IN THE HIGH COURT OF AUSTRALIA
SYDNEY REGISTRY

No S220 of 2011

BETWEEN:

AMABA PTY LMITED (ACN 000 387 342)
(UNDER NSW ADMINISTERED WINDING UP)
Appellant

and

JOHN WILLIAM BOOTH
First Respondent

AMACA PTY LIMITED (ACN 000 035 512)
(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. This is a case where there was a very limited grant of special leave, in circumstances where a much wider grant had been sought. The issue referred to in the appellant's ("Amaba's") submissions ("AS") at [3] is not within that grant. Nor are issues (raised elsewhere in the AS) as to the admissibility, weight or expert basis of the medical evidence.

3. Further, Mr Booth contends that the decision of the trial Judge was not on the basis that causation could be established by reference to an increase in risk, even a small increase in risk. Nor did the Court of Appeal decide the appeal on the basis that the trial Judge had so decided.

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

4. No notice is required under s78B 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 Amaba (and the Second Respondent "Amaca") contained chrysotile asbestos, and fourthly, that Mr. Booth inhaled chrysotile asbestos fibres liberated from products manufactured by Amaba (and Amaca).[1]

6. Mr. Booth accepts Amaba's summary of the proceedings at AS [6]-[7] with an important qualification. Unlike lung cancer, "*Mesothelioma is a signature malignancy for asbestos exposure*"[2], the experts describing asbestos inhalation as the "*principal*"[3], "*overwhelming*"[4], "*effectively the only known*"[5] and the "*accepted*"[6] 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 from four doctors.[7] On the contrary, Amaba (and Amaca) 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.[8]

7. The medical evidence from the four doctors included evidence as to the biological mechanisms of the pathogenesis of mesothelioma. Contrary to AS[8], it constituted evidence from which it was open to the trial judge to infer causation, applying (as he did) conventional common law principles to an injury with multiple cumulative causes rather than a single cause, or multiple competing causes.

8. Mr. Booth generally accepts Amaba's description of the relevant factual background at AS [9]-[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*"[9] and that Mr. Booth's work on brake linings was "*a very dusty process*"[10]. 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

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[1] TJ [22]
[2] CA Blue 1, 322 O; and Professor Henderson at CA Black 1, 122L-M
[3] Professor Berry (Amaba's expert epidemiologist and biostatistician): CA Black 2, 551M
[4] Professor Berry at CA Black 2, 551N
[5] Professor Henderson at CA Black 1, 122L-M
[6] Professor Musk at CA Black 1, 450P
[7] The doctors' evidence was given by way of reports and orally. In oral evidence, each gave evidence in chief and was cross-examined.
[8] Professor Berry did not accept the conclusion of those studies
[9] TJ [162(4)]
[10] TJ [19]

in which he worked[11] Further, he was exposed to asbestos from the work of others.[12]

(b) Contrary to Amaba's contention at AS [10] it was not "*common ground*" that "*the most significant exposure*" sustained by Mr. Booth was that common to all Australians ("*the background risk*") because by definition such exposure cannot be quantified. "*Background risk*" is a statistical representation of cases of mesothelioma for which identifiable episodes of exposure to asbestos are unable to be recalled or are unknown. Such cases may be the result of transient low level exposures to asbestos in the general environment. Professor Berry (an epidemiologist and biostatistician called by Amaba) thought that in Mr. Booth's circumstances, 70 per million per lifetime was the appropriate figure[13]. The eminent pathologist Professor Henderson agreed.[14] In any event 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*"[15].

(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.[16]

(d) At AS [13] Amaba omits reference to tremolite asbestos (fibres with similar carcinogenic potency to amosite[17]) a contaminant of commercial chrysotile used in the brake linings which it manufactured.

9. Insofar as they purport to be a summary of the trial judge's approach to and findings in respect of causation, the submissions at AS [14]-[15] are inaccurate. The trial judge found that asbestos dust liberated from brake linings manufactured by Amaba (and Amaca) materially contributed to Mr. Booth's mesothelioma[18] on the basis of his acceptance of the "*almost universally accepted*"[19] opinions of Mr. Booth's medical experts that mesothelioma is caused is by the total cumulative effect of all fibres inhaled ("total fibre burden") within an acceptable latency period (the "cumulative effect explanation").[20] Importantly, the trial judge rejected[21] Amaba's apparent reliance upon the "*single fibre theory*" and the "*threshold theory*" which respectively

