IN THE HIGH COURT OF AUSTRALIA

SYDNEY REGISTRY

S219

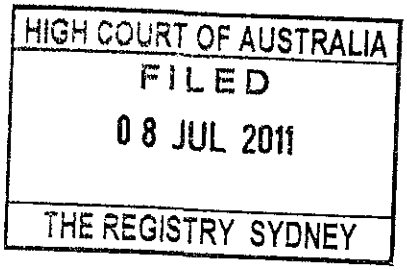
No. S of 2011

BETWEEN

AMACA PTY LTD (ACN 000 035 512)

(UNDER NSW ADMINSITERED WINDING UP)

Appellant



JOHN WILLIAM BOOTH

First Respondent

AMABA PTY LTD (ACN 000 387 342)

(UNDER NSW ADMINISTERED WINDING UP)

Second Respondent

APPELLANT'S SUBMISSIONS

Part I: Internet Publication

1. These submissions are in a form suitable for publication on the internet.

Part II: Statement of Issues

2. The issue that arises on this appeal is whether there was any evidence capable of establishing that the first respondent's mesothelioma was caused by exposure to asbestos products manufactured by the appellant.

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

3. The appellant considers that no notice pursuant to s. 78B of the *Judiciary Act* 1903 (Cth) is required.

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Part IV: Citation of Decisions Below

4. Neither the decision at first instance nor the decision of the Court of Appeal have been reported.

5. The medium neutral citation of the decision of the Dust Diseases Tribunal is *Booth v. Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8.

6. The medium neutral citation of the decision of the Court of Appeal is *Amaba Pty Ltd v. Booth* [2010] NSWCA 344.

Part V: Statement of Facts

7. The first respondent (Mr Booth) developed mesothelioma as a result of his inhalation of asbestos (CA [4]; [7]).

8. Mr Booth was exposed to asbestos:

   a. on two occasions in his childhood, while assisting his father in the construction of fibro buildings (CA [8]);

   b. for 20 minutes in 1959, while loading bags of asbestos onto a truck at the Sydney wharves (CA [9]);

   c. between 1953 and 1962, while working as a motor mechanic with brake linings manufactured by the applicant (Amaca) (70%) and other manufacturers (30%) (CA [10]-[11]);

   d. between 1962 and 1969, and 1971 and 1983, while working as a motor mechanic with brake linings manufactured by the second respondent (Amaba) (70%) and other manufacturers (CA [10]-[11]);

   e. in common with all Australians, at times and in amounts unknown.

9. In proceedings commenced in the NSW Dust Diseases Tribunal Mr Booth alleged that the asbestos to which he was exposed by Amaca was a cause of his mesothelioma, and that Amaca was negligent in failing to warn him of the dangers associated with working with asbestos brake linings.

10. The trial judge found that Amaca was negligent in failing to warn Mr Booth of the dangers of working with brake linings containing asbestos manufactured by it, and of steps that should be taken to minimize those dangers (TJ [220]-[221]). Those findings

were upheld by the Court of Appeal, and special leave to appeal to this Court on those grounds was not granted.

11. The trial judge further held that Mr Booth contracted mesothelioma because of Amaca's negligent failure to warn (TJ [221]). That conclusion entailed two findings:

    a. *First*, a finding that asbestos fibres released from brake linings manufactured by Amaca were a cause of Mr Booth's mesothelioma (TJ [62]); and

    b. *Secondly*, that a suitable warning from Amaca would have led to Mr Booth modifying his behaviour so as to avoid exposure to Amaca's asbestos (a finding not made explicitly by the trial judge, but implicit in his decision: see CA [238]).

12. This Court refused special leave to appeal on the ground that the second finding was erroneous. The grant of special leave to appeal was limited to the correctness of the first finding.

13. In particular, the issue before this Court is whether there was evidence entitling the trial judge to make the following finding (TJ [62]):

> "*Upon the facts in this case I specifically determine for the purpose of s. 25B that all exposures to chrysotile asbestos, other than trivial or de minimis exposure, occurring in a latency period of between 25 and 56 years, materially contributes to the cause of mesothelioma.*"

Part VI: Appellant's Argument

14. In Amaca's submission, the trial judge erred in finding that, where a person has developed mesothelioma, every exposure to asbestos (other than trivial or *de minimis* exposures - however one understands that qualification) within a relevant time period made a material contribution to the disease. Furthermore, the Court of Appeal erred in failing to carry out its function in an "appeal in point of law" to review the entirety of the evidence so as to conclude that it was insufficient to support that finding (CA [47]-[52], [118]).

