respondent

FIRST RESPONDENT'S SUBMISSIONS

Part I: Internet Publication

1. The first respondent ("Mr. Booth") certifies that this submission is in a form suitable for publication on the internet.

Part II: Statement of Issues

2. The only issue in this appeal is whether there was any evidence from which it was open to the trial judge to infer that Mr. Booth's mesothelioma was caused by exposure to asbestos fibres from brake linings manufactured by the appellant ("Amaca").

3. Issues as to the admissibility, weight or expert basis of the medical opinion evidence do not arise having been specifically excluded by the grant of leave and due to the limited nature of the statutory right of appeal from the Dust Diseases Tribunal of New South Wales ("DDT") to the New South Wales Court of Appeal under s.32 of the *Dust Diseases Tribunal Ac 1989* (NSW). Further Mr Booth refers to and adopts paragraph [3] of his Submissions in S220 of 2011.

Part III: Section 78B of the *Judiciary Act 1903* (Cth)

4. No notice is required under section 78B of the *Judiciary Act 1903* (Cth).

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Part IV: Statement of Material Facts

5. A number of critical facts were not in dispute. First, that Mr. Booth's mesothelioma was caused by the inhalation of asbestos fibre. Secondly, that chrysotile asbestos has the capacity to cause mesothelioma. Thirdly, that the brake linings manufactured by Amaca (and the Second Respondent "Amaba") contained chrysotile asbestos, and fourthly, that Mr. Booth inhaled chrysotile asbestos fibres liberated from products manufactured by Amaca (and Amaba).[1]

6. Mr. Booth accepts Amaca's description of the relevant factual background in the appellant's submissions ["AS"] at AS [7]-[12], with the following qualifications and additions:

   (a) The trial judge found that Mr. Booth's exposure to asbestos from sources other than the brake linings was "*trivial*"[2] and that Mr. Booth's work on brake linings was "*a very dusty process*"[3]. The processes which liberated asbestos dust in his breathing environment were the use of a hammer to punch rivets through the brake linings, an electric drill to remount holes for the rivets, a bench grinder to grind the leading edges of the brake linings and the use of compressed air to blow the dust from his work clothes, work bench and the floor of the workshops in which he worked.[4] Further, he was exposed to asbestos from the work of others.[5]

   (b) The trial judge found that "*mesothelioma very rarely occurs in persons who have not been exposed to asbestos fibres beyond the background level that pervades urban environments*"[6].

   (c) There is an accepted or proven dose/response relationship between the inhalation of asbestos and the number of cases of mesothelioma observed across populations.[7]

7. In so far as AS [13] purports to set out the trial judge's specific finding as to the causes of Mr. Booth's mesothelioma, it is incorrect. The passage reproduced by Amaca was his Honour's determination of an issue of a general nature for the purposes of s25B of the *Dust Diseases Tribunal Act, 1989* (NSW). This is highlighted by the trial judge's reference at TJ [60] to the "*cumulative effect theory*" having been accepted by the New South Wales Court of Appeal in *EM Baldwin & Son Pty Ltd v Plane* (1998) 17 NSWCCR 434.

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[1] TJ [22]
[2] TJ [162(4)]
[3] TJ [19]
[4] TJ [10]-[18]
[5] TJ [11], [12], [13], [15], [17], [18]
[6] TJ [162 (2)]
[7] See Professor Henderson at CA Blue 1, 67

8. The trial judge's specific findings in relation to the cause of Mr. Booth's mesothelioma were made in three cumulative stages. First, at TJ [59] his Honour said: "*At issue between the parties in this case is the proposition that all exposure to chrysotile asbestos, other than trivial or de minimis exposure, that occurred in a latency period of between 26 and 56 years, materially contributed to the cause of Mr. Booth's mesothelioma. I resolve that issue in favour of the plaintiff*". Secondly, having considered the issue of the materiality of Mr. Booth's total exposure to asbestos fibres from brake linings over 27 years, the trial judge found (at TJ [161]) that "*...the plaintiff's exposure to the asbestos contained within brake linings materially contributed to the causes of his mesothelioma*". Thirdly, after considering the materiality of Mr. Booth's proportionate exposure to asbestos fibres from each of Amaca's (10 years) and Amaba's (17 years) brake linings, his Honour held (at TJ [172]): "*I find that asbestos dust liberated from brake linings manufactured by each of the defendants Amaca and Amaba materially contributed to Mr. Booth's contraction of mesothelioma*".

9. The cumulative stages by which the trial judge determined causation is important because it reflects his acceptance of the "*almost universally accepted*"[8] opinions of Mr. Booth's medical experts that mesothelioma is caused by the total cumulative effect of all fibres inhaled ("total fibre burden") within an acceptable latency period (the "cumulative effect explanation"). Importantly, the trial judge rejected[9] Amaca's apparent reliance upon the "*single fibre theory*" and the "*threshold theory*", which holds that mesothelioma may be caused by one asbestos fibre or one particular episode of asbestos exposure to the exclusion of all others.[10]

10. Another important feature of this case is that unlike lung cancer, "*Mesothelioma is a signature malignancy for asbestos exposure*"[11], the experts describing asbestos inhalation as the "*principal*"[12], "*overwhelming*"[13], "*effectively the only known*"[14] and the "*accepted*"[15] cause of mesothelioma. Because asbestos is *the* proven cause of mesothelioma, this was not a case where Mr. Booth relied exclusively on epidemiological studies to support an inference of causation. He called medical evidence (including evidence on the biological mechanisms in the pathogenesis of mesothelioma) from four doctors. Amaca (and Amaba) called no medical evidence and attempted (unsuccessfully) to use controversial epidemiological studies to prove that Mr. Booth sustained no increase in risk of mesothelioma from his work with brake linings.[16]

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[8] CA [51], quoting Professor Henderson's evidence
[9] TJ [48]-[50]
[10] TJ [60]; TJ [55]-[57]
[11] CA Blue 1, 322 O; and Professor Henderson at CA Black 1, 122L-M
[12] Professor Berry (Amaca's expert epidemiologist and biostatistician) CA Black 2, 551M
[13] Professor Berry at CA Black 2, 551N
[14] Professor Henderson at CA Black 1, 122L-M
[15] Professor Musk at CA Black 1, 450P
[16] Professor Berry did not accept the conclusion of those studies: CA Black 2, 544S-U, 545C-I