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[11] TJ [10]-[18]
[12] TJ [11], [12], [13], [15], [17], [18]
[13] Professor Berry at CA Blue 7, 3018Q
[14] CA Black 1, 114H-M
[15] TJ [162 (2)]
[16] See for example Professor Henderson at CA Blue 1, 67
[17] Professor Henderson at CA Black 1, 86G
[18] TJ [172]
[19] CA [51], quoting Professor Henderson's evidence
[20] This evidence had been accepted by the trial judge and the New South Wales Court of Appeal in *E.M. Baldwin & Son Pty Ltd v Plane* [1998] NSWCA 23; 17 NSWCCR 434; cf TJ [58]
[21] TJ [48]-[49]

hold that mesothelioma is caused by one asbestos fibre or one particular episode of asbestos exposure to the exclusion of all others.[22]

Part V: Legislation

10. Mr. Booth accepts Amaba's statement of the relevant statutory provision.

Part VI: First Respondent's Argument

11. Mr Booth contends that parts of the "Argument" in the AS are outside the grant of special leave and should be rejected. AS[27]-[32] provide an example. Nonetheless it seems desirable, whilst maintaining that contention, to deal with the substance of those arguments.

12. Amaba, at AS[17], attempts to characterise the approach taken by the trial Judge in ways which do not reflect his actual approach. His reasons involved an acceptance of the "*almost universally accepted*"[23] (hardly "*controversial*"[24]) biological understanding that mesothelioma is caused by the cumulative effect of "*all asbestos exposures, both recalled and unrecalled*"[25] (with the exception of trivial exposures). That is quite opposed to Amaba's concept of risk (weighing up the chance of one exposure causing the mesothelioma as opposed to another exposure causing it). All exposures (acting at different stages and at different times during the biological process of carcinogenesis) were cumulatively (or in combination) causative of the singular disease. This finding is of paramount importance not only in relation to the identification of the applicable legal principles, but also to the relevance of the United Kingdom cases upon whose facts (not legal reasoning) Amaba (and Amaca) now seek to rely.

The Trial Judge Did Not Find Causation On the Basis of Increased Risk

13. The statement of principle enunciated at AS [18] is unexceptionable. However, it does not assist Amaba in circumstances where first, asbestos was the acknowledged cause of Mr. Booth's mesothelioma; secondly, the medical evidence established that the likely biological aetiology of mesothelioma was the cumulative effect of the totality of the fibres inhaled; and thirdly, where the medical evidence was that each of the undoubted (pre mesothelioma) increased risks of exposure had come home.

14. Contrary to AS [20], 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*".[26] In *Ellis* it was not proved that asbestos was a cause of (or necessary condition for) Mr. Cotton's lung cancer. In the present case,

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[22] TJ [60]
[23] Professor Henderson at CA Black 1, 92O
[24] AS [17]
[25] Professor Henderson at CA Black 1, 92O-P
[26] CA [103]

there was no dispute that asbestos caused the mesothelioma. Further, in *Ellis*, as the appeal was presented to the Court, there was no reliance[27] upon evidence of the biological mechanisms of causation of the kind which was available to support the trial judge's findings in this 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.*"[28]

15. Amaba's implicit assertion that the trial judge permitted a "*Fairchild exception*"[29] is without foundation. In *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32, the House of Lords proceeded on a 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 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.*"[30] The contest between a single fibre theory[31] and the cumulative effect explanation of the aetiology of mesothelioma was, on the evidence in the present case, resolved, with the cumulative effect explanation held to be the most probable biological mechanism by which mesothelioma is caused.

16. In *Sienkiewicz v Grief (UK) Ltd* [2011] 2 WLR 523, the Supreme Court of the United Kingdom revisited causation of mesothelioma. The case "*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*".[32] 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.*"[33] 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

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[27] *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111 at [47]
[28] Ibid
[29] AS [21]
[30] *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 per Lord Bingham of Cornhill at [7]
[31] 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]; per Lord Rodger to the same effect at [120]-[121]. The evidence of Professor Henderson in the present case was that the "single fibre theory" was biologically "ridiculous": CA Black 1, 97J.
[32] *Sienkiewicz v Grief (UK) Ltd* [2011] 2 WLR 523 per Lord Phillips at [18]
[33] *Sienkiewicz v Grief (UK) Ltd* [2011] 2 WLR 523 per Lord Phillips at [102]

contributed to causing the disease?[34] 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*".[35] In other words, if mesothelioma is a disease probably caused by the cumulative effect of inhaled asbestos, the *Fairchild* exception is unnecessary.

17. It is not readily apparent why the claimants in the United Kingdom cases referred to above did not (as Mr. Booth did) call evidence from internationally recognised medical authorities including Professor Henderson[36] and Dr Leigh[37], as to the probable biological explanation of the cumulative effect of asbestos fibres in the causation of mesothelioma. And it is, with respect, extraordinary that Amaba now implicitly seeks to rely on findings in other cases having called no evidence.

