HIGH COURT OF AUSTRALIA

FRENCH CJ,
GUMMOW, HAYNE, HEYDON AND CRENNAN JJ

Matter No S219/2011

AMACA PTY LIMITED (UNDER NSW ADMINISTERED WINDING UP) APPELLANT

AND

JOHN WILLIAM BOOTH & ANOR RESPONDENTS

Matter No S220/2011

AMABA PTY LIMITED (UNDER NSW ADMINISTERED WINDING UP) APPELLANT

AND

JOHN WILLIAM BOOTH & ANOR RESPONDENTS

Amaca Pty Limited (Under NSW Administered Winding Up) v Booth
Amaba Pty Limited (Under NSW Administered Winding Up) v Booth
[2011] HCA 53
14 December 2011
S219/2011 & S220/2011

ORDER

In each matter, appeal dismissed with costs.

On appeal from the Supreme Court of New South Wales

Representation

J T Gleeson SC and N J Owens for the appellant in S219/2011 (instructed by Holman Webb Lawyers)

G M Watson SC with J C Sheller for the appellant in S220/2011 (instructed by DLA Piper Australia)

D F Jackson QC with S Tzouganatos for the first respondent in both matters (instructed by Turner Freeman Lawyers)

Submitting appearance for the second respondent in both matters

CATCHWORDS

Amaca Pty Limited (Under NSW Administered Winding Up) v Booth
Amaba Pty Limited (Under NSW Administered Winding Up) v Booth

Evidence – Expert evidence – First respondent sued appellants in Dust Diseases Tribunal of New South Wales – First respondent claimed exposure to asbestos fibres in breach of each appellant's duty of care caused his mesothelioma – First respondent's expert evidence that cumulative exposure to asbestos contributed to mesothelioma accepted at trial – Appellants led epidemiological evidence disputing link between exposure to asbestos of members of first respondent's profession and risk of mesothelioma – Whether inference of fact concerning contraction of disease reasonably open on evidence.

Negligence – Causation – Whether more probable than not that appellants' negligence was a cause of first respondent's disease – Whether issues of causation lie within common knowledge and experience – Role of expert medical evidence.

Practice and procedure – Appeal – No evidence – Appeal from Dust Diseases Tribunal of New South Wales to Supreme Court of New South Wales – Section 32 of *Dust Diseases Tribunal Act* 1989 (NSW) confers a right of appeal to Supreme Court against decision of Tribunal "in point of law" – Whether Tribunal erred in point of law when deciding that appellants' negligence more probably than not a cause of first respondent's disease.

Words and phrases – "causation", "cause and consequence", "epidemiological evidence", "manifest error", "mesothelioma".

Dust Diseases Tribunal Act 1989 (NSW), s 32.

FRENCH CJ.

Introduction

1 John Booth, a retired motor mechanic, suffers from malignant pleural mesothelioma. He had two brief exposures to asbestos as a child and youth in connection with home renovations and one brief exposure when loading a truck in 1959. He attributes his disease to his exposures to asbestos in brake linings on which he worked over 30 years between 1953 and 1983[1]. In July 2008, he commenced proceedings in the Dust Diseases Tribunal of New South Wales ("the Tribunal") against Amaca Pty Ltd ("Amaca") and Amaba Pty Ltd ("Amaba"), the two companies which manufactured most of the brake linings on which he worked.

2 The primary judge found that an "overwhelming inference of causation" adverse to Amaca and Amaba could be drawn from the following facts[2]:

- Mr Booth's mesothelioma was caused by the inhalation of asbestos fibre;
- mesothelioma very rarely occurs in persons who have not been exposed to asbestos fibres beyond the background level that pervades urban environments;
- for a total of 27 years, week in and week out, Mr Booth was additionally exposed to asbestos fibres liberated from asbestos brake shoes by his own work and by the work of others in his vicinity;
- the previous exposure in the course of home renovations and truck loading was, in comparison, trivial.

His Honour held that proof of causation in the case did not "turn upon the epidemiological evidence, or upon questionable estimations of total fibre burden."[3]

3 His Honour also found that it was reasonably foreseeable by Amaca and Amaba at the relevant times that an automotive mechanic exposed to asbestos fibre released from brake linings over many years might contract an asbestos-

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1 Less a three year interregnum between 1969 and 1971.

2 [2010] NSWDDT 8 at [162].

3 [2010] NSWDDT 8 at [162].

related disease[4]. Each of Amaca and Amaba owed a duty to take reasonable precautions to prevent Mr Booth suffering harm in consequence of the use of their products[5]. Each had breached that duty by failing to provide adequate warnings to persons working on the brake lining products[6]. His Honour awarded judgment for Mr Booth against both Amaca and Amaba in the sum of $326,640[7]. He also ordered that Amaca and Amaba pay Mr Booth's costs.

4 Amaca and Amaba appealed to the Court of Appeal of the Supreme Court of New South Wales on questions of law pursuant to s 32 of the *Dust Diseases Tribunal Act* 1989 (NSW) ("the Act"). They also filed summonses pursuant to s 69 of the *Supreme Court Act* 1970 (NSW) ("the Supreme Court Act") seeking orders in the nature of certiorari to quash the Tribunal's decision for jurisdictional error. The argument before the Court of Appeal focussed on the appeals filed under s 32. No separate argument was raised in support of the summonses under s 69 of the Supreme Court Act[8]. The Court of Appeal held that Amaca and Amaba had failed to show that the primary judge had erred in law. It dismissed their appeals and their summonses[9].

5 On 10 June 2011, Gummow, Hayne and Heydon JJ made orders granting Amaca and Amaba special leave to appeal from the decision of the Court of Appeal limited to the following grounds:

> "The Court of Appeal erred in holding that any act or omission on the part of the Appellant caused the First Respondent's injury:
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> a) By declining to correct, or alternatively by approving, the primary Court's decision that causation could be established by reference to an increase in risk, even a small increase in risk;

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4 [2010] NSWDDT 8 at [186], [198].

5 [2010] NSWDDT 8 at [200].

6 [2010] NSWDDT 8 at [207]-[212], [219], [221].

7 [2010] NSWDDT 8 at [236].

8 *Amaba Pty Ltd (Under NSW Administered Winding Up) v Booth*; *Amaca Pty Ltd (Under NSW Administered Winding Up) v Booth* (2011) Aust Torts Reports ¶82-079.

9 (2011) Aust Torts Reports ¶82-079 at 64,603 [6].

b) By declining to correct, or alternatively by approving, the primary Court's reliance upon insufficient expert opinion evidence in respect of causation."

6 For the reasons that follow the evidence was sufficient to support the conclusion of the Tribunal that Amaca and Amaba's products caused Mr Booth's disease. The appeals should be dismissed with costs.

Factual background

7 The following factual background emerged from the findings of the primary judge and is not in issue in these appeals.

8 John Booth was born on 26 April 1937. He first experienced the symptoms of his disease, shortness of breath and chest pains, in February or March 2008. He was diagnosed with mesothelioma. There is no serious contest that his disease was caused by the inhalation of asbestos fibres.

9 Mr Booth was exposed to asbestos fibre of different kinds and at different times over many years since his childhood. There were three brief exposures between 1943 and 1959. As a child in 1943, he helped his father with the cutting of asbestos sheets for use in the renovation of their family home. He held the sheets in place. The cutting process took about half an hour. In 1953, he helped his father in the building of a fibro garage over a period of two days. He held asbestos sheets in place while his father cut and nailed them to a timber frame. In 1959, Mr Booth worked briefly as a truck driver. On one occasion during that period he spent about 20 minutes loading bags containing asbestos onto his truck.

10 The brief isolated exposures described in the preceding paragraph were dwarfed by Mr Booth's occupational exposure to asbestos in brake linings during his career as a motor mechanic. Mr Booth commenced his apprenticeship in February 1953 and completed it in April 1954. From 1954 until 1983 he worked as a motor mechanic, save for an interregnum between 1969 and 1971. His work, for a variety of employers, included the replacement of brake linings made from asbestos. The frequency of the replacement tasks varied from twice a month to three times a week depending upon the particular employment.

11 Mr Booth's work in replacing brake linings required him to hammer rivets through holes in the linings in order to fix the linings to metal shoes. He would drill holes in linings when the misalignment between the manufactured holes and corresponding holes in the metal shoe was too great to allow rivets to be forced through both. His work also involved grinding the leading edge of brake linings on a bench grinder. This grinding generated asbestos dust which collected on his clothes, on the workbench and on the floor of the workshop. It would be re-agitated into the atmosphere by brooms, passing feet and the use of compressed air to clean the workbench. It took Mr Booth about four hours to replace the

linings on a passenger vehicle and up to three hours per wheel to replace the linings on commercial trucks.

12 Hardie-Bestos and Hardie-Ferodo brake linings were manufactured by Amaca between 1953 and 1962. From 1962 to 1983 they were manufactured by Amaba. Mr Booth worked with a number of different brands of brake linings during his career as a motor mechanic. The primary judge found that 70 per cent of the asbestos fibres to which he was exposed between 1953 and 1962 were released from brake linings manufactured by Amaca and 70 per cent of the fibres to which he was exposed from 1962 to 1969 and from 1971 to 1983 were from linings manufactured by Amaba[10].

13 There are different kinds of asbestos fibre including amosite, chrysotile and crocidolite. Amphibole asbestos, which includes crocidolite or amosite, is a more powerful causal agent in relation to mesothelioma than chrysotile. The kind of asbestos fibre to which Mr Booth was exposed as a child and young man in assisting his father with home renovation work was not known. The asbestos fibre which he loaded on to the truck in 1959 was probably chrysotile. So too was the asbestos used in the Hardie-Bestos and Hardie-Ferodo brake linings on which he worked.

14 In addition to the preceding factual background, the following findings of the primary judge were either not in dispute or, given the limited grant of special leave, not able to be challenged on these appeals[11]:

- Mr Booth's mesothelioma was caused by the inhalation of asbestos fibre;
- chrysotile asbestos has the capacity to cause mesothelioma;
- the brake linings manufactured by Amaca and Amaba contained chrysotile asbestos; and
- Mr Booth inhaled chrysotile asbestos fibre liberated from Amaca and Amaba products.

The primary judge's reasons on causation

15 The primary judge found that exposure to asbestos dust liberated from brake linings manufactured by Amaca and Amaba "materially contributed to

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10 [2010] NSWDDT 8 at [164]-[165].

11 [2010] NSWDDT 8 at [22].

Mr Booth's contraction of mesothelioma."[12] Amaca and Amaba contended that there was no basis in the evidence to support that conclusion. Central to their argument was the proposition that the primary judge drew an impermissible conclusion, from evidence of risk, that Mr Booth's exposure to their products had caused his mesothelioma.

16 The primary judge had regard to Mr Booth's early exposure to asbestos, his work history as a motor mechanic, his exposure to asbestos in brake linings manufactured by Amaca and Amaba, and medical and epidemiological testimony. Based upon the medical evidence, he accepted that the effect of asbestos exposures on the development of mesothelioma was cumulative[13]. He applied estimates of Mr Booth's exposures to lifetime risk figures based upon epidemiological studies. He derived from that application an estimate of Mr Booth's lifetime risks attributable to the products manufactured by Amaca and Amaba[14].

17 The expert medical witnesses at trial called by Mr Booth were Professor Douglas Henderson, a professor of pathology, Dr James Leigh, a consultant occupational physician, Dr Maurice Heiner, a consultant thoracic physician and Professor William Musk, a respiratory physician. Mr Booth also called Mr Gordon Stewart, an occupational hygienist. Amaca and Amaba did not call any medical witnesses. They relied upon the evidence of Professor Geoffrey Berry, a biostatistician and epidemiologist, and Messrs Geoffrey Pickford and Alan Rogers, who are both occupational hygienists.

18 Professor Henderson gave evidence concerning the mechanical and chemical steps by which, in his opinion, the accumulation of asbestos fibres in the lungs causes mesothelioma. In a report dated 2 March 2009 he set out a number of propositions, including the following[15]:

> "Asbestos fibres including chrysotile are known Class 1 human carcinogens.
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> The [World Health Organisation] has concluded that asbestos fibres including chrysotile have the capacity to induce both lung cancer and mesothelioma.

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12 [2010] NSWDDT 8 at [172]; see also at [219].

13 [2010] NSWDDT 8 at [47]-[62].

14 [2010] NSWDDT 8 at [67]-[138].

15 Reproduced in the judgment of the Court of Appeal: (2011) Aust Torts Reports ¶82-079 at 64,615-64,616 [87].

> No safe threshold level of asbestos exposure has been delineated for the carcinogenic risks from asbestos fibre inhalation, including chrysotile fibre inhalation.
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> …
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> Given the no-threshold model for cancer induction by asbestos, including chrysotile, exposures above background will, following an appropriate latency interval, confer an increment in risk on top of any underlying pre-existing background risk.
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> …
>
> Exposures to asbestos dust from grinding of new brake blocks/linings/pads are known to have yielded increased airborne concentrations of respirable asbestos fibres.
>
> Such inhalation represents exposure in excess of any exposure derived from the general environment.
>
> …
>
> Although some epidemiological studies have failed to identify an increased risk of lung cancer among brake mechanics, some have …
>
> Data in Australian Mesothelioma Register – which records all mesotheliomas in a nation of almost 20,000,000 people – constitute the strongest evidence for an increased risk of mesothelioma among brake mechanics who ground and chamfered new brake pads/linings/blocks."

19 Professor Henderson said, in a passage quoted in the primary judge's reasons[16]:

> "When there are multiple episodes of asbestos exposures, and the individual concerned inhales increasing number 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 inter-action between the asbestos fibres and the mesothelial cells by way of secondary chemical [messengers].
>
> 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 progress goes on,

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16 [2010] NSWDDT 8 at [25].