15. The trial judge's findings in relation to the causative role of each exposure to asbestos are entirely at odds with the factual findings made in other jurisdictions on what is essentially the same body of international learning.

16. To take the most obvious example, in the United Kingdom, it has been recognised that "there is no way of identifying, even on a balance of probabilities, the source of the fibre or fibres which initiated the genetic process which culminated in the malignant tumour".[1] This lack of knowledge has been described as the "rock of uncertainty";[2] and upon that rock special judicial[3] and legislative[4] rules relating to the proof of causation in mesothelioma cases have been created.

17. In and of itself, of course, the fact that different courts may come to different conclusions in relation to questions of scientific or medical facts is neither surprising nor troubling.

10 18. In the present case, however, it serves to emphasise the importance of the careful scrutiny of the evidence relied upon to establish a scientific principle not recognised anywhere else in the world, and vigilance against the possibility that, as Professor Stapleton has put it:[5]

> "*mesothelioma victims [have] found success in Australian courts, not by the transparent judicial creation of a Fairchild-type rule, but by persuading state courts to accept certain forms of expert evidence ... [an] approach [that] seems to outstrip the limits of medical knowledge depicted in Fairchild ...*"

There should be particular pause for thought when it is seen, as here, that the experts were opining on conclusions to be drawn from what is essentially a common body of
20 international learning - there being no suggestion that Australian experts are privy to some special body of medical, scientific or epidemiological knowledge which is unavailable to experts in overseas jurisdictions.

The Trial Judge's Reasoning

19. The trial judge summarised the expert evidence adduced by Mr Booth at TJ [25]-[38]. In relation to that evidence, his Honour ultimately found that:

> "*The plaintiff's experts, conceding that some of the steps necessary to form the opinion on purely deductive and scientific grounds are yet to be discovered, are each*

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[1] *Fairchild v. Glenhaven Funeral Services Ltd* [2003] 1 AC 32 at [7], per Lord Bingham. See also *Sienkiewicz v. Grief (UK) Ltd* [2011] 2 WLR 523 at [133], per Lord Rodger. Further, see *Bolton Metropolitan Borough Council v. Municipal Mutual Insurance Ltd* [2006] 1 WLR 1492; *Re Employers' Liability Policy "Trigger" Litigation: Durham v. BAI (Run Off) Ltd* [2011] 1 All ER 605 (appeal from *Re Employers' Liability Policy "Trigger" Litigation: Durham v. BAI (Run Off) Ltd* [2009] 2 All ER 26).

[2] *Fairchild* at [7]; *Sienkiewicz* at [133].

[3] *Fairchild* and *Barker v. Corus UK Ltd* [2006] 2 AC 572. In the United States see, for example: *Menne v. Celotex Corporation* 861 F 2d 1453 (1988).

[4] *Compensation Act*, 2006 (UK), s. 3.

[5] Stapleton, "Factual Causation and Asbestos Cancers", (2010) LQR 351 at 355.

of the opinion that all asbestos fibres contribute to the development of a mesothelioma. They adopt that theory as most probably according with the actual aetiology of the disease. Their conclusions are not guesses, but reasonable inferences drawn from the current state of medical knowledge."

The staggering reach of this finding cannot be underestimated - it means that each and every source of exposure to asbestos referred to in paragraph 8 above is held to be a cause of Mr Booth contracting mesothelioma.

20. The plaintiff's experts were Professor Henderson, Dr Leigh, Professor Musk, and Dr Heiner. It is necessary to consider their evidence in order to determine if his Honour's was entitled to conclude that they were "each of the opinion that all asbestos fibres contribute to the development of a mesothelioma". Professor Henderson will be given the most attention, as his report, reasoning and referencing to the body of international learning was the most detailed of the experts. He is a leading and long experienced pathologist.

Professor Henderson

21. The trial judge dealt with Professor Henderson's evidence at [25]-[26].

22. The critical evidence supporting the trial judge's conclusion was set out at [26]. That evidence was given in examination in chief, and read in isolation, might be thought to support the trial judge's finding:[6]

Q. Professor, could you just consider this proposition please ...

All asbestos exposure within an acceptable latency period causes or materially contributes to mesothelioma. Do you agree with that proposition?

...

A. I agree with that proposition. 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."

23. The question and answer immediately following that evidence, however, are essential for a proper understanding of Professor Henderson's evidence (emphasis added):[7]

[6] CA Black at 91K-92Q.

[7] CA Black 92R-T.