18. Amaba's contentions at AS [23] in relation to the "but for" test are misconceived. They were comprehensively addressed and rejected by the Court of Appeal at CA [93]-[114], with Basten JA observing at CA [114] that the fact that "*contraction of mesothelioma independently of exposure from work as a motor mechanic could not be excluded...is not a denial of "but for" causation*" in the circumstances of Mr. Booth's case. No doubt, His Honour's comment reflected the conventional understanding that where *"multiple sufficient causes"* act cumulatively to cause an injury, "*general law principles undoubtedly include the concept of 'material contribution'*".[38] In any event, the trial judge's finding[39] that all exposure to asbestos beyond *de minimis* contributes to mesothelioma and that Mr. Booth's exposure to asbestos from the brake linings manufactured by Amaba and Amaca (over 27 years) each made a material contribution to his disease[40], is an implicit acceptance that all exposure to asbestos (other than trivial exposure) within an acceptable latency period was a necessary condition for the initiation, promotion and ultimate development of mesothelioma. The cumulative exposure dose-response understanding of the biological pathogenesis of mesothelioma necessarily means that the risk from all of the plaintiff's exposures came home.[41] Amaba (and Amaca) called no evidence to attempt to prove that absent any particular exposure, Mr. Booth would have probably developed mesothelioma anyway.

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[34] Ibid, see also Lord Dyson at [208]
[35] Ibid per Lord Phillips at [104]. See statements to a similar effect by Lord Rodger at [142]; per Lord Dyson at [213]
[36] Professor Henderson's CV is at CA Blue 1, 105-158
[37] Dr Leigh's CV is at CA Blue 1, 357-393
[38] CA [109].
[39] TJ [59]
[40] TJ [169]-[172]
[41] See eg the evidence of Professor Henderson at CA Black 1: 119H and the report of Dr Leigh at CA Blue 1: 299K-Q

19. In relation to the "but for" test, as Mason CJ said in *March v Stramare (E. & M.H) Pty Ltd* (1991) 171 CLR 506 at 516, *"the application of that test proves to be either inadequate or troublesome in various situations in which there are multiple acts or events leading to the plaintiff's injury: see, for example, Chapman v. Hearse; Baker v. Willoughby [1969] UKHL 8; (1970) AC 467; McGhee v. National Coal Board... The cases demonstrate the lesson of experience, namely, that the test, applied as an exclusive criterion of causation, yields unacceptable results and that the results which it yields must be tempered by the making of value judgments and the infusion of policy considerations."*[42] In this sense, causation at law requires the application of common sense to the facts.[43]

20. Amaba's assertions at AS [24] about Lord Reid's approach in *Bonnington Castings v Wardlaw*[44] are misconceived. As the trial judge observed[45], in *Ellis* this Court did not disapprove of Lord Reid's categorisation of "material contribution" as any contribution which does not fall within the exception *de minimis non curat lex.* On the facts of *Ellis*, the necessary causal connection (between the inhalation of asbestos and lung cancer) was not established and for this reason this Court considered that questions as to the extent of the contribution of asbestos, and what is a "material contribution" in law, did not arise.[46] In cases where the evidentiary gap which existed in *Ellis* has been bridged, and an issue arises as to materiality of the contribution, the *de minimis* threshold may be used to determine materiality.[47] As the Court of Appeal observed at CA [107] "*in light of the evidence relating to cumulative effect, the analogy with Bonnington Castings drawn by the primary judge was apt*". At CA [84] the Court of Appeal correctly enunciated the appropriate legal test for the determination of causation in Mr. Booth's case.

21. The reference at AS [24] to the comments of Lord Phillips in *Sienkiewicz,* do not support the contention that the concept of "material contribution" applies only to divisible injuries. As Lord Phillips pointed out the critical matter is the process by which the relevant agent causes the disease: "*if the disease is caused by the cumulative effect of the inhalation of dust...the defendant will be liable on the ground that its breach of duty has made a material contribution to the disease.*"[48] Indivisible injuries (including mesothelioma) may well have more than one contributing cause, as the evidence in this case established. Lung cancer caused by cigarette smoking is an example of an indivisible disease whose causes are cumulative.[49] Ultimately, as Lord

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[42] In *March*, see also Deane J at 522-23, Toohey J at 524, Gaudron J agreeing at 525
[43] *March v E & MH Stramare Pty Limited* (1991) 171 CLR 506 at 515 per Mason CJ; *Bennett v The Minister for Community Welfare* (1992) 176 CLR 406
[44] [1956] AC 613
[45] TJ [170]
[46] *Ellis* at [68]
[47] See also *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
[48] *Sienkiewicz v Grief (UK) Ltd* [2011] 2 WLR 523 per Lord Phillips at [17]
[49] Ibid; an example per Lord Phillips at [13]

Phillips said in *Sienkiewicz* it is doubtful whether it would ever be possible to define what constituted a causal contribution (not *de minimis*) in quantitative terms, the matter necessarily being "*a question for the judge on the facts of the particular case.*"[50]