> with increasing numbers of mesothelial cells interacting with increasing number of fibres, so that the ultimate development of mesothelioma, and its probability of development, will be influenced by the number 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."

Dr Heiner gave evidence to similar effect, which was accepted by his Honour[17]. Similarly, Dr Leigh, in a passage quoted by his Honour, said[18]:

> "the current consensus view is that asbestos is involved in both the initiation phase and the promotion/proliferation phase of mesothelioma tumour development".

20 His Honour quoted and clearly accepted Professor Henderson's important observation that it is "almost universally accepted that all asbestos exposure, both recalled and unrecalled, will contribute causally towards the ultimate development of a mesothelioma"[19]. Professor Musk had spoken of cumulative exposure increasing the risk of contracting mesothelioma. His Honour observed that in cross-examination he "did not … resile from his evidence", that where a mesothelioma had occurred all exposure had materially contributed to its development and that this was the case with Mr Booth[20]. His Honour referred to Dr Leigh's testimony that because of the capacity of asbestos fibres to be involved at several stages of tumour development, all cumulative exposure to asbestos fibre must play some part in "causation" in an individual case[21]. His Honour noted, however, that Dr Leigh agreed that if there had been no other exposure, the childhood exposure or the exposure as a truck driver, either separately or in combination, would have been sufficient to cause Mr Booth's mesothelioma[22].

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17 [2010] NSWDDT 8 at [31]-[32].

18 [2010] NSWDDT 8 at [34].

19 [2010] NSWDDT 8 at [26].

20 [2010] NSWDDT 8 at [27].

21 [2010] NSWDDT 8 at [35].

22 [2010] NSWDDT 8 at [37].

21 The primary judge rejected, on the basis of the medical testimony, the theory that mesothelioma can be caused by a single fibre of asbestos[23]. He held that the mechanical theory of the aetiology of mesothelioma which was based on physical properties of asbestos, the chemical theory and the "complete carcinogen theory" were complementary[24]. His Honour said[25]:

> "At issue between the parties in this case is the proposition that all exposure to chrysotile asbestos, other than trivial or *de minimis* exposure, that occurred in a latency period of between 26 and 56 years, materially contributed to the cause of Mr Booth's mesothelioma. I resolve that issue in favour of the plaintiff."

Amaca and Amaba submitted that there was no basis in the evidence for that resolution of the issue.

22 Amaca and Amaba relied, in the Tribunal, upon 19 epidemiological studies published in peer reviewed journals about the incidence of mesothelioma among automotive mechanics and three "meta-analyses" which had combined the results of several studies to produce what was said to be "a more precise estimate of the risk."[26] Each of the meta-analyses concluded that the epidemiological data showed that automotive mechanics are not at a greater risk of developing mesothelioma. The primary judge observed that the studies relied upon by the meta-analyses covered "motor mechanics", "garage workers" and "vehicle mechanics". His Honour said that the average exposure of motor mechanics might have "little in common with the particular exposure of Mr Booth."[27]

23 His Honour accepted Dr Leigh's criticisms of the epidemiological evidence to the effect that many of the studies on which the meta-analyses were based were themselves based on "weak case reference design, and low statistical

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23 [2010] NSWDDT 8 at [48]-[50].

24 [2010] NSWDDT 8 at [51].

25 [2010] NSWDDT 8 at [59].

26 [2010] NSWDDT 8 at [70]. Meta-analysis has been described as a new sub-science involving "the analysis of epidemiological analysis to enable the results of epidemiological studies of different types and of different validity to be combined to produce overall conclusions": Freckelton, "Epilogue: Dilemmas in Proof of Causation", in Freckelton and Mendelson (eds), *Causation in Law and Medicine*, (2002) 429 at 444.

27 [2010] NSWDDT 8 at [75].

power."[28] His Honour rejected, as not justified by the data, the unqualified statement, in the meta-analysis by Goodman et al, that the epidemiological data showed that "[e]mployment as a motor mechanic [did] not increase the risk of developing mesothelioma"[29]. Dr Leigh had argued that much of the data did support a strong correlation between exposure to asbestos as an auto mechanic and the contraction of mesothelioma[30]. His Honour concluded his consideration of the epidemiological evidence with a negative finding[31]:

> "I am not persuaded that the epidemiological evidence specific to automotive mechanics is adverse to the submission that causation has been proved in this particular case."

This may be taken as a finding that the epidemiological evidence did not displace the inference of factual causation which was open on the basis of Mr Booth's history and the medical evidence relating to the cumulative effects of exposure to asbestos.

24 The occupational hygienists called by Amaca and Amaba, Messrs Pickford and Rogers, sought to quantify Mr Booth's reported exposure to asbestos fibres from various sources. His Honour accepted Mr Pickford's expertise in that respect, but concluded that its application had been unfair[32]. Mr Pickford's initial estimates had made no allowance for exposure to background asbestos fibres in the workshop generated by the work of others and by cleaning. He had also been misinformed as to the duration of the work involved in loading asbestos in 1959[33]. His Honour did not accept Mr Rogers' evidence save for his estimate of the fibre concentration to which Mr Booth was exposed when loading bags of asbestos on to his truck[34].

25 After an extended discussion of the hygienists' estimates, his Honour made adjustments to them. He recast a table of lifetime risks prepared by

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28 [2010] NSWDDT 8 at [76].

29 [2010] NSWDDT 8 at [80].

30 [2010] NSWDDT 8 at [81].

31 [2010] NSWDDT 8 at [82].

32 [2010] NSWDDT 8 at [97].

33 [2010] NSWDDT 8 at [98].

34 [2010] NSWDDT 8 at [153].

Professor Berry, on the basis of the hygienists' estimates. The recast table was as follows[35]:

Lifetime Risks of Mesothelioma per Million

Brake Repairs	**Home Renovations**		**Loading Trucks**	
	50% amosite	15 x 4% = 0.6		
18 x 170% = 30.6	50% crocidolite	93 x 4% = 4	Chrysotile	29 x 0.5% = 0.15

His Honour found that the brake repair work undertaken by Mr Booth "increased the background causal component of 70 per million lifetime risks by a further 30.6 per million lifetime risks" and said that "[e]xpressed in terms of cause, the brake work increased by approximately 44 per cent that fibre burden which comprised the background risk."[36] His Honour regarded that contribution as "material."[37]

26 By way of a qualified conclusion from his quantitative findings, his Honour said[38]:

> "Although I do not think that the mathematics are necessarily compelling, if it were necessary to assign mathematical weight to the exercise, the products of Amaca were responsible for 70 per cent (the proportion of Amaca products) of 33 per cent (the proportion of the 27 years of brake work exposure) of 44 per cent (the excess accumulation of fibre burden beyond background exposure) which equals 10 per cent of the additional fibre burden beyond background which caused Mr Booth's mesothelioma.
>
> Upon the same basis, the products of Amaba were responsible for 70 per cent of 66 per cent of 44 per cent, which equals 20 per cent of the additional fibre burden."

In so saying his Honour accepted that the causal contribution of Amaba's products was probably somewhat less than indicated by the simple apportionment he had undertaken because of the greater potency of the earlier

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35 [2010] NSWDDT 8 at [133].

36 [2010] NSWDDT 8 at [137].

37 [2010] NSWDDT 8 at [138].

38 [2010] NSWDDT 8 at [166]-[167].

period of exposure to Amaca's products. He found, however, that the adjustment would be relatively minor[39]. His Honour concluded that[40]:

> "asbestos dust liberated from brake linings manufactured by each of the defendants Amaca and Amaba materially contributed to Mr Booth's contraction of mesothelioma."

As was to be pointed out later in the Court of Appeal, the qualified way in which the primary judge made his quantitative finding of fibre burden attributable to Amaca and Amaba, indicated that he did not rely upon the figures as part of his essential reasoning[41].

The Court of Appeal's reasons

27 The judgment of the Court of Appeal was given by Basten JA, with whom Beazley and Giles JJA agreed. It is necessary only to refer to that part of the reasoning of Basten JA which dealt with the question of causation.

28 Amaca and Amaba submitted in the Court of Appeal that the primary judge failed to apply the correct legal test to the question of causation. Basten JA characterised the correct test "at a high level of generality" as "no more than whether or not the respondent established on the balance of probabilities that, in respect of each appellant, exposure to inhalation of asbestos liberated from its products materially contributed to his injury."[42] Adversely to Amaca and Amaba, his Honour found that the evidence called by Mr Booth at trial was capable of supporting a finding of causation by reference to a scientific theory tested and accepted according to scientific method and secondly, by reference to the epidemiological evidence.

29 His Honour held that findings as to the cumulative effect of exposure to asbestos were open. Mr Booth's medical witnesses had sought to reconcile that view with the epidemiological studies which suggested there was no increased risk in the case of brake mechanics. It was open to the primary judge to accept their evidence and he did. The proposition advanced by Amaca and Amaba that the epidemiology was conclusive against Mr Booth's contention, did not give rise to a question of law but to a question of fact, which the primary judge had

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39 [2010] NSWDDT 8 at [168].

40 [2010] NSWDDT 8 at [172].

41 (2011) Aust Torts Reports ¶82-079 at 64,623 [133].

42 (2011) Aust Torts Reports ¶82-079 at 64,615 [84].

resolved against them[43]. This was not a case in which the plaintiff had relied solely upon epidemiological evidence.

30 His Honour held:

- 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 Mr Booth's injury[44];

- the evidence which the primary judge had accepted distinguished between the risk and the event. The concept of risk looks prospectively. If risk materialises, a causal connection may be inferred[45].

31 The primary judge did not rely upon fibre burden figures attributable to Amaca and Amaba (or thereby upon quantitative risk assessments) as part of his essential reasoning. Basten JA quoted the primary judge's key findings as to causation set out in the introduction to these reasons and said correctly[46]:

> "It was these findings which had to be shown, both to be erroneous and in respect of a point of law."

The appellants' contentions

32 The appeals to the Court of Appeal pursuant to s 32 of the Act were limited to questions of law. The grants of special leave were limited, in effect, to the question of the sufficiency of the evidence, taken as a whole, to support the finding by the primary judge that Mr Booth's exposures to the chrysotile asbestos in the brake linings manufactured by Amaca and Amaba had been causes of his mesothelioma.

33 The grants of special leave did not authorise a review of the correctness of the primary judge's findings of fact. It was not open to Amaca, for example, to advance the argument which it put in its written submissions that "[t]he 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." In any event, the function of the primary judge was to decide the case on the evidence before him, not on

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43 (2011) Aust Torts Reports ¶82-079 at 64,616 [90].

44 (2011) Aust Torts Reports ¶82-079 at 64,621 [118].

45 (2011) Aust Torts Reports ¶82-079 at 64,621 [119].

46 (2011) Aust Torts Reports ¶82-079 at 64,623 [134].

some asserted global consensus. Nor was it open to Amaba to offer arguments about the weight to be attributed to evidence given by the medical experts, including what was, given the confined nature of these appeals, the gratuitous suggestion that Dr Leigh's testimony as to the cumulative effect theory was "partly driven by an interest to reform the law in this area."[47]

34 Both Amaca and Amaba argued that the evidence of the medical witnesses was focussed on "risk" rather than "cause" and that there was a slippage between those two terms in their testimony. Professor Henderson, it was said, used the terminology of "cause" when in fact he was speaking of "risk" referable to a population of persons. Amaca offered a similar interpretation with respect to the evidence of Professor Musk. The nature of its interpretation was encapsulated in its submission that:

> "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."

Amaba characterised Professor Musk as rejecting the cumulative effect theory.

35 Dr Heiner was said by Amaca to have framed his evidence in terms of risk and likelihood rather than in terms of causation. He had made clear that medical science could only draw conclusions as to risk in populations as a whole. Amaca submitted that there was nothing in his evidence to support 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. Amaba made a submission to like effect.

36 In a similar vein, Amaca argued that Dr Leigh's testimony did not amount to evidence that every fibre to which a person is exposed plays some role in the development of mesothelioma. Amaba said that Dr Leigh was the "strongest proponent of what might be called the 'cumulative effect' theory". It acknowledged his statement that:

> "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."

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47 A suggestion footnoted by reference to what was said to be Dr Leigh's disagreement with this Court's decision in *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111; [2010] HCA 5.

Dr Leigh had referred to the "current consensus view" that asbestos is involved or can have effects at both the initiation and promotion phase and the proliferation phase of tumour development. Amaba submitted that his testimony was internally inconsistent and that his evidence supporting the cumulative effect theory fell short of offering any basis for it.

37 On its interpretations of the evidence of the medical witnesses, Amaca argued that there was no basis for the trial judge's 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. The effect of the expert evidence was said to be that while various exposures to asbestos had been shown by reference to what occurs 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 its development or when or why. Amaba submitted that "[p]roperly analysed" none of the medical witnesses supported the cumulative effect theory. Rather, they were suggesting that an increase in exposure to asbestos would increase the risk of mesothelioma.

38 Mr Booth's submissions drew attention to aspects of the evidence of each of the medical witnesses which supported the primary judge's findings and were, for the most part, referred to in his Honour's reasons. The submissions also pointed to Professor Henderson's evidence on specific causation, in respect of which he said:

> "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."

And further:

> "Given that [Mr Booth's] total cumulative brake-dust derived from chrysotile-tremolite exposure made a significant proportional causal contribution towards the development of his mesothelioma, it is also my opinion that the dust derived from the proportions set forth in paragraph 13 on page 14 (Amaca/Amaba brake materials) made a significant causal contribution towards the development of his mesothelioma, as a substantial fraction of his total brake dust-derived chrysotile-tremolite exposure."

Professor Henderson had also noted that his consultation and referral files included many cases of pleural malignant mesothelioma for which chrysotile-

tremolite only exposure derived from new brake linings was the only identified pattern of exposure.