Q. Thank you. And is that the phenomenon that you are describing towards the bottom of page 15 of that report we are dealing with at the moment [i.e., that dated 2 March 2009],[8] the paragraph starting "The inhaled dose of asbestos fibres by way of a no threshold dose response relationship"?

A. Yes, the text reads that the development of mesothelioma will be dependent upon the inhaled dose of asbestos fibres by way of a no threshold dose response relationship. So that is cumulative exposure increases, so does the risk of mesothelioma, and here I point out that the risk is not a theoretical construct, but rather it is a rate of the number of cases of mesothelioma one will see in the exposed populations.

24. In other words the "phenomenon" that Professor Henderson was describing in the passage quoted by the trial judge at [26], is that described on page 15 of his 2 March 2009 report. He specifically describes that phenomenon as one concerning "risk" of developing mesothelioma, risk understood as referrable to consequences which might come home across a population of persons as a whole.

25. The passage in the 2 March 2009 report to which Professor Henderson referred reads as follows:[9]

"*Appendix A that forms an attachment to this report sets forth a generic discussion on the scientific basis for causation of pleural malignant mesothelioma by asbestos. In particular, I emphasise that the risks and causal contributions from asbestos exposure towards the development of malignant mesothelioma are dependent upon the following factors in particular:*

- *The inhaled 'dose' of asbestos fibres, by way of a no-threshold dose-response relationship – so that as cumulative asbestos exposure increases so does the risk of mesothelioma as a consequence. It follows that each pattern/episode of asbestos exposure within an acceptable latency interval contributes causally towards the development of mesothelioma.*

...

All of these factors are discussed at some length in Appendix A."

26. The words "it follows" in the bullet point, by linking the second sentence with the first, emphasise that when Professor Henderson used the terminology of "cause", he was speaking of "risk" referrable to a population of persons. That is borne out by reference to Appendix A, which Professor Henderson said contained a more detailed treatment of the issues discussed in that passage.

[8] CA Blue(1) at 38, and specifically at 52.

[9] CA Blue(1) at 52.

27. At the commencement of Appendix A, Professor Henderson made some "general points", including the following:[10]

> "*From the Peto model and its modifications, the risk of mesothelioma can be related to cumulative asbestos exposure ..., so that other factors being equal, the time elapsed following commencement of exposure is a major determinant of risk: i.e., early exposures are more significant for mesothelioma risk than later exposures, other factors remaining constant.*
>
> ...
>
> *One factor that emerges from the Peto model and its modifications is that when there are multiple asbestos exposures, each contributes to cumulative exposure and hence to the risk and causation of mesothelioma, within an appropriate latency interval"*

28. The more detailed discussion of those general points in Appendix A,[11] contains nothing to suggest that Professor Henderson is speaking of anything other than an increase in risk.[12] It confirmed he was relying upon a body of internationally available material (not some special Australian learning). He also identified a puzzle of mesothelioma that while general background exposures or specific apparently limited exposures like in home renovations in some cases can be sufficient of themselves to lead to mesothelioma, at the opposite extreme mesothelioma occurs only in a minority of asbestos-exposed cases even to the heavily exposed. There may be a component of genetic susceptibility.

29. In Appendix B[13] he came to a cautious and qualified conclusion that the particular type of asbestos seen here, chrysotile, while regarded as the least harmful of all types, could still be seen to lead to some identifiable increase in risk of contracting mesothelioma across a population of brake mechanics. This however highlighted exactly how he used risk. "Relative risk" or "rate ratio", as he preferred, simply tells you the ratio of incidence rates derived from the actual number of observed cases in a population (e.g. brake mechanics) compared to a control group (viz. persons with general background exposure only). This is different to the risk for the individual within the population, which may be higher or lower.[14]

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[10] CA Blue(1) at 67.

[11] CA Blue(1) at 67-76.

[12] See also the further summary of Appendix A in the report, CA Blue(1) at 58-59.

[13] CA Blue(1) at 80-95.

[14] CA Blue(1) at 83-4.

30. His ultimate point is that he regards causation as the sum of the various risks which a person faced in advance. One starts with background risk and adds various increments of risk, including based on occupational or other specifically identifiable exposures, where the increment can be deduced from the relative risk identified across a population as a whole.[15] None of this constitutes a statement that, for a particular individual where that cumulative body or risks has come home, it is the asbestos fibre associated with any particular one, or combination of, those risks which in fact produced the disease, let alone that all of the fibres that did.

31. Professor Henderson's cross-examination further made clear that he was speaking of "risk" so explained and not "cause" as understood in law. The first relevant passage reveals the discomfort that Professor Henderson has with the term "risk".[16]

Q. What is understood, what the science establishes, is that inhaling asbestos increases the risk of contracting mesothelioma, do you agree?