22. The assertions at AS [25] about the trial judge's alleged mathematical errors in the alleged calculations of increased risk are incorrect for a number of reasons.[51] First, an examination of the structure of the trial judge's judgment (below) reveals he did not deal with causation on the basis of increase in risk; he was estimating additional fibre burden as a guide to materiality of contribution. Secondly, the trial judge was entitled to adopt the background rate of 70 per million per lifetime: the evidence of Professors Henderson and Berry explicitly supported it.[52] Thirdly, the trial judge did not use a "*false comparator*" because he was not calculating (and comparing) increases in risk from one exposure as opposed to all others (having earlier found that all fibres cumulatively caused Mr. Booth's mesothelioma), but rather estimating the increased fibre burden above background in order to deal with Amaba's (and Amaca's) submission that Mr. Booth's exposure was trivial or insignificant in comparison to the background.

23. The structure of the trial judge's judgment demonstrates that he did not equate increased risk with cause and that he adopted an entirely orthodox approach to causation. From TJ [22] to [38] his Honour set out Mr. Booth's contentions and summarised the medical evidence including the evidence supporting the "*mechanical and chemical steps by which...the accumulation of asbestos fibres cause mesothelioma...*"[53] At TJ [39] the trial judge set out Amaba's and Amaca's four contentions on causation including "*(1) Because the biological process whereby the inhaling of asbestos causes mesothelioma is incompletely understood medical science cannot support the proposition that all asbestos inhaled materially contributes to the causes of mesothelioma*", and "*(4) When quantified, Mr. Booth's cumulative exposure to the products of either Amaca or Amaba is insignificant in comparison to his background and other exposures, and made no material contribution to the causes of his mesothelioma*".

24. From TJ [40] to TJ [62] the trial judge rejected contention (1), concluding at TJ[59] 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*".[54] This was an acceptance of the biological and pathological evidence that the cause of Mr. Booth's mesothelioma ("*actual aetiology of the disease*"[55]) was his total cumulative exposure to asbestos (total fibre

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[50] *Sienkiewicz v Grief* (UK) Ltd [2011] 2 WLR 523 per Lord Phillips at [108]
[51] It is submitted that the issue sought to be raised in AS[25] and [26] is outside the grant of special leave.
[52] See Professor Henderson at CA Black 1, 114H-M and Professor Berry at CA Blue 7, 3018Q
[53] TJ [25]
[54] TJ [59]
[55] TJ [52]

burden) within the relevant latency period. Unlike *Fairchild* and *Sienkiewicz* the trial judge on the evidence, specifically rejected the single fibre theory and the threshold theory.[56] This approach was supported by the fact that medical science accepts "*the fact that the incidence* [of mesothelioma] *rises with increasing dose* [suggesting] *that the further exposure plays a causative part in the aetiology of the disease*".[57]

25. At TJ [63]-[66] the trial judge dismissed Amaba's (and Amaca's) second contention as to the relevance of the weaker potency of chrysotile asbestos, and at TJ [65] his Honour rejected as "*entirely to the contrary of the evidence*" a submission that because chrysotile fibres in brake linings were bound in resin they were non respirable. At TJ [67]-[82] the trial judge rejected Amaba's (and Amaca's) third contention in relation to the effect of the controversial epidemiological studies. Amaba's (and Amaca's) fourth trial contention as to the quantification of Mr. Booth's cumulative exposure to asbestos was dealt with at TJ [83]-[160]. In his analysis, his Honour was not calculating increases in risk for causation purposes, a finding he had already made at TJ [59]; rather he was dealing with an attempt by Amaba (and Amaca) to prove that Mr. Booth's exposure to asbestos dust from brake innings was trivial (and presumably not a material contributor to his mesothelioma). For this purpose Amaba and Amaca called two occupational hygienists Mr. Rogers and Mr. Pickford who attempted to retrospectively calculate (without actual measurements of the asbestos fibre concentrations in the air of the workshops in which Mr. Booth worked for 27 years) Mr. Booth's total cumulative dose of asbestos. The estimates were provided by Amaba and Amaca to Professor Berry, who attempt to calculate (using dose response statistics) how many extra cases of mesothelioma would be caused by such cumulative totals. Importantly, Professor Berry described this task as relevant to an assessment of apportionment between employers "*...where it hasn't been disputed that the asbestos has caused the mesothelioma but there is disputes or differences of opinion between different employers as to how much of the damages they are liable for*".[58] Professor Berry agreed with the propositions that it is the "*lifetime load of all asbestos exposure which causes the illness in the individual*"[59] and that "*...medically it would be unsafe to proceed on the basis of one or the other, everything rather suggests, because we cannot track fibres being inspired and exhaled harmlessly, that it is the lifetime load.*"[60] He also agreed that as an epidemiologist he was concerned with "*increased risk rather than cause*" and (referring to mesothelioma) he observed that "*...when the risk comes home there is the cause*".[61]