Part V: Legislation

11. Mr. Booth accepts Amaca's statement of the relevant statutory provision.

Part VI: First Respondent's Argument

12. Neither contention at AS [14] is substantiated. On the largely uncontroverted medical evidence of four doctors, each of whom opined that Mr. Booth's mesothelioma was causally contributed to by his exposure to asbestos from brake linings manufactured by each of Amaca and Amaba, it was clearly open to the trial judge to draw an inference that each exposure to asbestos, other than trivial exposure, within an accepted latency period, materially contributed to Mr. Booth's mesothelioma. *A fortiori* where Amaca and Amaba failed to call any medical evidence to challenge or cast doubt on the opinions of Mr. Booth's eminently qualified and internationally recognised medical experts. Further, the Court of Appeal did *not "fail to carry out its function...to review the entirety of the evidence"*.[17] On the contrary, in a unanimous decision it considered the abundant evidence and exercised its own evaluative judgment as to whether the evidence was capable of supporting the trial judge's findings of fact, in the sense that they were "*reasonably open*...[and] ...*logically available*".[18]

13. In this appeal Amaca seeks to negate its failure to call any medical evidence on causation by relying upon "*factual findings made in other jurisdictions*"[19] and by implicitly asserting that the medical opinions of Mr. Booth's doctors (including the "*internationally recognised expert on pleural tumours and mesothelioma*"[20] Professor Henderson) were in some unexplained way outside "... *the same body of international learning*".[21] In this regard, it is important to highlight that it was never suggested to Professor Henderson or any other doctor that their conclusions about the probable cumulative effect explanation of the aetiology of mesothelioma was contrary to the published medical science. This is unsurprising given that the "*common body of international learning*"[22] has been significantly contributed by the many years of peer reviewed and published work of Professor Henderson and Dr Leigh.[23]

14. The statement extracted by Amaca at AS [16] from *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32, was based on evidence different from the medical evidence in the present case. In *Fairchild*, the House of Lords proceeded on the particular factual basis that mesothelioma "...*may be caused by a single fibre, or a few fibres, or many fibres: medical opinion holds none of these possibilities to be more probable than any other...so* ...[the claimant] *could have inhaled a single fibre giving rise to his condition during employment A, in which case his exposure by B will have*

[17] AS [14]
[18] CA [23]-[26]
[19] AS [15]
[20] CA [60]
[21] AS [15]
[22] AS [18]
[23] See the extensive CV's of Professor Henderson at CA Blue 1, 105-158 and Dr Leigh at CA Blue 1, 357-393

no effect on his condition; or he could have inhaled fibres during his employment by B in which case his exposure by A will have had no effect on his condition; or he could have inhaled fibres during his employment with A and B which together gave rise to his condition; but medical science [on the evidence in that case] *cannot support the suggestion that any of these possibilities is to be regarded as more probable than any other.*"[24] This contest between the single fibre theory[25] and the cumulative effect explanation of the aetiology of mesothelioma was, on the evidence in Mr. Booth's case, concluded with the trial judge's finding that more probably than not the biological aetiology of mesothelioma was explained by the cumulative effect of all fibres inhaled (other than from trivial exposures) within the relevant latency period. The trial judge rejected the "single fibre theory"[26] and found that the experts' adherence to the cumulative effect explanation "*most probably* [accorded] *with the actual aetiology of the disease.*"[27]

15. In *Sienkiewicz v Grief (UK) Ltd* [2011] 2 WLR 523, the Supreme Court of the United Kingdom revisited causation of mesothelioma in a case that "*did not involve the introduction of detailed evidence of what is known today about mesothelioma, proceeding on the basis that findings in previous cases could be taken as read*".[28] Even so, Lord Phillips foreshadowed (in the absence of evidence in that case) that because "*the single fibre theory has ...been discredited...Causation may involve a cumulative effect with later exposure contributing to causation initiated by an earlier exposure.*"[29] He observed that in those circumstances, the "*conventional test of causation*" would apply: whether, on the balance of probabilities, the exposure in a particular case contributed to causing the disease?[30] For this reason, Lord Phillips suggested "*the possibility that mesothelioma may be caused as a result of the cumulative effect of exposure to asbestos dust provides a justification, even if it was not a reason, for restricting the Fairchild/Barker rule to cases, where the same agent, or an agent acting in the same way, has caused the disease, for this possibility will not exist in respect of rival* [or competing] *causes that do not act in the same causative way*".[31] This belies Amaca's assertion,[32] evidentially baseless, that the cumulative effect explanation of the cause of mesothelioma is a "*scientific principle not recognised anywhere else in the world*". Not only was the cumulative effect explanation implicitly recognised as a possible mechanism in *Fairchild*, but Lord Phillips explicitly referred to it in *Sienkiewicz*. As the trial judge observed[33] the New South Wales Court of

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[24] *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 per Lord Bingham of Cornhill at [7]
[25] In *Fairchild*, see also references to an inability to identify "*the day upon which he inhaled the fatal fibre*" per Lord Hoffman at [62]; "*...could be due to the action of a single fibre...*" per Lord Hutton at [78]; and per Lord Rodger to the same effect at [120]-[121]
[26] TJ [48]-[49]
[27] TJ [52]
[28] *Sienkiewicz v Grief (UK) Ltd* [2011] 2 WLR 523 per Lord Phillips at [18]
[29] *Sienkiewicz v Grief (UK) Ltd* [2011] 2 WLR 523 per Lord Phillips at [102]
[30] Ibid See also Lord Dyson at [208]
[31] Ibid per Lord Phillips at [104]. See statements to a similar effect by Lord Rodger at [142], per Lord Dyson at [213]
[32] AS [18]
[33] TJ [58]

Appeal endorsed the cumulative effect explanation in *Plane*. This Court refused special leave on this issue more than 10 years ago.[34]

16. Amaca's reference to the passage in Professor Stapleton's article[35] is of no assistance to it because it is apparent that she has assumed that the findings in *Fairchild* accord with the limits of medical knowledge, and further, that they were the only reasonably inferable conclusions on the medical evidence in that case. There is, with respect, simply no basis for this assumption. Further, Professor Stapleton's description of the evidence accepted in *Plane* as the "*every exposure contributes to the triggering of the cancerous process*" reveals a misunderstanding of the biological mechanisms underpinning the cumulative effect explanation. As Dr Leigh explained (see below) the fibres cumulatively initiate *and promote* mutations and DNA changes over many years with continued exposure contributing to the multistage development of the disease; it is not simply a question of "*triggering*".[36]

17. It is not readily apparent why the claimants in the United Kingdom cases did not (as Mr. Booth did) call evidence from internationally recognised medical authorities including Professor Henderson and Dr Leigh, as to the probable biological explanation of the cumulative effect of asbestos fibres in the pathogenesis of mesothelioma. And it is, with respect, extraordinary that in circumstances where it called no medical evidence Amaca now implicitly seeks to rely on findings in those cases. Amaca's contentions in this respect should be rejected.