A. I agree with that. But I would add the further comment that risk is not in this context simply a nebulous or theoretical construct. The risk is assessed on the basis of the numbers of cases which result from that type of inhalation. And I've always thought that risk is a bad term but everybody uses it."

32. That statement (similar to other evidence, given in examination in chief, quoted above[17]) demonstrates that Professor Henderson was concerned to make clear that the "risk" is not a "theoretical" construct, but an actual reflection of the incidence of mesothelioma in a population with a particular level of exposure to asbestos, as compared to a control population facing merely background risk. And the more asbestos to which a person is exposed, the greater the risk they have of developing mesothelioma. The reason that Professor Henderson considers "risk" to be a "bad term", therefore, is because the Professor appears to consider that it may not convey adequately the fact that in a relevant population of persons exposed to a particular level of asbestos, a certain percentage of people will, in fact, statistically, develop mesothelioma. It does not follow, however, that when one moves from speaking about the causal role of all asbestos in mesothelioma cases considered by what has in fact occurred across an entire population over a particular period of time, to the causal role of particular asbestos fibres in a particular mesothelioma case, that the word

[15] CA Blue(1) at 94.

[16] CA Black 115L-N.

[17] CA Black 92T-U.

"risk" is inappropriate or inaccurate, or that it is possible to infer "causation" in the sense relevant to lawyers from a mere identification of increase in risk.

33. The second relevant passage reveals the tendency of Professor Henderson to "slip" between the words "risk" and "cause", when he means "risk":[18]

Q. ... Professor, if Mr Booth had come to you in 2008 with a mesothelioma and did not have any other exposure above background, could that single episode [i.e., helping to build a fibro garage in 1953] to your mind explain his mesothelioma?

...

A. Okay, absent any other exposure, I would add a level of caution and not high confidence, say well, again it's one of the lowest doses of exposure I've ever found but it's an identified exposure in excess of any background, so absent any other identifiable exposure, I think it would've made a very small causal contribution also.

Q. And by that you are talking about a small increase in risk?

A. A small increase in risk.

Q. ... Putting aside anything else, no other exposure apart from that [i.e., unloading bags of asbestos for 20 minutes], would that by itself be sufficient to explain a mesothelioma in 2008?

A. Well, absent any other identifiable exposure whatsoever, I would say well, it would have made a small causal co-contribution along with his underlying background risk towards the development of his mesothelioma, so yes, the answer to that is yes.

Q. That is because it would have added to a risk?

A. Yes.

Q. And what you are really saying is that each episode of exposure adds to a risk?

A. That's right."

34. The third relevant passage reveals that when Professor Henderson uses the word "cause", in fact he means nothing more than some unidentified and identifiable part of the cumulative bundle of risks which the person faced in advance has eventuated:[19]

Q. I take it doctor when you say it caused an increase in risk, that was an increase of risk at the time?

A. No, an increase in risk subsequently, your Honour. There is no increase in risk at the time the fibre is inhaled but if the fibres are deposited in the lungs, reach the pleura, the risk such as it is, and again I think it's a very bad term, risk, because you can say, okay from this he is at risk but the risk is not – does not eventuate until the

[18] CA Black 118H-X.

[19] CA Black at 119D-I.

mesothelioma develops. And risk is always based on the numbers of cases in the exposed versus unexposed populations.

Q. In the case of Mr Booth, are you able to say whether or not that particular risk of that last exposure came home?

A. No. I'd say particularly the risk from all of his exposures came home because the model which I adopt is that of a cumulative dose response, so I think that all of the asbestos fibres that he's inhaled, or at least a proportion of them, will contribute to the risk and to the ultimate development of the mesothelioma.

Q. But I think what you are also saying is this, that individually you cannot say whether any of these risks, whether as a child, whether as a boy, whether on the back of the truck, whether from the background, or whether from [brake linings], you cannot say that any risk came home, you can only say it was an increment to the risk?

A. That's right."

35. Of particular relevance in that passage of evidence is Professor Henderson's statement that "all of the asbestos fibres that he's inhaled, *or at least a proportion of them*, will contribute to the risk and to the ultimate development of the mesothelioma" (emphasis added). The explicit acknowledgement that only a proportion of the total fibres inhaled will contribute to the development of the disease contradicts entirely the "every fibre makes a material contribution" theory.