39 Both Amaca and Amaba criticised the primary judge's quantitative findings in relation to the percentage of additional fibres to which Mr Booth was exposed by reason of his work with their products. Amaba, which carried the principal burden of that argument, referred to the primary judge's findings that Amaca was responsible for 10 per cent of Mr Booth's additional fibre burden beyond background and that Amaba was responsible for 20 per cent of the additional burden, and that each had materially contributed to the injury. Apart from criticising the calculations carried out by the primary judge, Amaba argued that his Honour had found causation by reference to "a small increase in risk." Amaba invoked *Amaca Pty Ltd v Ellis*[48] to contend that the small increase in risk found by his Honour could not support a finding on the balance of probabilities that exposure to its products had been a cause of Mr Booth's mesothelioma.

40 It should be said immediately that the present case is not of the kind considered in *Amaca Pty Ltd v Ellis*. In that case the evidence of a very limited exposure to asbestos coupled with epidemiological evidence simply did not support an inference that asbestos exposure was a factual cause of the deceased person's fatal lung cancer. In particular, and by way of contrast with the present case, it was not argued in *Amaca Pty Ltd v Ellis* that it could be concluded, independently of epidemiological analysis, that exposure to asbestos was a cause of the cancer[49]. It is necessary now to consider the relationship between risk and causation in the circumstances of this case.

Risk of harm and factual causation

41 Causation in tort is not established merely because the allegedly tortious act or omission increased a risk of injury[50]. The risk of an occurrence and the cause of the occurrence are quite different things[51]. That proposition is obvious enough and not determinative of these appeals.

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48 (2010) 240 CLR 111.

49 (2010) 240 CLR 111 at 131 [47].

50 *Roads and Traffic Authority v Royal* (2008) 82 ALJR 870 at 898 [144] per Kiefel J; 245 ALR 653 at 689; [2008] HCA 19.

51 Rizzo, "Foreword: Fundamentals of Causation", (1987) 63 *Chicago-Kent Law Review* 397 at 403: "A rise in the probability (frequency) of an outcome may be *evidence* of causation. It is not the causal phenomenon itself" (emphasis in original); *Restatement Third, Torts: Liability for Physical and Emotional Harm* §28, Reporters' Note at 432-433. See generally Wright, "Proving Causation:

(Footnote continues on next page)

42 It is necessary, nevertheless, to reflect upon the relationship between risk and causation. In ordinary usage "risk" refers to a hazard or danger or the chance or hazard of loss[52]. Assessment of the risk of an occurrence is prospective in character. It can be expressed as an ex ante probability that the occurrence will occur. If quantifiable, that probability may be expressed numerically as a figure greater than "zero" up to "one" which denotes certainty. The range of probabilities may be traversed by terms such as "mere possibility", "real chance", "more likely than not", "highly likely" and, ultimately, "certainty".

43 The existence of an association or a positive statistical correlation between the occurrence of one event and the subsequent occurrence of another may be expressed as a possibility, which may be no greater than a "real chance" that, if the first event occurs, the second event will also occur. The mere existence of such an association or correlation does not justify a statement, relevant to factual causation in law, that the first event "creates" or "gives rise to" or "increases" the probability that the second event will occur. Such a statement contains an assumption that if the second event occurs it will have some causal connection to the first. However, if the association between two events is shown to have a causal explanation, then the conclusion may be open, if the second event should occur, that the first event has been at least a contributing cause of that occurrence. An after-the-event inference of causal connection may be reached on the civil standard of proof, namely, balance of probabilities, notwithstanding that the statistical correlation between the first event and the second event indicated, prospectively, no more than a "mere possibility" or "real chance" that the second event would occur given the first event. There may of course be cases in which the strength of the association, as measured by relative risk ratios, itself supports an inference of a causal connection[53].

44 In coming to his view, expressed in his report of March 2009, that epidemiological studies had demonstrated quite conclusively that chrysotile has the capacity to induce malignant mesothelioma, Professor Henderson applied the "Bradford Hill criteria". They were set out by Sir Austin Bradford Hill in an

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Probability versus Belief", in Goldberg (ed), *Perspectives on Causation*, (2011) 195 at 207 ff.

52 *The Shorter Oxford English Dictionary*, 3rd ed (1973) at 1837.

53 See, eg, *Seltsam Pty Ltd v McGuiness* (2000) 49 NSWLR 262 at 278-285 [102]-[137] per Spigelman CJ; Freckelton, "Epilogue: Dilemmas in Proof of Causation", in Freckelton and Mendelson (eds), *Causation in Law and Medicine*, (2002) 429 at 452-453.

address in 1965, a copy of which was in evidence before the primary judge[54]. The criteria were expressed as the aspects of an association between two variables that should be considered before inferring that the most likely interpretation of the association is causation. In summary, they are:

- strength of association – eg, reflected in the ratio of the death rates between groups exposed to a suspected agent and those not so exposed;
- consistency in the observed association – eg, has it been repeatedly observed by different persons in different places, circumstances and times;
- the specificity of the association – if the association is limited to specific workers and particular sites and types of disease and there is no association between the work and other modes of dying, that is a strong argument in favour of causation;
- temporality – the temporal relationship of the variables;
- biological gradient – whether the association reveals a biological gradient or dose-response curve;
- plausibility – whether the expected causation is biologically plausible – a consideration which depends upon the biological knowledge of the day;
- coherence – the cause and effect interpretation of the data should not seriously conflict with the generally known facts of the natural history and biology of the disease;
- experiment – whether experimental or semi-experimental evidence supports a causation hypothesis;
- analogy – eg, given the effects of thalidomide and rubella it is easier to accept slighter but similar evidence with another drug or another viral disease in pregnancy.

The nine factors referred to by Sir Austin Bradford Hill were not presented in his paper as necessary conditions of a cause and effect relationship. They have the character of circumstantial evidence of such a relationship[55].

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54 "The Environment and Disease: Association or Causation?", (1965) 58 *Proceedings of the Royal Society of Medicine* 295.

55 Freckelton notes that these criteria were proposed when epidemiology was "a very young science" and correctly observes that "they do not displace the need for rigorous, scientific scrutiny of the individual items of epidemiological evidence

(Footnote continues on next page)

45 In a discussion of the application of the Bradford Hill criteria in the *Restatement Third, Torts*, it was said[56]:

> "Whether an inference of causation based on an association is appropriate is a matter of informed judgment, not scientific methodology, as is a judgment whether a study that finds no association is exonerative or inconclusive. No algorithm exists for applying the Hill guidelines to determine whether an association truly reflects a causal relationship or is spurious. Because the inferential process involves assessing multiple unranked factors, some of which may be more or less appropriate with regard to a specific causal assessment, judgment is required."

46 Applying the Bradford Hill factors in his report of March 2009, Professor Henderson said that the epidemiological data were inconclusive for brake lining workers specifically, but had also shown quite conclusively that chrysotile has the capacity to induce pleural malignant mesothelioma. A dose-response relationship had been demonstrated for non-brake chrysotile exposures, although not for brake lining exposures. The causal relationship was supported by experimental studies and also from the perspective of biological plausibility. Temporality was fulfilled, as was reasoning by analogy. On that basis Professor Henderson said:

> "This being so, it is my conclusion from pathobiological principles that substantial or protracted chrysotile (chrysotile-tremolite) exposure to dust derived from new (non heat-altered) brake linings probably does have the capacity to induce mesothelioma in dedicated brake mechanics. One of the problems with epidemiological studies on this issue is that they do not clearly distinguish between dedicated brake mechanics versus general automotive mechanics or garage mechanics."

In answer to the question posed for his opinion – Does exposure to dust derived from brake linings that contain chrysotile asbestos have the capacity to induce mesothelioma? – he wrote:

> "Accordingly, my response … is cautiously in the affirmative, 'on the balance of probabilities'. This opinion is not given at a high order of confidence because of the controversy over this issue in the scientific

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brought to court": Freckelton, "Epilogue: Dilemmas in Proof of Causation", in Freckelton and Mendelson (eds), *Causation in Law and Medicine*, (2002) 429 at 444.

56 *Restatement Third, Torts: Liability for Physical and Emotional Harm* §28, Comment c(3) at 406-407.

> literature at present. However, 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." (emphasis in original)

47 The distinction between a statistical correlation and factual causation precedes any consideration of the distinction between factual causation and legal causation which was discussed in *March v E & M H Stramare Pty Ltd*[57]. Factual causation which can be established by the application of the "but for" test is "the threshold test for determining whether a particular act or omission qualifies as a cause of the damage sustained."[58] That threshold must also be surmounted in the case of concurrent or successive tortious acts[59]:

> "it is for the plaintiff to establish that his or her injuries are 'caused or materially contributed to' by the defendant's wrongful conduct … Generally speaking, that causal connexion is established if it appears that the plaintiff would not have sustained his or her injuries had the defendant not been negligent".

48 The threshold requirement still holds good in Australia[60]. As appears from the 10th edition of *Fleming's The Law of Torts*[61]:

> "The first inquiry involves the factual question whether the relation between the defendant's breach of duty and the plaintiff's injury is one of cause and effect in accordance with objective notions of physical sequence. If such a causal relation does not exist, the plaintiff has no actionable claim in negligence. To impose liability for loss to which the defendant's conduct has not contributed is incompatible with the principle of individual responsibility upon which the law of torts is based." (footnotes omitted)

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57 (1991) 171 CLR 506; [1991] HCA 12.

58 (1991) 171 CLR 506 at 530 per McHugh J.

59 (1991) 171 CLR 506 at 514 per Mason CJ, Toohey and Gaudron JJ agreeing at 524-525.

60 *Tabet v Gett* (2010) 240 CLR 537 at 578 [112] per Kiefel J, Hayne and Bell JJ agreeing at 564 [65], Crennan J agreeing at 575 [100]; [2010] HCA 12; *Chappel v Hart* (1998) 195 CLR 232 at 255 [62] per Gummow J, 281-282 [111] per Hayne J; [1998] HCA 55; *Bennett v Minister of Community Welfare* (1992) 176 CLR 408 at 412-413 per Mason CJ, Deane and Toohey JJ; [1992] HCA 27.

61 (2011) at 227 [9.20].

Factual causation does not require that the propounded cause be one link in a chain of causative factors or events. It may be, as some commentators have suggested, a "necessary element of a sufficient set" of causes[62].

49 In summary, a finding that a defendant's conduct has increased the risk of injury to the plaintiff must rest upon more than a mere statistical correlation between that kind of conduct and that kind of injury. It requires the existence of a causal connection between the conduct and the injury, albeit other causative factors may be in play. As demonstrated by medical evidence in this case and in particular by Professor Henderson's evidence, a causal connection may be inferred by somebody expert in the relevant field considering the nature and incidents of the correlation. The Bradford Hill criteria provide a guide to the kind of considerations that lead to an inference of causal connection. As noted above[63], they may include reference to relative risk ratio as an indicator of the strength of the association. Where the existence of a causal connection is accepted it can support an inference, in the particular case, when injury has eventuated, that the defendant's conduct was a cause of the injury. Professor Henderson offered that inference of specific causation by reference to Mr Booth's exposure to the products of both Amaca and Amaba. Where such an inference is drawn, the probability that it is correct is not to be determined only by reference to epidemiologically based ex ante probabilities. In *Betts v Whittingslowe*[64], Dixon J employed apposite logic when he said:

> "the breach of duty coupled with an accident of the kind *that might thereby be caused* is enough to justify an inference, in the absence of any sufficient reason to the contrary, that in fact the accident did occur owing to the act or omission amounting to the breach of statutory duty." (emphasis added)

That logic encompasses the case of an ex ante probability, of accident given breach, supported by a causal explanation linking breach and accident. In this case an explanatory causal mechanism was proposed in the medical evidence.

50 Lord Reid applied similar logic in *Gardiner v Motherwell Machinery and Scrap Co Ltd* when he said[65]:

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62 Wright, "The NESS Account of Natural Causation: A Response to Criticisms", in Goldberg (ed), *Perspectives on Causation*, (2011) 285.

63 At [43].

64 (1945) 71 CLR 637 at 649; [1945] HCA 31.

65 [1961] 1 WLR 1424 at 1429; [1961] 3 All ER 831 at 832. See also [1961] 1 WLR 1424 at 1429 per Lord Cohen and 1430 per Lord Hodson agreeing with Lord Reid's reasons; [1961] 3 All ER 831 at 833.

> "when a man who has not previously suffered from a disease contracts that disease after being subjected to conditions likely to cause it, and when he shows that it starts in a way typical of disease caused by such conditions, he establishes a prima facie presumption that his disease was caused by those conditions."

51 It is enough for present purposes to say that an inference of factual causation, as against both Amaca and Amaba, was open on the evidence before the primary judge. The cumulative effect mechanism involving all asbestos exposure in causal contribution to the ultimate development of a mesothelioma had been propounded and was accepted by his Honour. It depended upon an understanding of physiological mechanisms. It did not depend upon the epidemiology. Whether or not medical science in the future vindicates or undermines that theory, is not to the point. That is not a question which can be agitated on these appeals. The cumulative effect mechanism, accepted by his Honour, implicated the products of both Amaca and Amaba in the development of Mr Booth's disease. The primary judge's interpretation of the expert evidence and his conclusions from it, were open as a matter of law.