26. Noting Professor Berry's evidence above, the trial judge observed that his calculations (based on estimates of Mr. Booth's cumulative exposure to asbestos) could be used "*as*

[56] TJ [48]-[49]
[57] TJ [57]. See also TJ [55] where the trial judge referred to this fact as being inconsistent with Amaba's contention that mesothelioma may be caused by a threshold dose with further exposure being irrelevant to causation
[58] CA Black 2, 553Y, 554C
[59] CA Black 2, 552V-W
[60] CA Blue 7, 3082M-N
[61] CA Black 2, 553E

estimates of causal contribution"[62]. However, his Honour ultimately, considered the cumulative estimates of fibre burden were underestimates and that it was "*unnecessary to decide*"[63]their accuracy. He held that "*proof of causation in this case does not turn upon the epidemiological evidence or upon questionable estimations of total fibre burden*"[64] but that an "*overwhelming inference of causation*" could also be drawn from the four undisputed facts set out at TJ [162]. At TJ [166], reiterating that he did not consider the mathematical calculations of "*additional fibre burden*" (as opposed to risk) to be "*necessarily compelling*"[65], the trial judge tentatively quantified "*the excess accumulation of fibre burden beyond background exposure*" from products manufactured by Amaba (and Amaca), describing this pragmatic approach as "*simple apportionment*".[66] Having decided that mesothelioma was probably caused by the cumulative effect of all fibres (other than from trivial exposures) the trial judge was persuaded that Mr. Booth's exposure to asbestos from each of Amaba's (and Amaca's) brake linings materially contributed to his mesothelioma.[67]

27. For the reasons discussed above, the trial judge's approach to causation was conventional. It revealed no error of law and the Court of Appeal was correct in so determining.

Response to Amaba's Arguments About "*Another reason to doubt the existence of a risk*"

28. Amaba's submissions at AS [27]-[32] were advanced to and conclusively rejected by the trial judge at TJ [67]-[82]. They are not available to Amaba in this limited appeal. They serve to reinforce the contradictory manner in which Amaba sought to utilise the controversial "*22 epidemiological studies*"[68] in circumstances where no expert agreed with their conclusions; Amaba's epidemiologist Professor Berry, declined to refer to them[69] and testified that Mr. Booth was undoubtedly at increased risk of contracting mesothelioma from brake work.[70] Amaba's contention[71] in relation to the effect of the studies is wrong. It was rejected by the trial judge[72], who found it was "*not justified by the data*"[73] and that much of the data did "*support a strong correlation...*"[74] Each medical and scientific expert including Professor Berry[75], Dr Leigh[76] and Professor Henderson, rejected it. Professor Henderson explained that the studies relied upon by Amaba "*did not negate conclusively a small increase in risk*" because they were

[62] TJ [93]
[63] TJ [160]
[64] TJ [162] referred to at CA [133]
[65] TJ [166]
[66] TJ [168]
[67] TJ [172]
[68] AS [27]
[69] CA Blue 7, 3018F-G
[70] CA Black 2, 544R-S
[71] AS [29]
[72] TJ [74]
[73] TJ [80]
[74] TJ [81]
[75] See TJ [74]
[76] TJ [76]-[81]

"*studies bedevilled by problems* [including]*...limitations to detect such a risk...*"[77] Amaba's contention in the last sentence of AS [27] is not supported by the reference to the evidence of Dr Leigh or any other expert. It was rejected by the trial judge as "*entirely... contrary*" to the evidence.[78]

The Cumulative Effect Explanation and the Evidence of Causation

29. In relation to AS [33] the trial judge did not causally implicate "*every asbestos fibre*". He specifically excluded any causal role for trivial or *de minimis* exposures to asbestos fibres. There was also the uncontentious medical evidence that some of the inhaled fibres from each exposure will be cleared (and therefore not reach the pleura) at different times by the body's inherent biological defence mechanisms[79].

30. Amaba's fundamental assertion[80] is that "*the medical witnesses were saying no more than the risk of mesothelioma increases with the dose of asbestos*" and that for this reason alone there was "no evidence" from which it was open to the trial judge to infer that asbestos from Amaba's brake linings was a cause of Mr. Booth's mesothelioma. In advancing this submission Amaba ignores the incontrovertible facts in Mr. Booth's particular circumstances set out above at [5]-[6], and [8]. It also ignores the trial judge's rejection of the single fibre theory in favour of the cumulative effect explanation of the aetiology of mesothelioma.

31. For Amaba to establish error in point of law on the basis that there was no evidence upon which the trial judge could infer that Mr. Booth's mesothelioma was caused or materially contributed to by his exposure to asbestos from Amaba's brake linings.