18. Amaca's reference at AS [19] as to the "*staggering reach of this finding*" is misplaced. The passage extracted does not represent a finding on causation (see [8]-[9] above) but rather the trial judge's summary of the thrust of the opinions of Mr. Booth's medical experts. Amaca does not attempt to explain why or how medical opinions that "*all fibres contribute to the development of a mesothelioma*" may be characterised as "*staggering*".[37] This omission is particularly significant when the statistical evidence it did call (Professor Berry[38]) was entirely consistent with the opinions of Mr. Booth's medical experts as to the likely causes of his mesothelioma.

19. Importantly, in setting the parameters for its selective analysis of the medical evidence, Amaca has posed the wrong question.[39] The medical opinion evidence was most

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[34] *Jsekarb Pty Limited v Plane & Anor* S13/1999 [1999] HCA Trans 368, per Gaudron J

[35] AS [18]

[36] See also Professor Stapleton's rhetorical question "*are these mechanisms ones that require only a single fibre or do they need a threshold load of fibres?*" making no allowance for even the possibility of causation by the cumulative effect of the total fibre load: Stapleton, "Factual Causation and Asbestos Cancers", (2010) LQR 351 at 356.

[37] It could hardly be regarded as "surprising". As noted above, similar findings were made in *Plane* more than 10 years ago.

[38] Professor Berry agrees that it was "*...the lifetime load of all asbestos exposure which causes the illness in the individual*": CA Black 2, 552V-W, and he agreed that "*...medically it would be unsafe to proceed on the basis of one or the other, everything rather suggests because we cannot track the fibres being inspired and exhaled harmlessly, that it is the lifetime load.*": CA Blue 7, 3,082M-N; and that in respect of mesothelioma "*...when the risk comes home there is the cause.*": CA Black 2, 553E

[39] AS [20]

important but it was not the only evidence before the trial judge. The correct question is: was the medical evidence alone or in combination with other evidence and facts, capable of providing a basis for the trial judge's inference that Mr. Booth's mesothelioma was caused or materially contributed to by his 10 years of exposure to asbestos from Amaca's brake linings?

The Medical Evidence

20. For Amaca to establish error in point of law it must establish that there was no evidence on which the trial judge could infer that Mr. Booth's mesothelioma was caused or materially contributed to by his exposure to asbestos from Amaca's brake linings.[40]

21. In light of these principles, effectively Amaca must establish each of the following:

(a) it was not open to the trial judge to infer that the probable biological mechanisms by which mesothelioma develops is the cumulative effect explanation; and

(b) despite the fact that the medical experts expressed their opinions on causation using terms such as "*cause*", "*ultimate development of*", "*causal contribution*", "*significant causal contribution*", "*material contribution*", "*cause **and** risk*", "***proven** risk*" and "*risks came home*", they spoke *only* of increased risk[41]; and

(c) it was not open to the trial judge to infer that the medical experts were probably referring to "cause" and/or "cause ***and*** risk" when they expressed their opinions; and

(d) even if the medical experts spoke *only* of increased risk, it was not open to the trial judge to infer from the combination of this evidence with other uncontested facts (see above) that Mr. Booth's mesothelioma was caused or materially contributed to by his exposure to asbestos from brake linings manufactured by Amaca.

22. An analysis of the largely unchallenged medical evidence (below) leads to the conclusion that Amaca cannot substantiate any of the matters referred to in [21] above. The medical evidence alone and in combination with other evidence and facts provided an overwhelming basis (let alone *"some basis"*) for the trial judge's finding. Moreover, the trial judge applied conventional common law principles.

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[40] See *Australian Broadcasting Tribunal v Bond* (1990) 170 CLR 321 at 356 per Mason CJ (No error of law has occurred when there is "some basis" for the inference.)

[41] A submission that the Court of Appeal described as "*not made out factually*": CA [120]

Professor Henderson

23. Professor Henderson is a Professor of Pathology and an "*internationally recognised expert on pleural tumours and mesothelioma*"[42]. He explained that pathology was "*the study of disease processes...including their causes, mechanisms of development, characteristics once they have developed and their natural history.*"[43] At AS [22]-[37] Amaca sets out its selection of Professor Henderson's evidence and despite the unambiguous nature of the words by which Professor Henderson expressed his opinions, it repeatedly asserts that "*he was speaking of "risk" referable to a population of persons*".[44] In selectively analysing Professor Henderson's evidence Amaca's repeated use of phrases such "*proper understanding*"[45], "*in other words*"[46], "*so explained*"[47], "*in fact he means*"[48], "*was not saying*"[49], "*in that sense*"[50], "*did not intend*"[51], "*appears to consider*"[52], highlights its attempt to re-characterise Professor Henderson's opinions and reinforces (at the very least) that the trial judge's inferences of causation were very clearly open.