36. Overall, it is submitted that Professor Henderson was not saying that every asbestos fibre to which a person who has developed mesothelioma was exposed contributes causally to the development of the disease. Rather, he was saying that every such exposure adds to the cumulative risk of development of the disease, and that cumulative risk has materialised. In other words, the heightened risk to which the person was subject by reason of exposure to all asbestos fibres has materialised. In that sense, each asbestos fibre has "caused" the person's mesothelioma. But that is not a statement, even at a level of probability as opposed to certainty, as to what in fact occurred to this person; it is not a statement in the sense in which the law uses the term "cause", and Professor Henderson did not intend to suggest causation in that sense was knowable, let alone present.

37. Finally on Professor Henderson, the point can be tested by his approach to Mr Booth's childhood exposures. While they were of apparent short duration, the fibres may have lasted in the air of the house for some time. Further the Peto model attributes greater risk power to exposures earlier in time. Overall they had a lower proportionate effect within the body of cumulative risks than, say, the later

occupational exposure.[20] While the relative risk analysis allowed one to conclude they were capable of playing a role in his development of mesothelioma, whether on their own or in combiation with others, Henderson did not, and could not, express any opinion whether they in fact did. The same is true for every source of exposure referred to in paragraph 8 above.

Professor Musk

38. The trial judge dealt with evidence of Mr Booth's treating physician, Professor Musk at [27]-[30]. Critically, the trial judge said (at [27]):

> "*Although at times Profesor Musk spoke in terms of cumulative exposure to asbestos increasing the risk of contracting mesothelioma, he did not in cross-examination resile from his evidence that, where a mesothelioma has occurred, all exposure has materially contributed to the development of that mesothelioma, and that this was so in the case of Mr Booth.*"

39. The evidence to which the trial judge was referring was presumably the following exchange during Professor Musk's examination in chief:[21]

> *Q. Do you consider that all exposure to asbestos within an acceptable latency period materially contributes to the mesothelioma in a particular patient?*
>
> ...
>
> *A. Yes I do, all periods of exposure outside the – more than ten to 15 years ago would have contributed to the risk.*
>
> *Q. I would like you to really draw your attention to the mesothelioma, the occurrence of the tumour, rather than the risk. Do you agree that all exposure to asbestos within an acceptable latency period materially contributes to the mesothelioma?*
>
> ...
>
> *A. Yes.*

40. Once again, in order to understand that evidence, it is necessary to have regard to other aspects of Professor Musk's evidence that explain precisely what it is that he was saying.

41. In his (brief) written report (unsupported by reference to literature) Professor Musk spoke in terms of Mr Booth's "risk" of developing mesothelioma, and the "sufficiency" of the Amaca (and Amaba) exposures to cause Mr Booth's disease:[22]

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[20] CA Blue(1) at 53-54.

[21] CA Black 447L-T.

[22] CA Blue(1) at 506.

"It is my opinion that Mr Booth's exposure to asbestos from brake linings manufactured and supplied by Amaca and Amaba in the circumstances outlined in your letter and his Statement of Particulars was sufficient to make a material contribution to the development of his mesothelioma because these were the main sources of the asbestos to which he was exposed and the period between exposure and the development of the disease was consistent with the known increasing risk with increasing time since first exposure. His earlier exposures as a child would also have contributed to his risk of developing mesothelioma to a much smaller extent because the levels of exposure would have been much less even though the time since exposure was more."

42. The relationship between "risk" and "cause" in Professor Musk's evidence was made clear in the following exchange during cross-examination:[23]

Q. Professor, do you think this is a fair way to express it, that give the biological processes remain incompletely understood, what the medical science establishes is that inhaling asbestos increases the risk of contracting mesothelioma?

A. Yeah, that's certainly true and the relationship between the inhalation of asbestos and the development of mesothelioma is so consistent that it's accepted as a causative relationship.

Q. And in fact we cannot say at a biological level how or why asbestos causes mesothelioma, we can only say that we know that inhaling asbestos is a proven risk for contracting mesothelioma?

A. Yes, it's a proven risk and – and most people, as far as I know, are prepared to say that it's a causative association.

43. In other words, like Professor Henderson, Professor Musk was saying that asbestos causes mesothelioma, and that experience across populations as a whole allows one to say that, the more a person is exposed to asbestos, the greater the risk they will develop mesothelioma. All of the asbestos to which Mr Booth was exposed served to increase his overall risk of developing mesothelioma, and Professor Musk considered that the Amaca and Amaba exposures (together – he did not consider them separately) were sufficient to "cause" Mr Booth's mesothelioma. Professor Musk was making the point that the increase in risk of developing mesothelioma in a population exposed to asbestos allows the inference to be drawn that asbestos causes that disease viewing the population as a whole. But he was not saying that, in Mr Booth's case, each exposure was in fact causative.