32. Amaba needs to establish:

(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[81]; 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

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[77] CA Black 1, 122E-J
[78] TJ [65]
[79] See Professor Henderson explanation at CA Black 1, 114V-Y
[80] AS [34]
[81] A submission that the Court of Appeal described as "*not made out factually*": CA [120]

(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 Amaba (and Amaca).

33. An analysis of the largely unchallenged medical evidence (below) leads to the conclusion that each contention referred to at [31] is unsubstantiated and that the medical evidence either, alone or in combination with the particular factual matrix in Mr. Booth's case, provided a more than adequate basis from which it was open to the trial judge to infer causation applying conventional principles.

Professor Henderson

34. Professor Henderson is a Professor of Pathology and an "*internationally recognised expert on pleural tumours and mesothelioma*"[82]. 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.*"[83] At AS [36]-[43] Amaba sets out a limited selection of Professor Henderson's evidence and despite the unambiguous nature of the words used by the Professor, boldly asserts that the Professor spoke only of risk.

35. In his report of 2 March 2009[84] Professor Henderson took into account the controversial epidemiological studies referred to by Amaba and (relying not only on epidemiological studies of risk)[85] 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...*"[86] That this 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*".[87] 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 or epidemiological risks. [88] He said that because "*biological systems such as human*

[82] CA [60]. His extensive CV is at CA Blue 1, 105-157
[83] CA Black 1, 85
[84] CA Blue 1, 38
[85] 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
[86] Ibid
[87] CA Blue 1, 57J
[88] CA Blue 1, 83

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*".[89] 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 Amaba's attempt to re-characterise his opinions on causation as relating solely to a nebulous statistical representation of risk rather than cause.

36. In relation to Mr. Booth's mesothelioma Professor Henderson expressed the view that it was "*...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.*"[90] Professor Henderson's evidence in relation to causation went further. He opined that "*Given that his 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.*"[91] 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*"[92]

37. 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*"[93] and 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.*"[94] This answer has two parts. The first sentence accepts the cumulative effect biological explanation of the aetiology of mesothelioma; the second deals with the dose response model by which estimates of causal contributions (such as those performed by Professor Berry) may be made. The

[89] CA Blue 1, 84G-H
[90] CA Blue 1, 100L-O
[91] CA Blue 1, 100P-R
[92] CA Blue 1, 61F-G
[93] Amaba's statement "*over objection with a leading question*" at AS [39] is irrelevant. It should be disregarded.
[94] CA Black 1, 91U, 92O-Q

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"*.[95] 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...*"[96] 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.*"

38. 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...*"[97] 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.***"[98] [Emphasis added] With this evidence, (which was not relevantly challenged) Professor Henderson conclusively dealt with the perceived uncertainty upon which *Fairchild* and *Sienkiewicz* proceeded.

39. During cross examination it was not put to Professor Henderson 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[99] in response to which he readily conceded: "*Not only do I not understand it completely, neither does anybody else.*"[100] He explained that: "... *We don't know all the details in humans, but we have some pretty good ideas*"[101], "*...the*

[95] CA Black 1, S-T.
[96] Ibid
[97] CA Black 1, 95W-Y
[98] CA Black 97C-J
[99] CA Black 1, 114S
[100] CA Black 1, 114T
[101] CA Black 1, 114V-Y, 116B-D

science is always incomplete..."[102] 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."*[103]

40. As noted at AS [39] Professor Henderson was cross examined about risk; but it was not suggested to him that his understanding of the biological mechanisms underpinning the cumulative effect explanation of mesothelioma causation was in some way reduced to a mere expression of increased risk or inconsistent with it. The question and answer set out at AS [39] does not support Amaba'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 differing concepts of risk) eschewed a *"nebulous or theoretical construct..."* of the term, describing it as *"...a bad term but everybody uses it."*[104] 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 above.

41. In so far as Amaba's reference to the evidence at AS [40] implies a connection with the evidence referred to at AS [39], it is inaccurate. The evidence at AS [40] was in relation to what counsel for Amaba (and Amaca) described as a *"new subject"*[105] 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.[106] 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 Amaba because this hypothetical exercise says nothing about the actual cumulative biological causal process described earlier in Professor Henderson's evidence.

42. The evidence referred to at AS [41] is consistent with (it hardly *"contradicts"*[107]) and confirms Professor Henderson earlier testimony as to biological cumulative process of causation and his rejection of the single fibre theory. Unsurprisingly, Professor Henderson was not prepared to say that Mr. Booth's mesothelioma was caused *"individually"*[108] by the risk of one particular exposure as opposed to another. Rather, 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.***"[109] Professor Henderson

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[102] CA Black 1, 115E
[103] CA Black 1, 115K
[104] CA Black 1, 115N
[105] CA Black 1, 117I
[106] See the exchanges at CA Black 1, 117I-T, X-Y, 118B-Y
[107] AS [43]
[108] That was the premise of the question extracted at AS [42]
[109] CA Black 1, 119H-I

was not cross examined about, nor did he resile from his opinion that Mr. Booth's exposure to asbestos from brake linings manufactured by Amaba (and Amaca) made a significant causal contribution to Mr. Booth's mesothelioma. It was not suggested to Professor Henderson that because of clearance mechanisms the fibres emanating from Amaba's (and Amaca's) brake linings did not materially contribute to Mr. Booth's mesothelioma.