24. In his report of 2 March 2009[53] Professor Henderson took into account the controversial epidemiological studies (upon which Amaba and Amaca relied) and relying not only on epidemiological studies of risk[54] reached the conclusion that chrysotile fibres contained in brake linings had the capacity to induce mesothelioma because "*In terms of dose-response, epidemiological studies on non brake chrysotile exposures have demonstrated a dose-response relationship...the relationship in causal terms is supported by experimental studies, and also from the perspective of biological plausibility...*"[55] That his evidence went further than risk was made clear by Professor Henderson when he explained that "*...from surveying all of the evidence (not only the epidemiological evidence) and from first principles and from what is known about other chrysotile-only exposures, a causal-contributory relationship follows*".[56] Importantly, in Appendix B of this report Professor Henderson revealed that he was acutely aware of the distinction between "*Absolute associative causal effects* [which] *involve assessment of the actual numbers of cases or incidences*" and "*relative risks*" which "*involve assessment of ratios*" in exposed and unexposed groups: i.e. statistical

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[42] CA [60]. His extensive CV is at CA Blue 1, 105-157
[43] CA Black 1, 85
[44] AS [26]. The same submission is made at AS [24], [28], [29], [30], [31], [32], [33], [34], [36], [37]
[45] AS [23]
[46] AS [24], AS [36]
[47] AS [31]
[48] AS [34]
[49] AS [36]
[50] AS [36]; appearing twice
[51] AS [36]
[52] AS [32]
[53] CA Blue 1, 38
[54] Professor Henderson invoked the landmark "Bradford Hill Criteria" for Medical Causation, epidemiology is only one criterion: see CA Blue 1, 56U and Professor Henderson's oral testimony at CA Black 1, 111O
[55] Ibid
[56] CA Blue 1, 57J

or epidemiological risks.[57] He said that because "*biological systems such as human beings vary in multitudinous different ways*" "*it is quite inappropriate simply to extrapolate the mean RR/OR to each and every individual comprising the population*".[58] Professor Henderson's acceptance of biological variability (and generic susceptibility) and his stated understanding of the differences between causal effects and relative risks, militates strongly against Amaca's attempt to re-characterise his opinions on causation as relating solely to a nebulous statistical representation of risk rather than cause.

25. In a passage not referred to by Amaca, Professor Henderson expressed the following opinion about the causes of Mr. Booth's mesothelioma: "...[it is] *attributable to his* ***total cumulative exposure*** *to asbestos ...* ***within the context of that total cumulative exposure*** *it also remains my cautious opinion "on the balance of probabilities" that Mr. Booth's total cumulative exposure to chrysotile-tremolite dust derived from brake linings made a* ***significant causal contribution towards the development of his mesothelioma,*** *by way of a* ***significant proportional causal effect*** *superimposed upon any antecedent exposure (such as any alleged childhood exposure) and also incremental upon any underlying "background" risk of mesothelioma.*"[59] Professor Henderson's evidence in relation to causation went further. He opined that "*Given that* [Mr. Booth's] *total cumulative brake dust derived from chrysotile-tremolite exposure made a significant proportional causal contribution towards the development of his mesothelioma, it is also my opinion that the dust derived from the proportions set forth in paragraph 13 on page 14 (Amaca/Amaba brake materials) made* ***a significant causal contribution towards the development of his mesothelioma,*** *as a substantial fraction of his total brake dust derived chrysotile-tremolite exposure.*"[60] Professor Henderson also noted that his "*...consultation and referral files now include many cases of pleural malignant mesothelioma for whom chrysotile-tremolite only exposure derived from new brake linings was the only identified pattern of exposure*".[61]

26. As referred to at AS [22], in his evidence in chief, Professor Henderson agreed with the proposition that "*All asbestos exposure within an acceptable latency period causes or materially contributes to mesothelioma*". Importantly, he added: "*...****It is, I think, almost universally accepted that all asbestos exposures, both recalled and unrecalled, will contribute causally towards the ultimate development of a mesothelioma.*** *The proportional causal contributions being dependent upon the asbestos fibre types and the cumulative exposures from each of the identified exposures, and modified by years following the commencement of each of those exposures.*"[62] This answer has two parts. The first sentence accepts the cumulative effect explanation of the aetiology of mesothelioma; the second deals with the dose

[57] CA Blue 1, 83
[58] CA Blue 1, 84G-H
[59] CA Blue 1, 100L-O
[60] CA Blue 1, 100P-R
[61] CA Blue 1, 61F-G
[62] CA Black 1, 91U, 92O-Q

response model by which estimates of proportional causal contributions (such as those performed by Amaca's epidemiologist, Professor Berry) may be made. For this reason, Amaca's contention at AS [24] is misconceived. The next question put to Professor Henderson directed his attention to the dose response model and he explained it by reference to the *"no threshold dose response relationship"*.[63] In this answer, echoing the comments made in his report (see above) about the difference between "risk" and "causal effects", he explained that "*the risk is not a theoretical construct...*"[64] That this is so, was further elucidated by Professor Henderson's response: "*That is correct, your Honour*" to the trial judge's comment: "*So I understand it, if all exposure is contributory, the next question is to what extent.*"[65]

27. In further testimony in chief, Professor Henderson was asked to explain the basis of his opinion that *"when there are multiple asbestos exposures each contributes to cumulative exposure and to the risk and causation of mesothelioma..."*[66] By reference to the biological and cellular mechanisms of the cumulative effect explanation of the pathogenesis of mesothelioma, he answered: "... *When there are multiple episodes of asbestos exposures and the individual concerned inhales increasing numbers of fibres on different occasions, that contributes to the* ***total burden of asbestos fibres*** *deposited in the lung and translocated to the pleura and it is thought that mesothelioma develops because of an interaction between the asbestos fibres and the mesothelial cells by way of secondary chemical messages and to simplify the answer, the point is that the more fibres there are the greater number of fibres there will be interacting with the mesothelial cells which themselves undergo proliferation and so the process goes on with increasing numbers of mesothelial cells interacting with increasing numbers of fibres,* ***so that the ultimate development of mesothelioma and its probability of development*** *will be influenced by the numbers of fibres interacting with mesothelial cells over multiple periods of time and probably over multiple different generations of mesothelial cells and* ***I think this is a fairly well accepted model now and it flies in the face of what used to be called the one fibre hypothesis that mesothelioma came about from a single fibre interacting with a single mesothelial cell which in biological terms is a ridiculous proposition.***"[67] [Emphasis added] With this evidence, (which was not relevantly challenged) Professor Henderson conclusively dealt with the perceived uncertainty upon which *Fairchild* and *Sienkiewicz* proceeded.