52 It is not necessary in this case to consider the application of any modified concept of causation of the kind developed in *Fairchild v Glenhaven Funeral Services Ltd*[66]. That concept was, as Lord Phillips of Worth Matravers PSC pointed out in *Sienkiewicz v Greif (UK) Ltd*[67], a response to "ignorance about the biological cause of the disease" which rendered it "impossible for a claimant to prove causation according to the conventional 'but for' test", a result which would have caused injustice to claimants[68]. In those cases, legal causation was extended beyond the limits of factual causation. In the result, a new head of tortious liability appears to have been created. The understanding of the aetiology of mesothelioma in *Fairchild* did not encompass the cumulative effect mechanism accepted by the primary judge in this case. In *Sienkiewicz,* Lord Phillips observed that[69]:

> "The possibility that mesothelioma may be caused as the result of the cumulative effect of exposure to asbestos dust provides a justification, even if it was not the reason, for restricting the *Fairchild/Barker* rule to cases where the same agent, or an agent acting in the same causative way,

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66 [2003] 1 AC 32.

67 [2011] 2 WLR 523; [2011] 2 All ER 857.

68 [2011] 2 WLR 523 at 531 [18]; [2011] 2 All ER 857 at 865.

69 [2011] 2 WLR 523 at 556 [104]; [2011] 2 All ER 857 at 890.

> has caused the disease, for this possibility will not exist in respect of rival causes that do not act in the same causative way."

53 The present case proceeds upon the foundation of findings, based on evidence before the primary judge, that Mr Booth's exposure to the chrysotile asbestos in brake linings manufactured by Amaca and Amaba not only prospectively increased the risk of his contracting the disease but, in the event, causally contributed to its development and continuation.

Conclusion

54 For the preceding reasons the appeals must be dismissed with costs.

55 GUMMOW, HAYNE AND CRENNAN JJ. These appeals from the New South Wales Court of Appeal (Beazley, Giles and Basten JJA)[70] were heard together. The appellant in the first appeal ("Amaca") was formerly named James Hardie & Coy Pty Ltd. In 1962 it entered into a joint venture with a British company, Ferodo Ltd, and they became the shareholders in the appellant in the second appeal ("Amaba"), then named Hardie-Ferodo Pty Ltd. However, no case has been presented to the effect that by reason of that relationship, Amaca is fixed with the tortious liabilities of Amaba.

The course of the litigation

56 The first respondent, Mr Booth, was born in Sydney in 1937. Between 1953 and 1962, Mr Booth worked as a motor mechanic using brake linings which contained asbestos manufactured by Amaca. Between 1962 and 1983 (excluding the period from 1969 to mid-1971 when he was doing other work), Mr Booth worked as a brake mechanic and was exposed to asbestos in Amaba products. He also worked with brake linings produced by other manufacturers but he estimated that about 70 per cent to 75 per cent of the brake linings with which he worked were the product of Amaca or Amaba.

57 Many years later, in 2008, Mr Booth was diagnosed with malignant pleural mesothelioma, and he instituted proceedings in negligence against Amaca and Amaba in the Dust Diseases Tribunal of New South Wales ("the Tribunal"). He alleged, inter alia, failure by Amaca and Amaba to warn in respect of the use of their brake linings. The Tribunal is established, as a court of record, by s 4 of the *Dust Diseases Tribunal Act* 1989 (NSW) ("the Act"). Any evidence that would be admissible in proceedings in the Supreme Court of New South Wales is admissible in Tribunal proceedings (s 25(1)).

58 The effect of s 3B(1)(b) of the *Civil Liability Act* 2002 (NSW) is that the provisions of Pt 1A, Div 3 thereof, headed "Causation" and comprising ss 5D and 5E, did not apply to the proceedings instituted by Mr Booth in the Tribunal. Accordingly, no reliance was placed by any party to this litigation upon that legislation.

59 On 10 May 2010, the Tribunal (Curtis DCJ) entered judgment against Amaca and Amaba in the sum of $326,640[71]. The Tribunal found that both

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70 *Amaba Pty Ltd (Under NSW Administered Winding Up) v Booth; Amaca Pty Ltd (Under NSW Administered Winding Up) v Booth* (2011) Aust Torts Reports ¶82-079.

71 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8.

companies had "failed to discharge their duty to warn Mr Booth of the dangers of asbestos, and that it is because of this failure that he has contracted mesothelioma". No issue arises on these appeals of any apportionment between Amaca and Amaba; Mr Booth recovered judgment for the full sum.

60 Section 32 of the Act confers a right of appeal to the Supreme Court from a decision of the Tribunal "in point of law" or "on a question as to the admission or rejection of evidence". Section 48(2)(f) of the *Supreme Court Act* 1970 (NSW) assigned the appeal to the Court of Appeal, the Tribunal being a "specified tribunal" as defined in s 48(1)(a).

61 Each of Amaca and Amaba appealed to the Court of Appeal. The appeals were heard together, and on 10 December 2010, they were dismissed. The grants of special leave by this Court were limited to alleged error by the Court of Appeal in holding that Mr Booth's condition was caused by an act or omission on the part of Amaca and Amaba.

62 Thus, as was the situation in the appeal to this Court in *Amaca Pty Ltd v Ellis*[72], neither the existence of duty nor breach of duty is in issue in these appeals. The central question is whether the Court of Appeal was correct in concluding that the Tribunal had not erred "in point of law" when deciding that, in respect of each appellant, it is more probable than not that its negligence was a cause of the contraction, by Mr Booth, of his disease.

63 An inference of fact, concerning the contraction of disease by Mr Booth, which was reasonably open on the evidence, will not manifest error "in point of law"[73]. In any event, the Court of Appeal assumed in favour of Amaca and Amaba that they were entitled to an appeal by way of a rehearing, but nevertheless dismissed the appeals.

Causation

64 It first should be emphasised that, as Windeyer J observed in *The National Insurance Co of New Zealand Ltd v Espagne*[74], the notion of cause and consequence "is a necessary element in law, especially in the law of crime and

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72 (2010) 240 CLR 111 at 122 [10]; [2010] HCA 5.

73 *Australian Broadcasting Tribunal v Bond* (1990) 170 CLR 321 at 356, 365, 369; [1990] HCA 33.

74 (1961) 105 CLR 569 at 593; [1961] HCA 15.

tort". Two issues commonly arise: first, the identification of the cause or causes of a particular occurrence or state of affairs; and, secondly, whether a legal right or liability is engendered by any one or more of those outcomes[75].

65 For a long period, matters of cause and consequence were said to be questions of fact for decision by the jury. In civil actions, for example, questions of cause and consequence arose on the issue joined on the pleaded averment that commonly commenced with the word "whereby"[76]. Hence the attraction in saying that questions of cause and consequence are to be decided by the jury applying "common sense" to the facts of each particular case[77]. The invocation of the "common sense" of the jury discredited judicial directions containing theoretical analysis and exposition[78]. So it was said in *Fitzgerald v Penn*[79]:

> "as soon as one attempts such analysis or exposition, one must enter on a field which is not really appropriate for exploration by a jury".

66 Further, the absolute defence of contributory negligence, as Mason CJ put it in *March v Stramare (E & M H) Pty Ltd*[80], provided a fertile source of confusion in the development of the common law. His Honour added[81]:

> "The existence of the defence, as well as the absence of any mechanism for apportionment of liability as between a plaintiff guilty of contributory negligence and a defendant and as between co-defendants who were concurrent tortfeasors, was a potent factor in inducing courts to embrace a view of causation which assigned occurrences to a single cause. So long

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75 French, "Science and judicial proceedings: Seventy-six years on", (2010) 84 *Australian Law Journal* 244 at 250.

76 *The National Insurance Co of New Zealand Ltd v Espagne* (1961) 105 CLR 569 at 590.

77 *Fitzgerald v Penn* (1954) 91 CLR 268 at 277-278; [1954] HCA 74.

78 cf *Adeels Palace Pty Ltd v Moubarak* (2009) 239 CLR 420 at 440 [43]; [2009] HCA 48.

79 (1954) 91 CLR 268 at 278.

80 (1991) 171 CLR 506 at 511; [1991] HCA 12.

81 (1991) 171 CLR 506 at 511.

as contributory negligence remained a defence, the adoption of this approach was more likely to produce just results."

67 However, this reasoning has lost some of its force with the decline in many jurisdictions in the trial by jury of civil actions, and the removal of contributory negligence as an absolute defence. Further, many issues of causation, including those recently considered in *Lithgow City Council v Jackson*[82] and those which arise on the present appeals, lie outside the realm of common knowledge and experience. They fall to be determined by reference to expert evidence, for example, medical evidence. In such cases, the investigation of difficult and complicated facts cannot be separated from an appreciation of any special branch of knowledge which affects them.

68 Speaking in September 1933 to the Medico-Legal Society of Melbourne, a month after delivery of judgment by the High Court in *Australian Knitting Mills Ltd v Grant*[83], Sir Owen Dixon referred to the extensive category of legal liabilities in which causation forms a chief element; he added that the field covered by the general statement of the law of negligence is enormous and, further, that the wealth of knowledge put by science at the disposal of the processes of the law meant that, in place of what in simpler times had been "the rough and ready answers of the practical man", an exact and reasoned solution now was required[84].

69 Even if the issue is one to which other disciplines may not be able to give any conclusive answer, questions of causation, as a step in the ascertainment of rights and the attribution of liability in law, call for sufficient reduction to certainty to satisfy the relevant burden of proof for the attribution of liability[85]. In *Tubemakers of Australia Ltd v Fernandez*[86], Mason J, with the concurrence of Barwick CJ and Gibbs J, referred to a statement by Dixon J as elaborating the general onus which lies upon the plaintiff where the issue of causation lies

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82 (2011) 85 ALJR 1130 at 1146 [66], 1149 [81]; 281 ALR 223 at 243, 247; [2011] HCA 36.

83 (1933) 50 CLR 387; [1933] HCA 35.

84 "Science and Judicial Proceedings", in Woinarski (ed), *Jesting Pilate*, (1965) 11 at 14. See further, French, "Science and judicial proceedings: Seventy-six years on", (2010) 84 *Australian Law Journal* 244 at 246-247.

85 *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111 at 121-122 [6].

86 (1976) 50 ALJR 720 at 724; 10 ALR 303 at 311.

outside the realm of common knowledge and experience. In *Adelaide Stevedoring Co Ltd v Forst*[87], Dixon J said:

> "I think that upon a question of fact of a medical or scientific description a court can only say that the burden of proof has not been discharged where, upon the evidence, it appears that the *present state of knowledge* does not admit of an affirmative answer and that competent and trustworthy expert opinion regards an affirmative answer as lacking justification, *either as a probable inference or as an accepted hypothesis*." (emphasis added)

70 The "but for" criterion of causation proved to be troublesome in various situations in which multiple acts or events led to the plaintiff's injury[88], for example, where the development of a particular medical condition was the result of multiple conjunctive causal factors. In such cases what may be unclear is the extent to which one of these conjunctive causal factors contributed to that state of affairs. These situations have been addressed by the proposition stated by Lord Watson in *Wakelin v London and South Western Railway Co*[89] that it is sufficient that the plaintiff prove that the negligence of the defendant "caused or materially contributed to the injury"[90]. In that regard, reference may be made to the well-known passage in the speech of Lord Reid in *Bonnington Castings Ltd v Wardlaw*[91]. Of that case it was said in the joint reasons in *Amaca Pty Ltd v Ellis*[92]:

> "The issue in *Bonnington Castings* was whether exposure to silica dust from poorly maintained equipment caused or contributed to the pursuer's

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87 (1940) 64 CLR 538 at 569; [1940] HCA 45. This statement may be compared with the passage in *Australian Knitting Mills Ltd v Grant* (1933) 50 CLR 387 at 426 in which Dixon J declined to act on the evidence of the chemist called by the plaintiff.

88 *March v Stramare (E & M H) Pty Ltd* (1991) 171 CLR 506 at 516-517.

89 (1886) 12 App Cas 41 at 47.

90 See *March v Stramare (E & M H) Pty Ltd* (1991) 171 CLR 506 at 514 per Mason CJ; *Athey v Leonati* [1996] 3 SCR 458 at 466-468 per Major J; Tse, "Tests for factual causation: Unravelling the mystery of material contribution, contribution to risk, the robust and pragmatic approach and the inference of causation", (2008) 16 *Torts Law Journal* 249 at 252-256.

91 [1956] AC 613 at 621.

92 (2010) 240 CLR 111 at 136 [67].

> pneumoconiosis, when other (and much larger) quantities of silica dust were produced by other activities at the pursuer's workplace. Those other activities were conducted without breach of duty. As Lord Reid rightly pointed out[93], the question in the case was not what was the most probable source of the pursuer's disease: dust from one source or the other. The question was whether dust from the poorly maintained equipment was *a* cause of his disease when the medical evidence was that pneumoconiosis is caused by a gradual accumulation of silica particles inhaled over a period of years." (emphasis in original)

71 It should be emphasised that the resolution of the issue before this Court in *Ellis* does not govern the issues in the present appeals. *Ellis* involved alternative causes of the plaintiff's lung cancer, asbestos inhalation and inhalation of tobacco smoke; the plaintiff had not shown that it was more probable than not that exposure to asbestos had made a material contribution to his cancer[94]; but the evidence in the present case, to which further reference will be made, was that, unlike the situation regarding lung cancer, exposure to asbestos is effectively the only known cause of mesothelioma.

The evidence

72 The state of medical and scientific knowledge concerning what may be sufficient exposure to asbestos to engender mesothelioma may develop as further study is made. This advancing state of knowledge may be reflected in the evidence given from one case to the next. What is taken, in one case, to be a proposition of law derived from the attribution of legal liability, or its absence, may require consideration of the particular state of the evidence from which the court reduced a question of causation to the relevant standard of legal certainty.

73 In the present litigation the following matters were not in dispute: (1) Mr Booth's mesothelioma was caused by inhalation of asbestos fibre; (2) chrysotile asbestos has the capacity to cause mesothelioma; (3) the brake linings manufactured by the appellants contained chrysotile asbestos; and (4) Mr Booth inhaled chrysotile asbestos from the appellants' products.