[23] CA Black 450N-R.

Dr Heiner

44. Dr Heiner was a consulting physician who examined Mr Booth and provided a report (without reference to the literature). The trial judge dealt with Dr Heiner's evidence at [31]-[33]. The trial judge said (at [31]):

> "*Dr Heiner says that causation in cases of mesothelioma is best explained by total cumulative asbestos exposure because there is no threshold dose below which mesothelioma will not occur, and the incidence of mesothelioma increases with cumulative dose.*"

45. It is immediately apparent that that summary of Dr Heiner's evidence does not support the "every asbestos fibre contributes" theory. Rather, it makes clear that Dr Heiner was saying that incidence of mesothelioma in the population as a whole increases with greater exposure – not that where a particular person has in fact developed mesothelioma, that every fibre has made a material contribution to the development of the disease.

46. That Dr Heiner was not suggesting that every asbestos fibre to which a person who suffers from mesothelioma is exposed makes a material contribution to the disease (as opposed to increasing the risk of the disease developing) is confirmed by a consideration of his evidence.

47. In examination in chief, Dr Heiner gave the following evidence:[24]

> *Q. Do you consider the causation is best explained by total cumulative exposure to asbestos?*
>
> *A. Well, the academic teaching, and I think the state of the academic debate at this time is (1) there is no threshold dose but (2) if one has ongoing exposure to asbestos, one then has a greater risk of developing mesothelioma. In fact, there's an equation that defines that.*
>
> *Q. Yes, and in that sense do you consider that all asbestos exposure within an acceptable latency period contributes to the ultimate mesothelioma?*
>
> ...
>
> *A. One may have a threshold exposure at age eight to asbestos and that may or may not result in a mesothelioma developing 20 or 30 years later and that depends on genetic factors, et cetera. But if that person at age eight, even if he had a very mild exposure and then through the rest of his life was continually exposed to asbestos fibres, the likelihood of him developing mesothelioma would increase and he would be more likely to develop a mesothelioma, but alone that exposure at age eight may not result in a mesothelioma from occurring. That's how I understand it.*

[24] CA Black 408G-W.

48. It may thus be seen that Dr Heiner refused to speak in terms of "causation", and framed his evidence in terms of risk and likelihood. In cross-examination, Dr Heiner made clear that it was in fact not possible to go beyond risk to cause:[25]

Q. Is this what you were saying, that what is known about it is that inhaling asbestos can, at least in some circumstances, increase the risk of contracting mesothelioma?

A. Inhaling asbestos can certainly cause mesothelioma, yes.

Q. And inhaling asbestos increases the risk, depending upon dose, fibre type and latency periods?

A. It does.

Q. And that's the best medical science can offer us in explanation at the moment is that depending on dose, fibre type and latency periods, what is known that inhaling asbestos can increase the risk of contracting mesothelioma?

A. Correct.

49. Dr Heiner thus made clear that medical science could only draw conclusions as to risk in populations as a whole. Nothing in his evidence supports the conclusion that every fibre to which a person who has in fact developed mesothelioma was exposed made a material contribution to the development of the disease.

Dr Leigh

50. Dr Leigh is a consultant physician of long standing. He was the only expert other than Professor Henderson to engage with medical and scientific literature - again on the basis of a body of learning internationally available.[26]

51. The trial judge dealt with Dr Leigh's evidence at [34]-[38]. The trial judge recorded Dr Leigh's opinion that asbestos is involved in both the initiation and promotion/proliferation of mesothelioma and said (at [35]-[36]):

"*It is because of this capacity of asbestos fibres to be involved at several stages of tumour development that Dr Leigh considers that, in an individual case, all cumulative exposure to asbestos fibre must play some part in causation.*

Although Dr Leigh at times used to word "risk" interchangeably with "cause" in his evidence, he explained that once the disease had occurred, the accumulating risk had come home, and that it was the accumulation of fibres that caused the disease in the particular case."

[25] CA Black 413Y-414E.

[26] See, e.g., CA Blue(1) at 296-305.

52. The evidence to which the trial judge was referring was presumably the report of Dr Leigh where he said:[27]

"*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.*"

53. Immediately following that sentence, however, Dr Leigh went on to explain what he meant:

"*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 being activated and initiated. 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. All these processes at cellular level are stochastic in that probabilities of fibre/cell interaction depend on the number of fibres and number of cells present at any point in time. Hence, simplistically, the more fibres, the more free radicals and greater probability of initiated, promoted or proliferated cells at any given point in time.*"

54. In other words, Dr Leigh was not saying that every fibre to which a person is exposed plays some role in the development of mesothelioma. He was saying the more fibres to which a person is exposed, the greater the probability that some process will occur to initiate or progress the disease.