28. Amaca's reliance upon Professor Henderson's use of the phrase "*it follows*" in the passage reproduced at AS [25] is misplaced. In this passage Professor Henderson made it clear that he was speaking of "*risk and causal contributions*" to the "*ultimate development of mesothelioma*". The word "population" as used in AS[26] does not appear in the evidence extracted by Amaca in AS[25].

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[63] CA Black 1, S-T.
[64] Ibid
[65] CA Black 1, 93E
[66] CA Black 1, 95W-Y
[67] CA Black 97C-J

29. Amaca's description of "Appendix B" to Professor Henderson's report at AS [29] is completely misconceived for the reasons set out at [24] above. Professor Henderson explained that his opinion on causation was based on the Bradford Hill criteria, experimental studies, and "*pathobiological principles*"[68] rather than concepts of risk across populations. Further, the evidence referred to at AS [30] does not support Amaca's contention that Professor Henderson spoke only in terms of increased risk. The point is, the cumulative effect explanation of the aetiology of mesothelioma is entirely consistent with and reinforced by the epidemiological studies that when a person develops mesothelioma the prospective risks occasioned by repeated exposure have each materialised. That does not mean that Professor Henderson as a pathologist referred to causation as the sum of epidemiological risks.

30. It was not put to Professor Henderson during cross examination that his understanding of the biological process underpinning the cumulative effect explanation was wrong or scientifically implausible or improbable. Instead, he was asked whether he claimed to have a complete understanding of the biological processes whereby inhaling asbestos causes mesothelioma[69], in response to which he readily conceded: "*Not only do I not understand it completely, neither does anybody else.*"[70] He explained that: "... *We don't know all the details in humans, but we have some pretty good ideas*"[71], "*...the science is always incomplete...*"[72] and "*There is some uncertainty about the science but I think that the evidence is fairly good. But all scientific evidence is incomplete and carries a degree of uncertainty.*"[73]

31. As noted at AS [31] Professor Henderson was cross examined about the issue of risk; but it was not suggested to him that his understanding of the biological mechanisms underpinning the cumulative effect explanation of the development of mesothelioma was in some way reduced to a mere expression of increased risk or inconsistent with it. The question and answer set out at AS [31] does not support Amaca's contentions for a number of reasons. First, the question did not suggest that an increase in risk was the *only* matter established by "*the science*". Secondly, in his answer, Professor Henderson (echoing the evidence referred to above about the difference between "*causal effects*" and "*risks*") eschewed a "*nebulous or theoretical construct...*" of the term, describing it as "*...a bad term but everybody uses it.*"[74] Thirdly, Professor Henderson's answer is prefaced with "*in this context*"; a reference to the assessment of proportional causal contributions as described in the evidence extracted at [26] above. From this evidence it is apparent that in mesothelioma cases there is no conflict between the biological understanding of the cumulative effect explanation and the increasing number of cases of mesothelioma caused by further exposure to asbestos. In

[68] CA Blue 1, 93N
[69] CA Black 1, 114S
[70] CA Black 1, 114T
[71] CA Black 1, 114V-Y, 116B-D
[72] CA Black 1, 115E
[73] CA Black 1, 115K
[74] CA Black 1, 115N

light of the totality of his evidence, the assertion that Professor Henderson's evidence amounted to a "*mere identification of increased risk*"[75] let alone that this was the only conclusion open to the trial judge should not be accepted.

32. The evidence set out at AS [33] was in relation to what counsel for Amaca (and Amaba) described as a "*new subject*"[76] in which a number of hypothetical propositions were put to Professor Henderson about whether each non brake lining exposure "*by itself*", if "*that was the only exposure above background*" would be sufficient to cause Mr. Booth's mesothelioma.[77] Professor Henderson's acquiescence that absent any other exposure, each exposure would have made a "*small causal contribution*" and that each exposure increased the risk, is of no assistance to Amaca because this hypothetical exercise says nothing about the actual cumulative biological causal process described earlier in Professor Henderson's evidence.

33. Amaca's contention AS [34] is illuminating. Not only is the contention entirely consistent with Professor Henderson's cumulative effect explanation of the cumulative biological aetiology of mesothelioma, but even if the trial judge was persuaded that in respect of each exposure Professor Henderson meant "*nothing more than some unidentified and identifiable part of the cumulative bundle of risks which the person faced in advance has eventuated*"[78] (i.e. "*come home*"), that provided a more than adequate basis at law for the trial judge's finding on causation. Given Professor Henderson's earlier evidence as to biological cumulative process of causation and his rejection of the single fibre theory, it is unsurprising that Professor Henderson was not prepared to say that Mr. Booth's mesothelioma was caused "*individually*" by the risk of one particular exposure as opposed to another. He reiterated that "*...the risk from all of his exposures came home because the model which I adopt is that of a* ***cumulative exposure dose response,*** *so I think that all of the asbestos fibres that he has inhaled, or at least a proportion of them contribute to the risk* ***and to the ultimate development of the mesothelioma.***"[79] The biological model Professor Henderson was referring to was the cumulative effect explanation which, in his opinion, was the most likely description of the actual aetiology of the disease.

34. The reference by Professor Henderson to "*at least a proportion of them*"[80] in the passage above is nothing more than an acknowledgment of the scientific fact that some fibres (of the millions of fibres inhaled during each exposure) may be cleared by the natural defence mechanisms of the lung at different times and different rates in the cumulative process (see also Dr Leigh's evidence below). This is consistent with the cumulative effect explanation and accords with the trial judge's finding that trivial or *de minimis* exposure had no causative effect. Allowing for the clearance of some

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[75] AS [32]
[76] CA Black 1, 117I
[77] See the exchanges at CA Black 1, 117I-T,X-Y, 118B-Y
[78] AS [34]
[79] CA Black 1, 119H-I
[80] AS [35]

fibres Professor Henderson did not resile from his opinion that Mr. Booth's inhalation of asbestos dust from the brake linings of Amaca and Amaba each made a significant causal contribution to the development of his mesothelioma. It was not suggested to Professor Henderson that because of clearance mechanisms the fibres emanating from Amaca's (and Amaba's) brake linings did not materially contribute to Mr. Booth's mesothelioma.