74 Curtis DCJ found that 70 per cent of the asbestos fibres to which Mr Booth was exposed over the period between 1953 and 1962 were released from Amaca products and the same percentage of Amaba products represented

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93 [1956] AC 613 at 621.

94 (2010) 240 CLR 111 at 135 [65].

exposure in the period between 1962 and 1983 (excluding the period he was doing other work). His Honour also found that his exposure from other activities, including home renovations when he was a child, were insignificant, trivial or *de minimis*.

75 Section 25B of the Act provides that, without leave of the Tribunal, "[i]ssues of a general nature" determined by the Tribunal may not be relitigated or reargued in other Tribunal proceedings, whether or not they are between the same parties; in deciding to grant leave, the Tribunal is to have regard to the availability of new evidence, whether or not previously available. The primary judge stated that, for the purposes of s 25B, he determined 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".

76 Mr Booth relied upon, and Curtis DCJ accepted, the expert evidence of Professor Douglas Henderson (Professor of Pathology at Flinders University), Dr James Leigh (a consultant occupational physician), Dr Maurice Heiner (a consultant thoracic physician) and Professor William Musk (Clinical Professor of Medicine at the University of Western Australia). Writing extra-judicially, Sir Owen Dixon described the three true functions of such witnesses as follows[95]:

> "First, to provide the court with the abstract knowledge which is requisite in order to understand and use the considerations which should determine its decision upon the scientific questions involved.
>
> Second, to collate and describe the facts, scientifically material, which the witness has obtained.
>
> Third, to state his own conclusions and opinions, and the grounds upon which he has formed them."

77 Several points respecting this evidence should be noted. The first is that the appellants called no expert clinicians, rather relying upon cross-examination of the four experts called by Mr Booth and upon Professor Geoffrey Berry, a biostatistician and epidemiologist. The second is that Professor Henderson, Dr Heiner and Professor Musk had each encountered cases of mesothelioma where the only identified exposure to asbestos was from working with brake linings. Professor Musk said in evidence that he had "seen brake lining exposed

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95 "The Law and the Scientific Expert", a paper delivered in 1934 and reprinted in Woinarski (ed), *Jesting Pilate*, (1965) 24 at 34.

mechanics with mesothelioma who [did not] appear to have had significant other exposure". Professor Henderson concluded his written report of 2 March 2009:

> "I would also emphasise that my 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."

78 The third point concerns what, in *Fairchild v Glenhaven Funeral Services Ltd*[96], Lord Bingham of Cornhill said was the state of medical knowledge in about 2000 respecting the cause of mesothelioma:

> "the condition 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, and the condition once caused is not aggravated by further exposure".

The reasons of the Court of Appeal in that litigation had included the passage[97]:

> "It was therefore common ground on these appeals that it could not be said whether a single fibre of asbestos was more or less likely to have caused the disease, alternatively whether more than one fibre was more or less likely to have caused the disease. In the latter event, it could not be shown that it was more likely than not that those fibres came from more than one source. In other words, none of these scenarios could be proved on the balance of probabilities. Similarly, it could not be proved on the balance of probabilities that any one man's mesothelioma was caused cumulatively by exposure to asbestos dust in more than one employment."

79 The "single fibre" theory was not accepted in the evidence in the present case as representing current expert opinion. In the course of his cross-examination, Dr Leigh said of the proposition that mesothelioma could be generated from a single fibre that this was not physically possible. In his evidence-in-chief Professor Henderson gave a long answer to a question that he explain his statement that each of multiple asbestos exposures contributes to the causation of mesothelioma. His answer included the following:

> "[W]hen there are multiple episodes of asbestos exposures and the individual concerned inhales increasing numbers of fibres on different

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96 [2003] 1 AC 32 at 43.

97 *Fairchild v Glenhaven Funeral Services Ltd* [2002] 1 WLR 1052 at 1064.

> 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 messengers[. A]nd to simplify the answer, the point is that the more fibres there are the greater number of fibres there will be interacting with mesothelial cells which themselves undergo proliferation and so the progress 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[. A]nd 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."

Finally, it should be noted that the witnesses were appreciative of the need to indicate the relative degrees of strength of the conclusions they reached. For example, Professor Henderson expressed "at a high order of confidence" his opinion that chrysotile has the capacity to cause malignant mesothelioma; and, "cautiously ... 'on the balance of probabilities'", his opinion that exposure to dust derived from brake linings which contain chrysotile asbestos has the capacity to cause mesothelioma.

The United Kingdom authorities

80 The expert evidence in the present case shows that the limits in medical knowledge disclosed by the (now discredited) "one fibre" theory accepted in the evidence in *Fairchild* have been removed by further advances in medical science. However, in the United Kingdom the decision in *Fairchild* has left in place a common law principle, now supplemented by a statutory regime[98], designed to bridge what Professor Jane Stapleton has called an "evidentiary gap"[99]. The problem of legal coherence which thus is presented was recognised in *Fairchild* by Lord Rodger of Earlsferry when he observed[100]:

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98 *Compensation Act* 2006 (UK).

99 "Factual Causation and Asbestos Cancers", (2010) 126 *Law Quarterly Review* 351 at 356.

100 [2003] 1 AC 32 at 97.

> "In future more may be known. As Mr Stewart rightly observed, in the course of submissions that were both helpful and sensitive, this may change the way in which the law treats such cases. But the House must deal with these appeals on the basis of the evidence as to medical knowledge today and leave the problems of the future to be resolved in the future."

81 In considering the recent decision of the United Kingdom Supreme Court in *Sienkiewicz v Greif (UK) Ltd*[101] it is important to appreciate the statement by Lord Phillips of Worth Matravers PSC[102]:

> "The special rule of causation applied to mesothelioma was devised because of ignorance about the biological cause of the disease. It was accepted in *Fairchild* and *Barker*[103] that this rendered it impossible for a claimant to prove causation according to the conventional 'but for' test and this caused injustice to claimants. It is not possible properly to consider the issues raised by this appeal without reference to what is known about mesothelioma. This has been summarised in many cases, and much of my own summary in *Bryce v Swan Hunter Group plc*[104] of what was known 25 years ago remains true today. *The cases under appeal 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.*" (emphasis added)

82 The case which Amaca and Amaba were required to meet thus differed significantly in its evidentiary foundation from that in *Fairchild* and in *Sienkiewicz.*

Conclusions

83 Mr Booth developed his case in the following steps: (1) he had contracted mesothelioma; (2) the only known cause of that disease is exposure to asbestos; (3) the expert evidence at trial, accepted by the primary judge, was that: (a) exposure to asbestos contributes to the disease; and (b) the prospective risk of contracting the disease increases with the period of significant exposure;

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101 [2011] 2 WLR 523; [2011] 2 All ER 857.

102 [2011] 2 WLR 523 at 531; [2011] 2 All ER 857 at 865.

103 *Barker v Corus UK Ltd* [2006] 2 AC 572.

104 [1988] 1 All ER 659.

(4) Mr Booth had two periods of significant exposure; (5) it is more probable than not that each period of exposure made a material contribution to bodily processes which progressed to the development of the disease.

84 The response of the appellants was to emphasise that step (3) did not make proper allowance for the epidemiological evidence which they had presented. The appellants relied upon 19 studies upon the incidence of mesothelioma in automotive mechanics. These had been published in peer reviewed literature. In particular, the appellants relied upon three analyses of the literature, by Wong, Goodman and others, and Laden and others.

85 For example, Wong concluded that "there is no evidence to support or even to suggest an association between an increased risk of mesothelioma and exposure to brake linings or clutch facings among garage mechanics". However, in the course of his cross-examination, Professor Berry said that although Wong had found "no significant evidence of effect", for himself he accepted that there might be some risk due to chrysotile exposure as a result of working with brakes, "for example drilling holes in them to make them fit the car".

86 The discipline of epidemiology, and its application in answering issues of causation in litigation, was described by Lord Phillips in *Sienkiewicz* as follows[105]:

> "Epidemiology is the study of the occurrence and distribution of events (such as disease) over human populations. It seeks to determine whether statistical associations between these events and supposed determinants can be demonstrated. Whether those associations if proved demonstrate an underlying biological causal relationship is a further and different question from the question of statistical association on which the epidemiology is initially engaged.
>
> Epidemiology may be used in an attempt to establish different matters in relation to a disease. It may help to establish what agents are capable of causing a disease, for instance that both cigarette smoke and asbestos dust are capable of causing lung cancer, it may help to establish which agent, or which source of an agent, was the cause, or it may help to establish whether or not one agent combined with another in causing the disease."

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105 [2011] 2 WLR 523 at 551; [2011] 2 All ER 857 at 885.

Lord Mance JSC left for consideration on another occasion the question whether "epidemiological evidence can by itself prove a case"[106], that is to say, a plaintiff's case. *Sienkiewicz* was decided on other grounds, namely, that as a matter of law *Fairchild* applied[107].

87 In the present case, the plaintiff, Mr Booth, did not challenge the reception of epidemiological evidence, represented principally by studies in published papers, which was tendered by the defendants. Rather, his attitude in this Court was close to that of Lord Mance in *Sienkiewicz*[108], namely that such evidence can be admissible and relevant but its weight will depend upon the nature of the evidence and the particular factual issues before the court.

88 The epidemiological evidence, considered by itself, did leave open the inference that cumulative exposure to asbestos increased the risk of contracting mesothelioma by developing bodily processes to an irreversible point. Further, as Dr Leigh emphasised in his report, inability to demonstrate epidemiologically a statistically significant increase in risk in motor mechanics, relative to other occupational categories, does not, in any way, negate a causal inference in an individual case where, beyond the general background environment, the only asbestos exposure was incurred in that occupation.

89 Professor Henderson accepted that epidemiological data respecting work with brake linings was inconclusive. But he wrote in his report that "[o]ne of the problems with epidemiological studies on this issue is that they do not clearly distinguish between dedicated brake mechanics versus general automotive mechanics or garage mechanics". That report further stated that a dedicated brake mechanic includes one "who frequently machined/ground new and not heat-altered brake linings". Mr Booth had done grinding work throughout the periods in question. Professor Henderson also discounted the epidemiological data for other deficiencies in the methodology employed. Dr Leigh, who is trained in epidemiology, gave what the primary judge described as cogent evidence, criticising the methodology and case design upon which many of the studies were based.

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106 [2011] 2 WLR 523 at 583; [2011] 2 All ER 857 at 916.

107 Laleng, "*Sienkiewicz* v *Greif (UK) Ltd* and *Willmore* v *Knowsley Metropolitan Borough Council*: A Material Contribution to Uncertainty?", (2011) 74 *Modern Law Review* 777 at 788-790.

108 [2011] 2 WLR 523 at 583; [2011] 2 All ER 857 at 916.

90 It was open to the primary judge to decide that he was "not persuaded that the epidemiological evidence specific to automotive mechanics is adverse to the submission that causation has been proved in this particular case".

91 The Court of Appeal, with respect, correctly concluded[109]:

> "Findings as to the cumulative effect of exposure to asbestos were undoubtedly open. [Mr Booth's] witnesses, including Professor Henderson and Dr Leigh, sought to reconcile that approach with the epidemiology which suggested there was no increased risk in the case of brake mechanics. It was open to his Honour to accept their evidence, as he did. The underlying proposition put forward by the appellants, that the epidemiology was conclusive, in accordance with the principles applicable to such evidence, did not give rise to a question of law, but to a question of fact, which his Honour resolved against the appellants."

Orders

92 Each appeal should be dismissed with costs.

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109 *Amaba Pty Ltd (Under NSW Administered Winding Up) v Booth; Amaca Pty Ltd (Under NSW Administered Winding Up) v Booth* (2011) Aust Torts Reports ¶82-079 at 64,616.

93 HEYDON J. Mesothelioma is a painful illness leading to death. It is a cancer of the lining of the lung. It is very commonly caused by inhaling asbestos fibres, though perhaps not always. It can be caused by very brief intense exposures whether occupational, domestic or recreational, and by lower-level environmental exposures – sometimes after exposures which are very short – a day – or very slight. On the other hand, many people can have heavy and sustained exposures to even the most dangerous types of asbestos without suffering the disease. This phenomenon, like much else about the disease, is something which scientists have found difficult to explain. The disease has a latency period of at least 10 years, and sometimes much longer – as long as 75 years. The disease is often not diagnosed until many years after exposure to asbestos. It is therefore difficult for plaintiffs suffering from mesothelioma to establish the facts necessary for success in negligence actions. In particular it can be difficult for them to establish that the conduct of a given defendant caused the disease. A related difficulty for plaintiffs springs from the fact that the earlier the exposure the greater the chance that it could cause harm. Because of the valuable characteristics of asbestos, particularly its capacity to retard fires, it has been commonly used until quite recently. The extent of exposure to asbestos amongst those now living, the likely exposure amongst those yet to be born, and the likelihood of further injury taking place when asbestos is removed from the many places where it is now found, mean that problems of the kind thrown up in these appeals will remain for decades to come. Perhaps a social-medical problem of this size requires a legislative solution. In some places solutions have been sought in judicial or legislative changes to the law relating to causation. New South Wales is not one of those places. In New South Wales a special court called the Dust Diseases Tribunal has been established. It has attracted considerable admiration for the energy it throws into the urgent resolution of controversies involving dying plaintiffs. But it is bound by the general rules of causation in negligence. The question which these appeals raise is whether the Tribunal's causation findings in this case, and cases like it, are supported by any evidence.

The factual background

94 In 2008 John William Booth ("the plaintiff"), then aged 71, was diagnosed as having pleural mesothelioma. It was probably caused by inhaling asbestos fibres. The plaintiff probably inhaled asbestos fibres from four sources.