55. So understood, the passage of Dr Leigh's evidence just quoted is not at all inconsistent, and is indeed to the same effect, as the earlier – quite conventional – statement in his report that:[28]

"*All exposure, recalled and unrecalled or unrecognized, would have contributed cumulatively to the risk of mesothelioma.*"

56. To the extent that Dr Leigh's evidence could even arguably be read as asserting that every asbestos fibre to which a person suffering from mesothelioma was exposed made a material contribution to the development of the disease, it was demonstrated to be entirely lacking in any credible scientific basis. In cross-examination, Dr Leigh conceded that he was not aware of a single study or authority that has ever stated such a conclusion.[29] In such circumstances, a mere assertion by a witness (assuming it to have been made), with a conceded lack of any support for it, cannot be regarded as

[27] CA Blue(1) at 299.

[28] CA Blue(1) at 294.

[29] CA Black 482D-G.

evidence sufficient to establish a proposition otherwise unproved. Even apart from reputable studies, Dr Leigh offered no chain of reasoning, based on his specialised knowledge, why the court should adopt a proposition which no other expert here or overseas adopted.

Conclusion Regarding Evidence on Causation

57. It is submitted that the above analysis of the evidence upon which the trial judge relied reveals no basis for the conclusion 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" (TJ [59]).

58. Rather, the expert evidence was uniformly to the effect that while various exposures to asbestos had been shown by reference to what occurrs across populations to increase the cumulative risk of development of mesothelioma, it was not possible to say which exposures in fact made a material contribution to the development of the disease, when or why.

59. It is clear, in other words, that the expert evidence went no further than to establish that increments to a person's exposure to asbestos might increase a person's *risk* of developing mesothelioma. The statements to the effect that "it follows" that if a person does *in fact* develop mesothelioma each exposure may be regarded as "contributing causally" to the cancer are a short-hand way of expressing the concept that the cumulative risk brought about by each exposure has materialised, but should not be read as suggesting that, in fact, each and every exposure caused the cancer. Indeed, it was very clear that not all fibres to which a person is exposed (especially chrysotile fibres) will even potentially play a causative role.

60. Properly understood, therefore, the evidence in this case was entirely consistent with the state of scientific knowledge described in *Sienkiewicz*.[30]

The Court of Appeal's Consideration of the Evidence

61. The Court of Appeal correctly (although without reference to the decision of this Court in *Kostas v. HIA Insurance Services Pty Ltd* [2010] HCA 32 at [90]) identified the bases upon which it could set aside the trial judge's decision as including:

[30] [2011] 2 WLR 523 at 531-2 and 558-562.

a. the absence of any evidence to support the factual findings made (CA [21]); or

b. the absence of any logical grounds to support the factual findings made (CA [26]).

62. In Amaca's submission, the Court of Appeal failed properly to review the whole record of the evidence given on the question to the trial judge; to consider the manner in which the experts were using a distinction between "risk" and "cause"; to consider how such distinction was reflected in the body of medical and scientific learning that they drew upon; and to ensure that the trial judge had properly applied the legal concept of causation to the record.

10 63. In significant respects, the Court of Appeal merely assumed that the evidence was to the effect summarised by the trial judge, without looking at it in its context. For example, in referring to the passages of Professor Henderson's evidence quoted by the trial judge at [25]-[26], the Court of Appeal's simply stated (CA [52]):

"*If [that evidence] were properly admitted and accepted, it provided a basis for the conclusion that all exposure (and, by inference, inhalation) contributed to the mesothelioma suffered by the plaintiff.*"

64. Further examples of the Court of Appeal's failure to consider and/or recognise the true import of the expert evidence in determining the question before it may be found at CA [64], [66], and [118]-[119]. The attempt in the later passage to explain how a
20 proposition about risk viewed in advance, identified from the behavior of populations, converts to a finding of causation for an individual after the event, suffers from the same "it follows" fallacy identified above.

65. Further the Court of Appeal never grappled squarely with the central question whether the experts truly were purporting to opine that where there were a series of risks faced by an individual in advance, contraction of the disease meant every one of those risks had come home.

66. Overall, in Amaca's submission, the Court of Appeal failed to perform the task required of it in order to determine the existence of an error of law in the trial judge's decision.