35. Ultimately, Amaca contends that Professor Henderson's evidence amounted to an opinion that "*every such exposure adds to the cumulative risk of development of the disease, and that cumulative risk has materialised*" and further that "*the heightened risk to which the person was subject by reason of exposure to all asbestos fibres has materialised*" [81]. If this was the extent of Professor Henderson's opinion, this alone was evidence from which the trial judge could reasonably infer causation based on orthodox principles[82]. However, Professor Henderson's evidence was explicitly not limited to expressions of risk or cumulative risk. He made it clear that in drawing conclusions about causation epidemiology was important, but "*...one needs to take into account pathobiological principles and what we know about the biology of causation of the disease...*"[83] Amaca's assertion at AS [37] is misconceived and factually incorrect. Professor Henderson's opinion was that it was the totality of the inhaled fibres that cumulatively caused Mr. Booth's mesothelioma. Each exposure made a *"significant causal contribution"* to the ultimate development of his disease. [84]

Professor William Musk

36. At AS [38] Amaca incorrectly describes Professor Musk (Respiratory Specialist) as Mr. Booth's treating physician. It is apparent from Professor Musk's evidence (extracted at AS [39]) that the trial judge's summation of it at TJ [27] was accurate. Importantly, the extract confirms that when Professor Musk's attention was specifically drawn to the distinction between "*the occurrence of the tumour, rather than risk*" he agreed that "*all exposure to asbestos within an acceptable latency period materially contributes to the mesothelioma*".[85] The contention that Professor Musk spoke only of risk is without foundation.

37. In his report Professor Musk said "*Mr. Booth's exposure to asbestos from brake linings manufactured and supplied by Amaca and Amaba...was sufficient to make a material contribution* ***to the development of his mesothelioma...***"[86] In his oral testimony he agreed with the conclusions and reasoning of Professor Henderson[87] and

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[81] AS [36]
[82] *Roads & Traffic Authority v Royal* (2008) 82 ALJR 870 per Keifel J at 898 [144]
[83] CA Black 1, 119S-Y, 120B
[84] See for example Professor Henderson's evidence at CA Black 1, 117Q-T118B-D (in relation to the childhood exposure)
[85] CA Black 1, 447L-T
[86] CA Blue 1, 505-506
[87] CA Black 1, 446T-X7, 447C-K

Dr Leigh[88] as to the causes of Mr. Booth's mesothelioma and that it was the "*total fibre burden which causes the mesothelioma*".[89] Amaca's reliance[90] on Professor Musk's acknowledgment that the biological mechanism is incompletely understood does not assist it: the law does not require a complete understanding of the mechanism. In any event, Professor Musk's additional evidence that the (statistical) relationship between the inhalation of asbestos and mesothelioma was "*so consistent that's* [sic] *accepted as a causative relationship*" was more than capable of sustaining a causal inference in this case. There was no inconsistency between this evidence and the opinions of Professor Henderson and Dr Leigh. The absence of any reference to "population" in Professor Musk's evidence, belies Amaca's contention that the only available inference from his evidence was that asbestos increased the risk of mesothelioma in populations of exposed people.[91]

Dr Maurice Heiner

38. Dr Heiner is a respiratory specialist in clinical practice. At AS [44]-[49] Amaca omits reference to Dr Heiner's written opinion (from which he did not resile) that Mr. Booth's "*mesothelioma is related directly to his exposure to asbestos incurred from his course of employment fixing brake linings*"[92]. Also omitted is Dr Heiner's oral testimony that Mr. Booth's exposure to asbestos from brake linings "*materially contributed*" to his mesothelioma.[93] Ignoring Dr Heiner's agreement with Professor Henderson's views (cumulative effect explanation) regarding causation of Mr. Booth's mesothelioma[94], Amaca contends that Dr Heiner's opinion was at odds with the trial judge's summary of his evidence at TJ [31]. This is incorrect. In re-examination Dr Heiner emphatically confirmed: "*All fibres of asbestos contribute, in my opinion, to the development of mesothelioma*". Importantly, he explained that he did not need an epidemiological study to reach the conclusion that Mr. Booth's exposure to asbestos brake linings had caused or materially contributed to his mesothelioma.[95]

Dr James Leigh

39. Dr Leigh is a physician who holds a Ph.D. in occupational medicine. He is also an epidemiologist and has been a researcher in the field of asbestos related disease for more than 30 years, having published over 60 peer reviewed articles on occupational medicine including asbestos related disease, and conducted (and published) in vitro

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[88] CA Black 1, 447W
[89] CA Black 1, 446Q
[90] AS [42]
[91] AS [43]
[92] CA Blue 1, 471R-S
[93] CA Black 1, 406W-X, 407E
[94] CA Black 1, 409B-L
[95] CA Black 1, 416N-O. Dr Heiner had previously encountered cases of mesothelioma where the only identified exposure to asbestos was from brake linings: CA Black 1, 410K-Q. So too had Dr Musk: CA Black 1, 448S-T, 449M-O.

tests in cellular biology including on the clearance rates of fibres from lung tissue.[96] Dr. Leigh was in charge of the Australian Mesothelioma Register from 1988 to 2001.

40. Dr. Leigh's opinions and reasoning process on the cumulative effect of asbestos in the induction of mesothelioma was closely aligned to (and supported by) the evidence of Professor Henderson.[97] In a written report[98] Dr Leigh expressed his views not only about the epidemiology but also the cumulative cellular and biological mechanisms in the development of mesothelioma. He explained (citing a number of publications in support) that the *"current consensus view is that asbestos is involved in both the initiation phase and the promotion/proliferation phase of mesothelioma tumour development."*[99]

41. Importantly, Dr Leigh opined: *"In view of the capacity of asbestos fibres to be involved at several stages of tumour development,* ***all cumulative exposure to asbestos in an individual case must be considered to play some part in causation.*** *In an individual case current understanding suggests that cells are being initiated, initiated cells promoted and altered cells proliferating at different times. DNA repair processes are occurring, and oncogenes and suppressor genes are being activated and inactivated. Altered cells are being removed by apoptosis, necrosis and immunological means. Fibres are being cleared at differing rates and, if exposure is continuing, being deposited in the lung."*[100] [Emphasis added] As Amaca points out at AS [54] Dr Leigh added that at the cellular level this cumulative process is "stochastic" (or probabilistic); no doubt reflecting the fact that not every person who inhales asbestos will develop mesothelioma. This is entirely consistent with and does not detract from his opinion in respect of the cumulative effect of asbestos in individuals (like Mr. Booth) who actually develop the disease. This distinction is important because as Basten JA correctly pointed out "*The concept of 'risk' looks at the matter prospectively; if the risk materializes, a causal connection may be inferred*".[101] This is particularly so when the acknowledged cause of mesothelioma is asbestos and where the consensus view of medical science is that cumulative exposure initiates and promotes its development.