95 The first source comprised those asbestos fibres which exist as part of "the background … that pervades urban environments"[110]. This "background risk" or "background level" is the sum of all exposures to asbestos fibres which those

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110 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [162] (2) per Judge Curtis.

suffering from mesothelioma cannot attribute a specific cause to, either because they did not identify the cause or because they could not remember having been exposed. The proportion of those suffering from mesothelioma who cannot identify any prior exposure to asbestos is 15%-30%. One estimate of the risk of an Australian contracting mesothelioma without any specific exposure to asbestos that can be recalled is 70-140 per million per lifetime of 70 years.

96 The second source comprised asbestos fibres released into the atmosphere when the plaintiff, aged 8, was holding down asbestos sheeting which his father cut with fibro cutters in order to build a house, and those released when the plaintiff, aged 16, helped his father in cutting and handling asbestos cement sheets they were using to build a garage. There was evidence that once dust containing asbestos is introduced into domestic premises – for example, dust from renovation work – it can persist: it is difficult to remove the fibres even by vacuuming, and everyday activity can cause the fibres to be resuspended and to persist in the air for considerable periods.

97 The third source comprised asbestos fibres released into the atmosphere when the plaintiff had to load hessian bags of pure asbestos onto his truck on the Sydney wharves. This may have been amphibole asbestos including crocidolite – a much more dangerous form of asbestos than chrysotile.

98 The fourth source comprised those asbestos fibres to which the plaintiff was exposed while working on brakes during the 27 years he spent working as a motor mechanic. The fibres in this category came in part from brake parts manufactured by Amaca Pty Ltd ("Amaca"), in part from brake parts manufactured by Amaba Pty Ltd ("Amaba") and in part from brake parts manufactured by other manufacturers. The linings in brakes on which the plaintiff worked contained asbestos. The process of replacing brakes released asbestos into the atmosphere. The plaintiff worked on Amaca brake linings from 1953 to 1962. He worked on Amaba brake linings from 1962 to 1969 and from 1971 to 1983. Although some of those who have worked on brake linings have contracted mesothelioma, most have not. Over a 16 year period (1986-2001) 78 sufferers from mesothelioma had brake lining exposure (compared to 38 who lived in asbestos dwellings and 85 who built or renovated asbestos dwellings, and 5,546 notifications overall); in 1997 there were 83,000 vehicle mechanics, of whom many would have worked with brake linings.

99 All asbestos is dangerous. But the products manufactured by Amaca and Amaba contained the least dangerous type of asbestos, chrysotile. The trial judge found that the combined lifetime risk created by the home renovations, the loading of the truck with asbestos, and brake repair work was 31.4 per million (of which the brake exposure contributed 97%) if the asbestos used in home renovations was chrysotile and 35 per million (of which the brake exposure contributed 87%) if it was crocidolite.

100 The trial judge found that 30% of the fibres to which the plaintiff was exposed came from sources other than Amaca or Amaba over the periods in which he was a mechanic. The trial judge found that the brake repair work done by the plaintiff increased the background risk by 44%. As a result of calculations which Amaca and Amaba challenge[111], the trial judge found that the asbestos for which Amaca was responsible increased the background risk of mesothelioma by 10%, that the asbestos for which Amaba was responsible increased the background risk of mesothelioma by a little less than 20%, and that an increase in risk of these magnitudes "materially contributed" to the contracting of mesothelioma by the plaintiff. It follows that despite the dusty nature of the brake repair work on which the plaintiff laid stress, which the trial judge set out and which the trial judge no doubt took into account, the respective contributions of Amaca and Amaba were much less than the background risk.

101 Further, if the respective contributions of Amaca and Amaba were compared with all other exposures (ie background, home renovations, truck loading, and those for which other brake manufacturers were responsible) those contributions would be even lower.

102 One causation difficulty created by these facts is that, leaving aside the exposures for which Amaca and Amaba were responsible, any of the groups of asbestos fibres to which the plaintiff was exposed either alone or in combination with others could have caused his disease. Another is that there was no evidence as to when the plaintiff contracted the disease – ie when the asbestos fibres injured him by causing changes in the lungs and pleura which were irreversible and led him later to display the symptoms of mesothelioma.

103 It follows that to prove causation against Amaca, for example, the plaintiff had to prove two things. First, that the exposures before he became a motor mechanic in 1953 had not caused the irreversible changes in his body which led him later to display the symptoms of mesothelioma. Secondly, that some of the fibres to which he was exposed as a brake repairer were Amaca fibres (as distinct from the fibres of other brake manufacturers), and that they caused those changes in his body. Alternatively, he had to prove that even if the Amaca fibres to which the plaintiff had been exposed did not cause those changes up to 1962, after 1962 there were exposures to fibres which, in combination with Amaca fibres, caused those changes.

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111 They said that the calculations purported to be based on Professor Berry's estimate that background exposure corresponded to a lifetime risk of 70 per million: in fact Professor Berry's figure was 70-140 per million. And they said that it was wrong to compare the contributions of Amaca and Amaba with background risk only, rather than with all other sources of risk.

The nature of the appeals

104 An appeal only lies to the Court of Appeal from a decision of the Dust Diseases Tribunal on a question of law. It is an error of law to make a material finding which is not supported by any evidence. However, an appeal on a "no evidence" point in this type of case is difficult to succeed in. It must necessarily be conducted in a much more restricted way than the trial which gave rise to the appeal. At the trial Amaca and Amaba relied positively on epidemiological evidence. But in these appeals positive reliance on their own evidence could bring them little aid: they had to concentrate on what they said were gaps in the plaintiff's evidence. The "no evidence" issue was a narrow one, from which the parties' submissions often strayed.

The primary argument of Amaca and Amaba

105 The key issue is not whether chrysotile *can* cause mesothelioma. Nor is it whether chrysotile dust from brake linings *can* cause it – a question to which Professor Henderson gave a "response … cautiously in the affirmative, 'on the balance of probabilities'." At least in this Court, Amaca and Amaba did not dispute the proposition that chrysotile *can* cause mesothelioma and that chrysotile dust from brake linings *can* do so. They did not dispute that partly because the evidence supported the proposition, and partly because it was in their interests to rely on evidence that all forms of exposure to asbestos can cause mesothelioma. Nor did Amaca and Amaba dispute the proposition that particular instances encountered by some of the experts were instances of mesothelioma caused by exposure to brake linings. What Amaca and Amaba did dispute was the following finding of the trial judge: "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 [sic] to the cause of mesothelioma."[112] In particular, they contended, as they had to, that there was no evidence to support that finding, which was an essential step to the trial judge's conclusion that the plaintiff's exposure to the asbestos in brake linings materially contributed to his mesothelioma. The significance of the finding goes well beyond this particular case. That is because the trial judge preceded that finding with the words: "I specifically determine for the purpose of s 25B that". Section 25B of the *Dust Diseases Tribunal Act* 1989 (NSW) prevents this issue from being re-litigated in other cases without leave. In short, however erroneous the trial judge's finding may be as a matter of fact, unless it can be demonstrated that there was no evidence to support it, later litigants will be bound by it without having been heard in relation to it. Amaca and Amaba also submitted that there was no evidence that the asbestos exposure for which they were responsible was a cause of the plaintiff's mesothelioma, as distinct from it being caused by other exposures.

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112 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [62].

106 Amaca and Amaba relied on evidence that no epidemiological study had ever shown that motor mechanics were at an increased risk of mesothelioma from brake work. One study supporting that view stated that "auto mechanics do not have an increased risk of malignant mesothelioma as a result of exposure to asbestos fibers from brake linings and clutch facings." The trial judge said that statements of that kind in the study were correct but misleading. He did not, however, give reasons for that view. The trial judge concluded by saying[113]:

> "I am not persuaded that the epidemiological evidence specific to automotive mechanics is adverse to the submission that causation has been proved in this particular case."

Apart from reversing the burden of proof, this passage did not say that there was epidemiological evidence favourable to causation.

107 The "no evidence" battle was largely fought on a different field. One step in the trial judge's reasoning was his conclusion that while inhalation of a single fibre of asbestos could not cause mesothelioma, the four experts called by the plaintiff "are each of the opinion that all asbestos fibres contribute to the development of a mesothelioma."[114] Amaca and Amaba submitted that although fragments of the evidence of each expert considered in isolation might be thought to support the trial judge's finding – words like "cause" and "made a material contribution" appear – as a whole their evidence did not and the fragments were to be read down in that light. Amaca and Amaba therefore referred to the various parts of the evidence of each expert which qualified the evidence on which the trial judge seemed to have relied.

108 The plaintiff attacked the submissions of Amaca and Amaba as depending on selective quotation from the experts' evidence. The plaintiff submitted that the evidence of each expert read more fully did support the trial judge's conclusion. The questions whether Amaca and Amaba are right that the pieces of evidence relied on by the trial judge read in context do not support him, or whether the plaintiff is right that on any view they do support him, are questions only to be answered by reading the evidence as a whole. In view of the conclusion reached by other members of the Court it would be unduly wasteful of space to analyse every piece of evidence to which the parties pointed. It is necessary to evaluate what the experts meant by the various verbal formulae they used. The plaintiff's citation of evidence was fuller than that of Amaca and Amaba, but the latter were not misleadingly selective. Further, to some extent

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113 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [82].

114 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [52].

the parties were not squarely at issue, for the plaintiff concentrated on whether asbestos can cause mesothelioma, while Amaca and Amaba concentrated on whether every exposure to asbestos (and in particular the plaintiff's exposure to asbestos from brake linings) contributed to mesothelioma. With that background, it is necessary to go to some of what the four experts said.

Professor Henderson's evidence

109 The trial judge quoted the following evidence of Professor Henderson: "It is, I think, almost universally accepted that all asbestos exposure, both recalled and unrecalled, will contribute causally towards the ultimate development of a mesothelioma"[115]. However, the next answer which Professor Henderson gave revealed that the "phenomenon that [he was] describing" was that as "cumulative exposure increases, so does the *risk* of mesothelioma" (emphasis added). Professor Henderson continued: "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" (emphasis added). Amaca correctly submitted that by "risk" Professor Henderson meant consequences which might come home against a population of persons as a whole – not "cause" the particular plaintiff's mesothelioma. The Court of Appeal set out the answer quoted by the trial judge, and said it provided a basis for the conclusion that all exposures contributed to the mesothelioma suffered by the plaintiff. It did not refer to the subsequent evidence qualifying and explaining the answer[116].

110 The correctness of Amaca's submission is supported by the fact that the oral evidence was given in the context of page 15 of Professor Henderson's report of 2 March 2009. Professor Henderson there said:

> "*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

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115 The evidence was inadmissible: it was given in answer to a leading question in chief to which the cross-examiner objected.

116 See below at [119]-[120].

> acceptable latency interval contributes ***causally*** towards the development of mesothelioma." (bold emphasis added)

Amaca submitted:

> "The words 'it follows' in the bullet point, by linking the second sentence with the first, emphasised 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."

The plaintiff criticised the submissions on the ground that the word "population" did not appear in the passage quoted from page 15 of Professor Henderson's report. But the word and the idea appeared in other passages[117] relevant to the line of thought being developed in that passage. Amaca then drew attention to the following passages from Appendix A (which dealt with "The Scientific/Medical Evidence for Causation of Malignant Mesothelioma by Asbestos"):

> "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*: ie, 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." (publication references omitted; emphasis added)

111 The Peto model gives the "relationship between asbestos exposure and the risk/incidence of mesothelioma". It reveals the number of cases of mesothelioma one would expect to see within a population of persons who bear the characteristics of exposure and latency reflected in the formula. For example, it might reveal that for persons suffering particular intensities of exposure over particular periods with particular latency, there will be 10 cases of mesothelioma per million persons per year. A little later Professor Henderson said:

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117 Quoted below at [114].

> "*No minimum threshold dose of inhaled asbestos has been delineated below which there is no increase in the risk of mesothelioma*, as indicated by the following publications". (emphasis in original)

He then set out numerous publications reflecting the incidence of mesothelioma in a population of defined characteristics either absolutely or relatively to a controlled group of persons who face only background risk. One of these was a Swedish study revealing that some occupations, located entirely or primarily in the country, had a standardised incidence ratio of less than 0.5 – persons in farming, gardening, religious, forestry and food manufacturing occupations. Farmers had the lowest figure of 0.28. The study went on to compare the much higher standardised incidence ratio of city groups not exposed occupationally to asbestos. The studies go on to deal with concepts similar to standardised incidence ratio like "relative risks", "odds ratios" and "proportional mortality ratios". Professor Henderson said that these calculations "for cohort and case-control studies on mesothelioma represent cases *in excess* of any 'background' risk from 'background' exposures" (emphasis in original).

112 Professor Henderson summarised the discussion in Appendix A by saying:

> "In other words, causal attribution of mesothelioma to antecedent asbestos exposure(s) requires evidence that the exposure(s) constituted cumulative exposures in excess of so-called 'background' exposure sustained from the general environment".

That is, he described the risk analysis based on a comparison of particular populations with a control group as a process of "causal attribution". That may be an apt term in science. It may be a useful term in deciding what response there should be from government and employers to public health issues arising from dangers to particular groups of the public. But it is not a usage corresponding with the expression "causation" as used in relation to the legal rule that one particular sufferer from mesothelioma suing in negligence must prove that the disease was caused by an exposure.