43. Moreover, acknowledging the limitations in drawing causal conclusions from epidemiology (i.e. risk alone) Professor Henderson said: "*...I think one needs to take into account pathobiological principles and what we know about the biology of causation of the disease...*"[110] This cogent evidence underscores the conclusion that when Professor Henderson spoke of cause or causal contribution in relation to Mr. Booth, he was not referring to a nebulous statistical risk alone but a biological model (see Professor Henderson's reference the biological "model" above) of causation of mesothelioma in accordance with the cumulative effect explanation.

44. Professor Henderson's evidence was not only capable of sustaining the inference that he was referring to cause but it expressly supported the relevant causal connection.

Dr James Leigh

45. 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 tests in cellular biology including on the clearance rates of fibres from lung tissue.[111] Dr. Leigh was in charge of the Australian Mesothelioma Register from 1988 to 2001. Having taken no objection to Dr Leigh's expertise at trial (under s79 of the *Evidence Act 1995* (NSW)), Amaba inaccurately, impermissibly (and with respect, inappropriately) seeks to denigrate his opinions and him personally.[112]

46. Contrary to the implication at AS [44] 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.[113] In a written report[114] Dr Leigh expressed his views not only about the epidemiology but also the cumulative cellular and biological mechanisms in the causation of mesothelioma. He explained (citing a number of publications) that the *"current consensus view is that asbestos is involved in both the initiation phase and the promotion/proliferation phase of mesothelioma tumour development."*[115] Importantly, Dr Leigh opined: *"In view of the capacity of asbestos fibres to be involved at several stages of tumour development,* ***all***

[110] CA Black 1, 119S-Y, 120B
[111] Dr Leigh's impressive CV is at CA Blue 1, 357-393
[112] AS [44], AS [Footnote 39], AS [59(a)]
[113] Professor Henderson was in "*complete agreement*" with the substance of Dr Leigh's report: CA Blue 1, 60P-Q
[114] CA Blue 1, 292-344
[115] CA Blue 1, 298P-R

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."*[116] [Emphasis added] While 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 of the cumulative causative process in individuals 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*".[117] 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.

47. At AS [46] Amaba repeats its impermissible attempt to challenge the admissibility of Dr Leigh's opinion. At AS [47]-[49] it selects limited passages (not touching upon the cumulative biological mechanisms earlier described by Dr Leigh) in support of its contention that Dr Leigh conflated risk with cause. Not only are the selected passages capable of supporting the conclusion that Dr Leigh effectively distinguished between risk and cause, but his other evidence did so in terms. In his report, referring to each of Mr. Booth's exposures he wrote "*This exposure would have added to the "background exposure" and increased the risk of mesothelioma. This risk has now been expressed and in my opinion it is more likely than not that each of the above exposures would have made a material contribution to causation."*[118] In the oral testimony reproduced at AS [48] Dr Leigh confirmed his opinion that all cumulative exposure to asbestos contributes to "*cause as well as risk*" and that "*Once the disease has occurred the risk has come home or been expressed."*[119] In the very next answer Dr Leigh explained at length by reference to cellular mechanisms "*on a biological basis why this is so*".[120] He agreed that the cumulative cellular mechanism he described applied to all fibres.[121] His evidence to this effect was not challenged.

48. Contrary to Amaba's assertion at AS [50] Dr. Leigh characterised as "*reductio ad absurdum*"[122] the suggestion that exposure to a single fibre in addition to background could make a "*contribution*"[123] because "*...that single fibre...would have been*

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[116] CA Blue 1, 299K-Q
[117] CA [119]; see also the comment of Lady Hale that "*risk is a forward looking concept*" in *Sienkiewicz* at [170]
[118] CA Blue 1, 306G-I
[119] CA Black 1, 216G-H
[120] See Dr Leigh extensive answer at CA Black 1, 216I-Y, 217C-D.
[121] CA Black 1 220C
[122] CA Black 1, 269G
[123] It was in this sense that Dr Leigh understood the question, not the single fibre theory: see CA Blue 1, 268N

cleared"[124] and " *...it is not physically possible.*"[125] This evidence is entirely consistent with and supports the biological cumulative effect explanation and the trial judge's finding at TJ [59]. Importantly, Dr Leigh testified that his understanding of the cellular basis of the cumulative effect explanation is consistent with the "definitive text" by Dodson and Hammar[126] which, inter alia, 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.]