42. At AS [56] Amaca attempts to challenge Dr Leigh's opinion and his expertise. Despite Dr Leigh's extensive reference to scientific literature (and his own original research) Amaca attacks his opinions as "*entirely lacking in any scientific basis*". Not only was his evidence consistent with the evidence of the other doctors (particularly Professor Henderson) but as Dr Leigh explained, his understanding of the cellular basis of the cumulative effect explanation was supported by the "definitive text" by

[96] Dr Leigh's impressive CV is at CA Blue 1, 357-393
[97] Professor Henderson was in "*complete agreement*" with the substance of Dr Leigh's report: CA Blue 1, 60P-Q
[98] CA Blue 1, 292-344
[99] CA Blue 1, 298P-R
[100] CA Blue 1, 299K-Q
[101] CA [119]; see also the comment of Lady Hale that "*risk is a forward looking concept*" in *Sienkiewicz* at [170]

Dodson and Hammar[102], the conclusion to which describes in detail the complex biological process whereby multiple "*Inhaled asbestos fibres...*[cause] *pathological events through their multiple interactions between fibres and cells, cells and cells, clearance and retention, retention and relocation* ***that cumulatively lead to the causation of asbestos related diseases***..." [Emphasis added.] It may be noted that no obligation had been taken to the admissibility of Dr Leigh's evidence, or to his expertise. See *Evidence Act 1995* (NSW), s.79.

43. Critically, in his testimony in chief, Dr Leigh said "*All asbestos exposure cumulatively contributes to cause as well as risk*". He explained "*the biological basis why this is so*"[103] as follows:

> "*...asbestos fibres are continually being breathed in because you've got your background fibres being breathed in, you've got your recognised specific exposures being breathed in for the period the exposure occurs, you've got your unrecognized specific exposures being breathed in for the period that they may have occurred. Also the body is at the same time attempting to remove these fibres by various mechanisms, one of which is just engulfing them in mucus and transporting them back up the lung to be swallowed. The other is macrophages which are defensive cells which pick up or focus or oppose those fibres, or attempt to. If they successfully do it that macrophage then is taken through the lymphatic system to a part of the lymphatic system of the body where it just stays. Again, they may even go up and be swallowed. If it fails to successfully engulf these fibres then, because these fibres are long, are much longer than a macrophage...they have difficulty engulfing the complete fibre. This difficulty generates a wide range of chemical responses, some of which are cytokines which are peptide molecules which trigger other cells to come to the party. These cells can also produce free radicals which are molecules containing an oxygen atom with a free electron which are extremely reactive molecules which are capable of causing DNA damage by oxidizing the actual chemicals in the DNA which damage the DNA and cause mutation which then causes the irregularities and subsequent cell division so the mesothelioma[104]* [sic] *cell when it divides doesn't become two normal cells mesothelioma* [sic] *cells, it becomes two slightly damaged mesothelioma* [sic] *cells. This process goes on over time...and possibly a chain of eight to ten of these mutations maybe occur before the dividing cells are so damaged that they start to divide so rapidly and they become* [a] *clinically recognisable tumour. While all this is going on the fibres don't actually need the macrophages to generate the free radicals, the fibre surface themselves due to chemical reactions on the surface of the fibre can itself produce free radicals and cytokines. The clearing process is still going on, the deposition process is*

[102] CA Blue 1, 409-431; Dr Leigh has been invited to write two chapters in the next edition: CA Black 1, 220U
[103] CA Black 1, 216G-I
[104] This is a transcription error, Dr Leigh said "mesothelial".

going on, there's further – once these cells have been damaged they may not divide any further because they may die. They may die of necrosis which is the normal toxic death of a cell, or they may die because of apoptosis which is the cell itself committing suicide...There are tumour promoter genes, activation genes, onco genes activated and there are tumour suppressor genes also activated. There are also immunological mechanisms designed to control these damaged cells. All these things are going on at different points in time, at different rates, so you have to assume that the fibres are doing something all the time, both at the initiation that's the genetic change point and the subsequent proliferation point, that is the point at which the damaged cells, the tumour starts to multiply up and the damaged cells multiply further."[105]

44. Importantly, following the lengthy answer set out above, Dr Leigh confirmed that the process applied to "*all fibres*".[106] In cross-examination, Dr Leigh testified that "*they all* [each identified exposure] *would have made a material contribution*" to Mr. Booth's mesothelioma[107] and reiterated his opinion that "*Any identifiable exposure above the background* ***must*** *be considered to have had some part in causation.*"[108] Consistent with his opinion about the cumulative effect of asbestos fibres in causing mesothelioma, Dr Leigh said in relation to each exposure "*...You don't need any single one, if you had one you don't need the other three. I mean hypothetically, but there is a difference between talking hypothetically and what actually happened.*"[109] He agreed that "risk" is to be weighed prospectively "*but once a thing has occurred the exposure is to be weighed causally*".[110] Dr Leigh was acutely aware of the difference between risk and cause. Referring to Mr. Booth he said "*the risk of him getting mesothelioma is now 100% because he has got it. ... so that the whole risk calculus goes out the window.*"[111] Further, in a passage omitted from Amaca's submissions, when (in cross examination) it was put directly to Dr. Leigh that his conclusions were only that the brake work contributed to Mr. Booth's risk of contracting mesothelioma, he answered "*Both his risk and the cause.*"[112]

45. The totality of Dr Leigh's evidence alone (and in combination with the other medical evidence, particularly Professor Henderson's) provided a more than adequate foundation for the trial judge's findings on causation.