113 That is further highlighted by an earlier part of Appendix A:

> "Mesothelioma occurs in only a minority of asbestos-exposed individuals, even in those exposed heavily to amphibole asbestos. This observation might be explicable by mesothelioma induction as a chance event: that is, mesothelioma is the outcome of a multistage process involving multiple mutational and epigenetic events, so that most of those exposed by asbestos simply do not strike the 'correct' combination of a complex set of events necessary for development of mesothelioma. Alternatively one of the mutations induced by asbestos may be lethal to the initiated cell, so that subsequent steps cannot occur. However, alternative explanations include: (i) modulation of the asbestos-imposed risk by genetic or

> acquired susceptibility/resistance factors; or (ii) a combination of randomness and predisposition." (publication references omitted)

Hence Professor Henderson was conscious that while risk analysis enables one to predict how many of a particular group or population will suffer mesothelioma it does not enable one to predict which ones will, or, once the disease is diagnosed, whether the disease in a particular sufferer is the result of a particular exposure or only a background exposure. Very many people who suffer the same exposure do not contract the disease; relatively few do. Professor Henderson is there revealing the incapacity of such analysis to say precisely why those who have contracted the disease have done so. He is revealing that to move from "risk" to "cause" is an impermissible attempt to leap a gap.

114 What Professor Henderson meant by "risk" is also seen in Appendix B to his report – which dealt with "Mesothelioma and Exposure to Asbestos Dust Derived from Brake Linings/Materials (Chrysotile-Only Exposure)". Early in Appendix B there is a section headed "Some Preliminary Remarks on Relative Risk (RR) *Versus* Individual Risk" (emphasis in original). That section amplifies the words quoted above[118] – "the risk is not a theoretical construct". In that section Professor Henderson said:

> "*Absolute* associative or causal effects involve assessment of the actual numbers of cases or incidences, whereas *relative* effects involve assessment of ratios: hence RR represents the ratio of the incidence for cases seen in the *exposed* group divided by the incidence for the same disease in the *controls* …
>
> It should also be emphasised that 'risk' in this context is no theoretical construct: instead, it represents the ratio of the incidence rates derived from the actual number of observed cases relative to the control/reference group. 'Rate ratio' would be preferable but 'relative risk' is well entrenched. Because RR is derived as an *average* across a population/group, it is unlikely to correspond to the *individual* risk for each and every individual who makes up the population under study, because individual risks will vary from one individual to another …
>
> In other words, an RR or odds ratio (OR) is essentially a net or average (mean) population-based assessment: although the mean RR/OR value is suitable for public health policy planning and for assessment of causal effects on a population-wide basis, it is quite inappropriate simply to extrapolate the mean RR/OR to each and every individual comprising the population – for the simple reason that biological systems such as human

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118 At [109].

> beings vary in multitudinous different ways." (footnote omitted; emphasis in original)

This passage is centred on the notion of a "population/group". In short, the "relative risk" or "rate ratio" describes the ratio of incidence rates derived from the actual number of observed cases in a population (for example, brake mechanics) compared to a central group (for example, persons with general background exposure to asbestos, but no other exposure). A rate ratio is an average across a group. It will not correspond with the risk to an individual member of a group, which may vary from person to person. The assessment of the risk applying to a particular member of the population is another question; and an assessment of whether illness in a member of the population was caused by the condition giving rise to the risk is yet another question.

115 Professor Henderson said a little later:

> "If one approaches causation of mesothelioma relative to brake dust exposures using *The Bradford-Hill Criteria*, one can state that the epidemiological data are inconclusive for brake lining work specifically, but epidemiological studies have also demonstrated quite conclusively that chrysotile – whether contaminated with tremolite or not – does have the capacity to induce malignant mesothelioma (at least pleural malignant mesothelioma, leaving aside for the moment the issue of peritoneal mesothelioma). In terms of dose-response, epidemiological studies on non-brake chrysotile exposures have demonstrated a dose-response relationship, although this has not been demonstrated clearly for brake lining exposures. The relationship in causal terms is supported by experimental studies, and also from the perspective of biological plausibility. Of course, temporality in this case (and in others) is fulfilled, as is reasoning by analogy (perhaps the weakest of the criteria).
>
> This being so, it is my conclusion from pathobiological principles that substantial or protracted chrysotile (chrysotile-tremolite) exposure to dust derived from new (non heat-altered) brake linings probably does have the capacity to induce mesothelioma in dedicated brake mechanics." (publication reference omitted)

That is a cautious conclusion. But Professor Henderson deals only with capacity in general. He does not purport to say whether a particular brake mechanic's mesothelioma was caused by a particular exposure.

116 At the end of Appendix B he said:

> "[T]he grounds on which I would attribute a significant *causal* contribution to asbestos derived from chrysotile-containing brake linings/pads/blocks include the following". (emphasis added)

The last three grounds he referred to were:

> "Given the no-threshold model for lung cancer induction by asbestos, including chrysotile, exposures above background will, following an appropriate latency interval, confer an increment in *risk* on top of any underlying pre-existing background risk for mesothelioma.
>
> Although some epidemiological studies have failed to identify an increased *risk* of cancer among brake mechanics, some have …
>
> Data in Australian Mesothelioma Register – which records all mesotheliomas in a nation of almost 20,000,000 people – constitute the strongest evidence for an increased *risk* of mesothelioma among brake mechanics who ground and chamfered new brake pads/linings/blocks." (publication reference omitted; emphasis added)

By "causal" contribution, Professor Henderson was referring to increase in risk. And by "brake mechanics" he was referring to a particular population.

117 Professor Henderson also said at the end of Appendix B:

> "causation for any mesothelioma can be considered to represent the sum of the risks for the true spontaneous mesothelioma rate + mesotheliomas related to non-identifiable exposures from the general environment only (ie, 'known no exposure') + mesotheliomas for which there is exposure in excess of 'general environmental' exposure (ie, unrecognised above-'general environmental' exposure + recognised above-'general environmental' exposure – whether occupational (direct/bystander) or non-occupational). That is, the model is one of a cumulative exposure-causal effect model with no threshold, whereby each exposure adds to the risk of any exposures that have gone before and incremental upon any spontaneous ('known no exposure') risk."

That is, used in this way, "causation" refers to the sum of the risks which a person faces. The model does not establish which of the exposures brought about the disease from which a particular person suffers. Some of the risks relate to what disease has occurred across an entire population over a particular period. But the model does not reveal which particular exposure caused mesothelioma in a particular victim.

118 Professor Henderson was asked in chief what he meant by the following statement in Appendix A in his report, which was quoted above[119]:

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119 At [110].

> "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."

Over objection, he answered:

> "Well it goes to the issue of the dose response model for mesothelioma induction by asbestos and that is that 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 messengers and to simplify the answer, the point is that the more fibres there are the greater number of fibres there will be interacting with mesothelial cells which themselves undergo proliferation and so the progress 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". (emphasis added)

As it did of the first answer of Professor Henderson quoted above[120], the Court of Appeal said of this evidence, which the trial judge quoted[121], that it provided a basis for the conclusion that all exposure contributed to the mesothelioma suffered by the plaintiff. The reference to "probability", however, highlights the generality of the testimony: it was evidence of a biological process in relation to how mesothelioma probably develops, but it was not evidence about which exposures caused the plaintiff's mesothelioma, and in particular it was not evidence about whether the exposures for which Amaca and Amaba were responsible caused it.

119 Professor Henderson said that if the plaintiff had had no exposure to asbestos beyond background exposure and the exposure which took place when he helped his father in building operations, those exposures would have "made a very small causal contribution", that is, a "small increase in risk." Professor Henderson gave the following evidence in answer to questions from the trial judge:

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120 At [109].

121 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [25].

> "Q. … 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 mesothelioma develops. And risk is always based on the numbers of cases in the exposed versus unexposed populations.
>
> Q. In the case of [the plaintiff], 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 exposure 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."

The Court of Appeal quoted the last question and answer. That answer was explained in an answer given to the next question asked by counsel:

> "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."

120 As Amaca and Amaba submitted, this reveals that by "cause" Professor Henderson meant nothing more than an identifiable part of the cumulative bundle of risks faced by a person. In saying that "at least a proportion of [the fibres inhaled] will contribute to the risk and to the ultimate development of the mesothelioma", he was not saying that every exposure caused mesothelioma. The plaintiff criticised the submission put by Amaca and Amaba, but did concede that the passage reveals that Professor Henderson was not prepared to say that the plaintiff's mesothelioma was caused by the risk of one particular exposure as opposed to another. That means that it was not evidence of causation.

121 The Court of Appeal then said of the answer it quoted[122]:

> "That evidence, which his Honour effectively accepted, distinguished between the risk and the event. Thus, a person who is in a

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122 *Amaba Pty Ltd (Under NSW Administered Winding Up) v Booth; Amaca Pty Ltd (Under NSW Administered Winding Up) v Booth* (2011) Aust Torts Reports ¶82-079 at 64,621-64,622 [119].

> room containing asbestos dust is at risk of inhaling asbestos fibres. If the risk materialises and the fibre is inhaled, he will be at risk of some fibres lodging in his lung. If that happens, there is a risk that some of those fibres will translocate to the pleura. If that happens, he is at risk of contracting mesothelioma. The concept of 'risk' looks at the matter prospectively; if the risk materialises, a causal connection may be inferred. Professor Henderson's evidence accepted the causal connection at each stage. It was open to his Honour to conclude that Professor Henderson, for example, did not use risk synonymously with cause and to conclude that Professor Henderson did not 'prefer' to describe the state of medical science in terms of risk; indeed, he described 'risk' as 'a very bad term'".

With respect, this analogy is overstretched. It is remote from the present case, for the analogy postulates only one source of risk and one possible cause, while in the present case there are several sources of risk and several possible causes. It was not open to the trial judge to reason as the Court of Appeal said he could because his reasoning rested on a misreading of the evidence.

122 A final piece of evidence from Professor Henderson was his answer in other proceedings to a question about what he meant by "causal contribution from the asbestos exposures in this case". He said:

> "I suppose what I was trying to say was that we know that there is a relationship, or a dose response relationship between asbestos exposure and the likelihood of the development of mesothelioma and that when there are multiple exposures each exposure is considered to add to the overall risk of the development of mesothelioma, so that each exposure will exert an incremental increase in risk on top of background and on top of exposures that have gone before. In terms of the causation, again it comes down to probabilities and I really couldn't do better than to quote from page 4 of Dr James Leigh's report where in dealing with the development of mesothelioma he comments, 'All of these processes at cellular level are [stochastic] in that the probabilities of fibre cell interaction depend on the number of fibres and the number of cells present at any point in time, hence simplistically the more fibres the more free radicals and the greater probability of initiated, promoted or proliferated cells at any given point, at any given time point.' So there is a theoretical basis to explain the increase in risk in terms of the numbers of fibres inhaled and the more fibres that you have the greater the probability that these fibres will interact with mesothelia cells and eventually [lead] by a multistage process to mesothelia."

That shows a sense in which a great increase in risk can lead to a conclusion that mesothelioma will follow. But the passage does not show that all exposures materially contribute to mesothelioma. It could not do so without accounting for

the fact that most asbestos fibres which have been inhaled do no harm, even in people who contract mesothelioma.

123 Professor Henderson's evidence did not support the view that all exposures to chrysotile asbestos materially contribute to mesothelioma.

Professor Musk's evidence

124 The trial judge said[123]:

> "Although at times Professor 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 [the plaintiff]." (emphasis in original)

Professor Musk said in his report:

> "It is my opinion that [the plaintiff's] exposure to asbestos from brake linings manufactured and supplied by Amaca and Amaba … was sufficient to make a material contribution to the development of his mesothelioma because these were the main sources of the asbestos to which he was exposed and the period between exposure and the development of 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."

125 Professor Musk in his oral evidence in chief gave affirmative answers to a number of leading questions to which objection was taken. One was whether he agreed with Professor Henderson's "conclusions concerning causation in this case." Another was whether he considered "that all exposure to asbestos within an acceptable latency period materially contributes to the mesothelioma in a particular patient." A third was whether "all exposure to asbestos within an acceptable latency period materially contributes to the mesothelioma." All these leading questions were impermissible and should have been rejected. The answers were inadmissible.

126 Professor Musk also gave an affirmative answer to the question whether he agreed "with the reasoning that Professor Henderson uses to reach the

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123 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [27].

conclusions that he reaches." It has been seen that Professor Henderson's reasoning is that all exposures to asbestos contribute to the risk that persons so exposed will suffer mesothelioma: it is not that all exposures to asbestos cause mesothelioma in a particular person so exposed. That circumstance suggests that Professor Musk's evidence does not support the trial judge's conclusion any more than Professor Henderson's does. He did, however, decline to concede in re-examination that his understanding of the biological processes leading to mesothelioma was inferior to those of Professor Henderson and Dr Leigh.

127 In cross-examination Professor Musk gave the following evidence:

> "Q. Professor, do you think this is a fair way to express it, that given 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.
>
> Q. Well, that's right, because they infer that in cases [where] the evidence is there in respect of the risk. Is that so. A – Yes. We hardly ever see mesothelioma in the general population and we see it increasingly in people exposed to asbestos and the different varieties of asbestos have a different propensity to cause mesothelioma and the risk increases with time since first exposure after the first 10 to 15 years."

Professor Musk was not asserting that the asbestos exposures for which Amaca and Amaba were responsible caused the plaintiff's mesothelioma. Read as a whole, Professor Musk's evidence is similar to that of Professor Henderson – experience across populations as a whole supports the conclusion that the greater the exposure to asbestos, the greater the risk of mesothelioma – but it does not permit the conclusion that all exposures experienced by any particular individuals in those populations caused mesothelioma in those individuals.

Dr Heiner's evidence

128 The trial judge said[124]:

> "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."

That summary was correct to the extent that it suggested that Dr Heiner's opinion was that the "incidence of mesothelioma" – that is, considered across a population – increases the greater the exposure of individuals within it. But it does not follow that he held the opinion that the mesothelioma suffered by a particular individual was caused by any particular exposure. Dr Heiner's actual evidence is consistent with those points.