10 49. In cross-examination, counsel for Amaba engaged Dr Leigh in a similar hypothetical exercise[127] to that described above in relation to Professor Henderson. During this exchange Dr Leigh testified that "*they all* [each identified exposure] *would have made a material contribution*" to Mr. Booth's mesothelioma[128] and that "*Any identifiable exposure above the background must be considered to have had some part in causation.*"[129] Consistently 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.*"[130] He agreed that "risk" is to be weighed prospectively "*but once*
20 *a thing has occurred the exposure is to be weighed causally*".[131] 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.*"[132] In a passage omitted from Amaba'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.*"[133] The totality of Dr Leigh's evidence alone (and in combination with Professor Henderson's) provided a more than adequate foundation for the relevant findings of causation.

Professor William Musk and Dr Maurice Heiner

30 50. Professor Musk and Dr Heiner are respiratory physicians called by Mr. Booth at trial.

51. Amaba is wrong in its assertion that Professor Musk's report described causation in terms of increasing risk only. [134] Professor Musk's opinion was that "*Mr. Booth's*

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[124] CA Black 1, 269K
[125] CA Black 1, 269Y
[126] CA Blue 1, 409-431; Dr Leigh has been invited to write two chapters in the next edition: CA Black 1, 220U
[127] CA Black 1, 263U-265P
[128] CA Black 1, 266O
[129] CA Black 1, 265H
[130] CA Black 1, 268C-D
[131] CA Black 1, 276E-G
[132] CA Black 1, 268G-J
[133] CA Black 1, 275T-UT(?)
[134] AS [51]

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..."[135] In his testimony he agreed with the conclusions and reasoning of Professor Henderson[136] and Dr Leigh[137] as to the causes of Mr. Booth's mesothelioma and that it was the "*total fibre burden which causes the mesothelioma*".[138] At AS [52] Amaba omits the very next question and answer in which Professor Musk's attention was drawn to "*the occurrence of the mesothelioma, rather than the risk*" where he agreed that "*all exposure to asbestos within an acceptable latency period materially contributes to the mesothelioma*".[139] Further Amaba's reliance[140] on Professor Musk's acknowledgment that the biological mechanism is incompletely understood does not assist it, because the law does not require a complete understanding of the mechanism. In any event, Professor Musk's 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 an available causal inference in this case. There was no inconsistency between this evidence and the opinions of Professor Henderson and Dr Leigh. The submission in relation to Professor Musk relative expertise in comparison to the other doctors is irrelevant to this appeal and (without any disrespect to Professor Musk) unsubstantiated.[141]

52. In relation to Dr Heiner, at AS [56]-[57] Amaba omits reference to his 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*"[142] and his oral testimony that this exposure "*materially contributed*" to his mesothelioma.[143] Dr. Heiner also agreed with Professor Henderson's views regarding causation of Mr. Booth's mesothelioma.[144] In re-examination Dr Heiner confirmed his opinion that "*All fibres of asbestos contribute, in my opinion, to the development of mesothelioma*" and (importantly) 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.[145]

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[135] CA Blue 1, 505-506
[136] CA Black 1, 446T-X7, 447C-K
[137] CA Black 1, 447W
[138] CA Black 1, 446Q
[139] CA Black 1, 447P-T
[140] AS [54]
[141] See also Professor Musk's evidence that "*we know a lot about cell biology but I'm not the right person to comment on that*" at CA Black 1, 450M .
[142] CA Blue 1, 471R-S
[143] CA Black 1, E
[144] CA Black 1, 409B-L
[145] 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

Conclusion

53. 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 Amaba's brake linings over 17 years was probably a contributing cause of his mesothelioma and that the biological mechanisms of the pathogenesis of the disease was the cumulative effect of the totality of the fibres inhaled. Amaba called no medical evidence. The statistical evidence it did call (Professor Berry) supported and complemented Mr. Booth's medical evidence on causation. In this Court, Amaba seeks to re-litigate its defence referring to findings based on other evidence in other cases. It dismisses 27 years of Mr. Booth's daily inhalation of asbestos fibres contending that his mesothelioma "*seems to be the product of the rare, but real, background risk.*"[146] Paradoxically, (using phrases such as "*properly analysed*"[147]) it attempts (in a fashion described unanimously by the Court of Appeal as "*not made out factually*"[148]) to re-characterise the doctors' opinions as expressions not of cause but only increased risk; and further, that it was not open to the trial judge to conclude otherwise.

54. 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.*"[149]

55. The appeal should be dismissed with costs.[150]

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

56. Not applicable.

Dated: 27 July 2011

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[146] AS [61]
[147] AS [58]
[148] CA [120]
[149] CA [118]
[150] Amaba 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.