30 67. Furthermore, to the extent necessary, s. 75A of the *Supreme Court Act* 1973 (NSW) provided the Court of Appeal with ample power to review the record in order to

determine the question of the existence of evidence or logical grounds to support the finding of causation.

An Inference of Causation from Increase in Risk?

68. The trial judge found that Amaca was responsible for 10% of the additional fibres to which Mr Booth was exposed over and above his background exposure (TJ at [166]).

69. Amaca adopts the submission of Amaba in support of its submission that:

a. the true percentage of additional fibres for which Amaca was responsible was 4%; and

b. the additional risk of developing mesothelioma arising from the exposure to Amaca's fibres does not permit an inference of causation on the balance of probabilities. That is the evidence did not permit or require the conclusion that, against the background of cumulative risks faced by Mr Booth in advance, it was more probable than not that some or all of the fibres of Amaca to which he was exposed played a necessary part in his actual contraction of the disease.

Conclusion

70. The failure of the trial judge and the Court of Appeal to recognise the different sense in which the medical and scientific witnesses were using the term "cause" meant that Mr Booth was absolved from the requirement of proving that Amaca's negligence caused his loss. In substance, the trial judge and the Court of Appeal have permitted a "*Fairchild* exception" to causation (i.e., an exception to the ordinary rules of causation such as that created by the House of Lords in *Fairchild v. Glenhaven Funeral Services Ltd* [2003] 1 AC 32).

71. The failure to distinguish properly between the notions of "risk" and "cause" is liable to produce various difficulties of analysis, or lead to various other errors, in cases of this sort. For example:

a. The causal irrelevance of exposures following the development of mesothelioma (but prior to symptoms or diagnosis) is likely to be overlooked: c.f. *Re Employers' Liability Policy 'Trigger' Litigation* [2011] 1 All ER 605 at [51].

b. The causal sufficiency of non-tortious exposures (or tortious exposures by other defendants) is also likely to be overlooked: c.f. Stapleton, "Factual Causation and Asbestos Cancers", (2010) LQR 351 at 355. In this case, for example, the evidence was clear that all of Mr Booth's exposures to asbestos other than those of Amaca and Amaba were capable of causing mesothelioma on their own.[31]

c. The causal effect of forms of negligence other than negligent exposure to asbestos is likely to be ignored or not appreciated. That is to say, where, as here, the negligence is a failure to warn of dangers and/or advisable precautions, and it is not alleged that the plaintiff would not have used the product, or that the precautions would have eliminated every single fibre, the "every fibre makes a material contribution to the disease" theory in fact tends to suggest that a finding of legal causation should not be made. This aspect of the causal inquiry appears, however, frequently to be overlooked, and a finding that a particular defendant's fibres caused the disease is instead regarded as sufficient to establish liability.

72. *Amaca v Ellis* (2010) 240 CLR 111 is a recent re-affirmation of the fundamental requirement that, in all actions for negligence, including those where injury is said to have been sustained by reason of exposure to asbestos, it is necessary for the plaintiff to prove, on the balance of probabilities, that the conduct of the defendant done in breach of duty was an actual cause of the plaintiff's injury.

73. That necessity remains despite the fact that the cause of a particular injury is unable to be known medical science (or any other branch of human knowledge). It is never sufficient merely to demonstrate that the defendant's conduct has increased the plaintiff's risk of injury, nor to point out that that conduct may (but cannot be proven to) have caused the plaintiff's injury: *Ellis* (2010) 240 CLR at [70].

74. No special rules relaxing proof of causation have yet been adopted in Australia in asbestos cases, contrary to the position in England: c.f. *Fairchild v. Glenhaven Funeral Services Ltd* [2003] 1 AC 32.

[31] See, e.g., CA Black 274Q-S.

75. For these reasons, it is submitted that the trial judge erred in finding that Amaca caused or made a material contribution to Mr Booth's mesothelioma, and the Court of Appeal erred in holding that there was evidence available to the trial judge to support such a finding.

Part VII: Applicable Statutory Provisions

76. *Dust Diseases Tribunal Act* 1998 (NSW), s. 32.

Part VIII: Orders Sought

77. Amaca seeks the following orders:

a. Appeal allowed.

b. Set aside order 1 of the Court of Appeal made on 10 December 2010 and in lieu thereof order:

i. Appeal allowed.

ii. Set aside Order 1 made by Judge Curtis on 10 May 2010, and in lieu thereof order verdict and judgment for the defendants.

iii. The appellant to pay the respondent's costs of the appeal.

c. Appellant to pay the first respondent's costs of the appeal.

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