129 Thus in chief he gave the following evidence:

> "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. …
>
> 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 continually was 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 … occurring. That's how I understand it."

130 And in cross-examination he gave the following evidence:

> "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

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124 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [31].

> contracting mesothelioma. A – Inhaling asbestos can – 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 upon dose, fibre type and latency periods, what is known that inhaling asbestos can increase the risk of contracting mesothelioma. A – Correct."

But Dr Heiner's evidence is not evidence that the conduct of Amaca and Amaba caused the plaintiff's mesothelioma in law. It is also notable that Dr Heiner denied the "cumulative effect" theory – the theory that all asbestos exposure materially contributes to the development of a particular person's mesothelioma – on which the plaintiff's primary case rested.

Dr Leigh's evidence

131 The trial judge quoted Dr Leigh as saying[125]:

> "the current consensus view is that asbestos is involved in both the initiation phase and the promotion/proliferation phase of mesothelioma tumour development".

The trial judge then said[126]:

> "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 the 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." (emphasis in original)

132 Those remarks appear to be based on the following statement in Dr Leigh's report:

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125 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [34].

126 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [35]-[36].

> "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."

A similar proposition appeared earlier:

> "[inability] to demonstrate epidemiologically a statistically significant increase in risk in an occupational category of work, relative to all other occupational categories does not negate in any way a causal inference in an individual case where the only asbestos exposure, above general background environment, was incurred in that occupation."

But what Dr Leigh meant by these two passages was explained immediately after the first of them as follows:

> "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 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. 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 time point."

133 Thus Dr Leigh was not saying that every exposure of a person suffering from mesothelioma to asbestos caused the mesothelioma. He was saying that the more fibres to which the person was exposed, the greater the chance that they would initiate the disease. He said in his report: "All exposure, recalled and unrecalled or unrecognized, would have contributed cumulatively to the risk of mesothelioma." However, it does not follow that all exposures caused the disease, or that any particular exposure did. The plaintiff submitted that passages in Dr Leigh's evidence similar to the one last quoted provided a "biological basis" for Dr Leigh's view that asbestos exposure cumulatively contributes to cause as well as risk. But those passages do not provide evidence for the view that every non-trivial exposure of a person to asbestos fibres is causative of mesothelioma. Further, there was expert evidence from Professor Henderson and Dr Leigh that the plaintiff's exposures to asbestos prior to the exposures he received from Amaca and later Amaba products were capable of causing mesothelioma on their own. And, as already noted, there was no expert evidence as to when the plaintiff developed mesothelioma.

134 Dr Leigh was asked the following question in chief:

> "Q. … do you consider that all the exposure contributes cumulatively to the cause of mesothelioma.
>
> …
>
> A – All exposure cumulatively contributes to cause as well as risk, as I think his Honour was alluding to. Once the disease has occurred the risk has come home or been expressed."

The question was leading, and it went well outside the witness's report: for those reasons the evidence was inadmissible. The answer, however, was explained in cross-examination. Dr Leigh was asked: "Are you in a position to say that but for the exposure, say, on the wharfs, [the plaintiff] wouldn't have contracted mesothelioma[?]" This was a reference to the plaintiff's exposure to asbestos while loading hessian bags of asbestos onto his truck on the Sydney wharves. Dr Leigh answered: "No, I am not, I'm saying no to that." The evidence continued:

> "Q. Is what you're saying really in effect that the exposure on the wharfs can't be excluded. A – I'm saying that, yes. It can't be excluded as part of the overall causation.
>
> Q. That's because it added to the risk. A – Yes."

135 A little later Dr Leigh gave the following evidence:

> "Q. … Dr Leigh, you can't say that except for the brake work [the plaintiff] wouldn't have got his mesothelioma, can you. A – No, I can't say that.
>
> Q. What you're saying is you can't exclude the brake work. A – Yes."

Two points emerge. First, Dr Leigh often conflated "risk" and "cause". Secondly, the asbestos inhaled during the "brake work" did not satisfy the "but for" test for causation in Dr Leigh's eyes. It followed that the risk was not a cause in law.

136 Evidence of Dr Leigh in another case revealed that, while rejecting the theory that one single asbestos fibre could cause mesothelioma, he believed that the more fibres that were inhaled the greater the chance of the disease. That was because while some might initiate the disease others would promote it, by acting on a number of cells in a "probabilistic" way, thereby increasing the "overall risk". On being asked: "Again it's all a matter of risk and increase of risk?", he answered:

> "Well, it's a matter of the fact that he's got it and he had this exposure, so that you have to assume, you know, from the end point that all the exposure must have had something to do with it, whatever the risk-creating potential was. You have to assume that some of those fibres had something to do with it."

He accepted that there was still a question whether mesothelioma could occur spontaneously without any exposure to asbestos. But after referring to the Peto model, he was asked whether fibres inhaled 40 years ago are "causally much more potent than a similar number of fibres of similar type of asbestos inhaled only 10 years ago". He said: "Statistically, yes, statistically." He was then asked what it meant to say "causally more potent and statistically more potent", and answered:

> "It is statistically because you can't actually say, you know – there is no direct way of knowing which fibre did what to which cell at which time. That's what I mean by stochastic or statistical. So it's not really a question of cause or potency, it's just statistical. It is probability, I think, it's probability."

That is, there is no direct way of discovering what any fibre – from home improvement, or loading asbestos bags, or Amaca or Amaba products, or the general background – did to which cell at which time. Later he gave this evidence:

> "Why must [the handyman exposure] have had something to do with it? – Well, you can't say that it didn't because they were additional fibres. As I said, the whole process is probabilistic. There must have been some probability that those additional fibres had something to do with it. You can't exclude that possibility. You can't exclude that.
>
> If a single fibre may initiate the … cell change is it necessary that there be further fibres to promote the process? – That's a good question. I would say yes. Certainly there needs to be some more – some agents acting to further process. Whether they are further fibres or something else I don't know but I would say yes.
>
> And if one or more fibres have initiated the process commencing with the cell change will the inhalation of further fibres necessarily play some part in the promotion or further promotion of that process? – You can't exclude the possibility that they do. You can't exclude it."

This is evidence of risk and possibility, not of causation.

137 Both Dr Leigh and a work he relied on – Dodson and Hammar (eds): *Asbestos: Risk Assessment, Epidemiology, and Health Effects* – contend that it is possible that multiple asbestos fibres have roles to play in initiating and

developing mesothelioma over a process which takes some time. But they do not amount to evidence that fibres from every exposure over the entire period of exposure more probably than not play a role in causing a particular person to contract mesothelioma.

138 It is necessary to return to what the trial judge said about Dr Leigh. The trial judge's movement from what he quoted from Dr Leigh's evidence to the next two paragraphs of the reasons for judgment[127] rested on invalid reasoning. It was a movement from a statement about risk which was then treated as a statement about cause and which led to a conclusion about cause that was not open. The trial judge said[128]:

> "In cross-examination Dr Leigh agreed that, *if* there had been no other exposure, the childhood exposure, or the exposure as a truck driver, either separately or in combination was sufficient to cause [the plaintiff's] mesothelioma. He further agreed that he could not say that, because of this earlier exposure, [the plaintiff] would not have contracted mesothelioma in the absence of the work on asbestos brake linings." (emphasis in original)

These concessions amount to a denial that causation of the plaintiff's mesothelioma by the exposures for which Amaca and Amaba were responsible has been, more probably than not, established.

Conclusion in relation to the plaintiff's four experts

139 Thus, while there was evidence that examining what happened across populations revealed that a succession of exposures to asbestos increased the cumulative risk of suffering mesothelioma both for the populations as a whole and for individuals within them, the evidence did not show that in the case of a particular individual like the plaintiff it could be said that all exposure to chrysotile asbestos materially contributed to his mesothelioma. Each exposure increased his risk of developing mesothelioma. It does not follow that each exposure caused the mesothelioma. With respect, the Court of Appeal misunderstood the expert evidence by accepting the snippets of it quoted by the trial judge as representative, and by accepting the trial judge's characterisation of it as correct.

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127 See above at [131].

128 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [37].

Professor Berry's evidence

140 The plaintiff submitted that the contention of Amaca and Amaba that there was *no* evidence to support the trial judge's conclusion as to causation was capable of refutation in two ways: by examining the medical evidence alone, or by examining it in combination with other evidence. The first way rested on the proposition that the references by the medical experts to causation meant causation in law, not increased risk. That proposition has just been discussed. The second way contended that even if the medical experts spoke only of increased risk, the trial judge could infer causation from increased risk and other evidence.

141 The plaintiff submitted that there was expert evidence apart from the four experts discussed above supporting the theory that every exposure to asbestos materially contributes to mesothelioma. He said it was to be found in the evidence of Professor Berry, who was called by Amaca and Amaba. The plaintiff submitted: "Professor Berry agrees that it was '… the lifetime load of all asbestos exposure which *causes* the illness in the individual'" (emphasis added). In fact that was a quotation from a question asked by counsel for the plaintiff. Professor Berry did not agree with the suggestion. He answered thus: "it's the total lifetime exposure and the components that make up that total lifetime exposure that *increase the risk*" (emphasis added).

142 The plaintiff also submitted that Professor Berry "testified that [the plaintiff] was undoubtedly at increased risk of contracting mesothelioma from brake work." That is not so. What Professor Berry actually said was: "I certainly wouldn't wish to argue that brake workers were at … lower risk than the general population."

143 Professor Berry did not support the theory that every exposure to asbestos materially contributes to mesothelioma; indeed he thought that in assessing causation it was necessary to know "the relativities of background which is part of the lifetime load, plus the increment".

Causation inferred from risk

144 The plaintiff did submit that even if Professor Henderson's evidence did not support the view that every exposure to asbestos was causative of mesothelioma, but only added to the cumulative risk of mesothelioma, it was open to the trial judge to infer causation from the increased risk of injury. The submission cited authority which did not support it, for it held[129] that an increase

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129 *Roads and Traffic Authority v Royal* (2008) 82 ALJR 870 at 898 [144]; 245 ALR 653 at 689; [2008] HCA 19.

in risk does not by itself support a conclusion of causation. Indeed, Amaba submitted, the trial judge's reasoning proceeded on the opposite view. A key element of the trial judge's reasoning on causation, under the heading "Specifically", was that 70% of the asbestos fibres to which the plaintiff was exposed in 1953-1962 were from Amaca products, and 70% of the asbestos fibres to which the plaintiff was exposed in later years were from Amaba products. This amounted respectively to 10% and 20% of "the additional fibre burden beyond background which caused [the plaintiff's] mesothelioma."[130] The trial judge arrived at these figures thus[131]:

> "Professor Berry says that it may be appropriate to assume that the background exposure of [the plaintiff] to asbestos fibre as a consequence of general low-level concentrations of asbestos in urban air corresponds to a lifetime *risk* of 70 per million.
>
> The brake repair work increased the background *causal* component of 70 per million lifetime risks by a further 30.6 per million lifetime risks. Expressed in terms of *cause*, the brake work increased by approximately 44 per cent that fibre burden which comprised the background risk." (emphasis added)

The reasoning treats "risk" and "cause" as being identical. Amaba's submission is to be accepted.

The trial judge's alternative route to causation

145 The trial judge considered that an[132]:

> "overwhelming inference of causation may be drawn from the following facts:
>
> (1) [The plaintiff's] mesothelioma was caused by the inhalation of asbestos fibre;
>
> (2) Mesothelioma very rarely occurs in persons who have not been exposed to asbestos fibres beyond the background level that pervades urban environments;

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130 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [166].

131 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [136]-[137].

132 *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8 at [162].

> (3) For a total of 27 years, week in and week out, [the plaintiff] was additionally exposed to asbestos fibres liberated from asbestos brake shoes by his own work, and by the work of others in his vicinity,
>
> (4) The previous exposure, in the course of home renovations and truck loading was, in comparison, trivial."

146 Amaca accepted propositions (1) and (2). Amaca attacked proposition (3) on three grounds. It did not discriminate between Amaca-Amaba brake exposure and other brake exposure. It insinuated that the additional exposure referred to was very substantial, when in fact it was only 10% for Amaca and 20% for Amaba, even on the trial judge's controversial calculations. Thirdly, the reasoning did not explain why it should be concluded, more probably than not, than between 1953 and 1962 changes occurred in the plaintiff's body leading to him later developing the symptoms of mesothelioma which were attributable to Amaca's fibres, or that between 1962 and 1969, and between 1971 and 1983, changes occurred in the plaintiff's body leading to him developing the symptoms of mesothelioma which were attributable to Amaca's fibres in combination with other fibres.

147 Amaca also attacked proposition (4). The home renovation could create an additional four cases per million per lifetime: that was not trivial relative to the additional seven cases per million per lifetime for Amaca.

148 Amaca was correct to submit that this alternative route to causation suggested by the trial judge was neither an "overwhelming inference" nor available at all.

The "but for" test

149 The trial judge did not inquire whether the plaintiff had established that but for the Amaca and Amaba exposures he would not have contracted mesothelioma. The "but for" test is a necessary but not sufficient test for causation[133]. There was specific evidence that it was not satisfied[134], and for the reasons given above, there was no evidence that it was satisfied.

Orders

150 Each appeal should be allowed. Order 1 of the Court of Appeal made on 10 December 2010 should be set aside, and, in lieu thereof, each appeal to that

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133 *March v E & M H Stramare Pty Ltd* (1991) 171 CLR 506 at 515-516; [1991] HCA 12.

134 See above at [134]-[135].

Court should be allowed. The order of the trial judge made on 10 May 2010 should be set aside, and, in lieu thereof, there should be verdict and judgment for the defendants. The appellants must pay the costs of the first respondent in the Court of Appeal and in this Court pursuant to a condition on the grants of special leave to appeal.