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[105] CA Black 1, 216I-Y, 217C-D.
[106] CA Black 1, 220C
[107] CA Black 1, 266O
[108] CA Black 1, 265H
[109] CA Black 1, 268C-D
[110] CA Black 1, 276E-G
[111] CA Black 1, 268G-J
[112] CA Black 1, 275T-UT

Other Issues Raised by Amaca

46. Amaca's repeated assertion[113] that the medical evidence established *"increased risk only"* at AS [57]-[60] is not supported by the medical evidence analysed above. Moreover, Amaca's contention that the doctors' evidence "*properly understood*" was only that "*the cumulative risk brought about by each exposure has materialised*" is sufficient to dispose of this appeal in Mr. Booth's favour. On this basis alone, Amaca's assertion[114] that the evidence in Mr. Booth's case was the same as the scientific knowledge in *Sienkiewicz* is untenable. In Mr. Booth's case, not only did the evidence establish that his undoubted increased risk of mesothelioma from each and all exposures to asbestos came home, but the evidence established the probable biological mechanisms by which the cumulative effect of the totality of the fibres he inhaled caused his illness.

47. The submissions[115] that the Court of Appeal's review of the evidence was "*inadequate*" and that it "*merely assumed that the evidence was to the effect summarised by the trial judge*" is without foundation, not least because the Court of Appeal specifically described the task it carried out as making its "*own assessment...as an exercise of its own evaluative judgment*".[116] And the Court of Appeal did review the evidence in detail by reference also to the transcript of the trial.[117] The criticism of the Court of Appeal's reference to risk being a prospective matter with cause being an available inference if "*the risk materialises*" is unjustified in circumstances where (as the Court of Appeal correctly observed) Professor Henderson "*accepted the causal connection at each stage*"[118], the inhalation of asbestos fibres was the accepted cause of Mr. Booth's mesothelioma and the biological evidence explained the probable cumulative mechanism responsible for the ultimate development of the disease.

48. At AS [68]-[69] Amaca relies upon Amaba's submission as to an alleged "*inference of causation from increased risk*" in S220 of 2011. In response, Mr. Booth repeats and relies upon [12]-[25] of his submissions in S220 of 2011.

49. In circumstances where asbestos was the acknowledged cause of Mr. Booth's mesothelioma, and where the evidence established the probable aetiology of the disease as the total cumulative inhalation of asbestos, the trial judge and the Court of Appeal undoubtedly applied[119] the conventional test of causation: whether Mr. Booth's inhalation of asbestos from products manufactured by Amaca and Amaba each caused

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[113] AS [57]-[60]
[114] AS [60]
[115] AS [61]-[67]
[116] CA [25]. See also CA [24],
[117] CA [50], [53], [66]-[70], [87], [113], [118], [119]
[118] CA [119]
[119] CA [84]; TJ [59], [172]

or materially contributed to his mesothelioma.[120] Amaca's contention that a "*Fairchild exception*" has been permitted is without foundation.

50. Amaca's submissions at AS [71] about "*various difficulties of analysis*" are without merit. The causal irrelevance of exposures following the development of mesothelioma is universally accepted and creates no evidentiary difficulty. The reference to the causal sufficiency of exposure tends to resurrect the discredited single fibre theory. The possibility that a particular exposure to asbestos on its own (in the assumed absence of other exposures) could have caused a mesothelioma does not arise because the medical evidence established that when the disease occurs it is the total fibre load that probably caused it. Further, defendants in mesothelioma litigation may freely make claims for contribution against other tortfeasors who negligently contributed to the illness. Similarly, there is no impediment to defendants in mesothelioma litigation calling evidence on the issue of whether, absent the breach of duty, the disease would have developed anyway. Amaca and Amaba called no evidence to this effect.

51. Contrary to Amaca's submissions[121], unlike in *Amaca Pty Limited v Ellis* (2009) 240 CLR 111, (where there were two alternative and competing causes) "*not only was* [Mr. Booth's] *cancer one which was peculiarly attributable to the inhalation of asbestos, but the evidence did ascribe a causal connection*".[122] In *Ellis* it was not proved that asbestos was a cause of (or necessary condition for) Mr. Cotton's lung cancer. In Mr. Booth's case, there was no dispute that asbestos caused his mesothelioma. Further, in *Ellis*, as the appeal was presented to this Court, there was no reliance[123] upon evidence of biological causation of the kind which was available to support the trial judge's causation conclusions in Mr. Booth's case. In this Court, the respondent in *Ellis* relied exclusively upon epidemiological studies of increased risk in an attempt to establish causation. As the Court put it "*It was not the plaintiff's argument in this Court that Dr Leigh's evidence (or the evidence of any other witness) should be understood as offering an opinion that, independent of epidemiological analysis, it could be concluded that exposure to asbestos was a cause of Mr. Cotton's cancer.*"[124]

Conclusion

52. Mr. Booth's mesothelioma was caused by asbestos, effectively the only known cause of this illness. Four doctors testified that Mr. Booth's inhalation of asbestos fibres from Amaca's brake linings over 10 years was probably a contributing cause of his mesothelioma and that the biological mechanism of the pathogenesis of the disease was the cumulative effect of the totality of the fibres inhaled. Amaca called no

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[120] *March v Stramare (E. & M.H.) Pty Ltd* (1991) 171 CLR 506 at 514 and 532, *Bennett v The Minister for Community Welfare* (1992) 176 CLR 408 at 419 and 428, *Chappel v Hart* (1998) 195 CLR 232 at [27], *Bendix Mintex Pty Limited v Barnes* (1997) 42 NSWLR 307 at 311

[121] AS [72]-[73]

[122] CA [103]

[123] *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111 at 131

[124] Ibid

medical evidence. The statistical evidence it did call (Professor Berry) supported and complemented Mr. Booth's medical evidence on causation. In this Court, Amaca seeks to re-litigate its defence referring to findings based on other evidence in other cases.

53. For the reasons discussed above, the Court of Appeal was correct in concluding that "*...there was evidence (from Professor Henderson among others) which provided a more than adequate basis for a conclusion that all inhalation of asbestos contributed to the injury.*"[125]

54. The appeal should be dismissed with costs.[126]

Part VII: First Respondent's Notice of Contention or Cross Appeal

55. Not applicable.

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[125] CA [118]

[126] Amaca obtained special leave upon giving undertakings that it would pay Mr Booth's costs in this Court in any event and would not seek to disturb the orders for costs made in the courts below.