IN THE HIGH COURT OF AUSTRALIA
SYDNEY REGISTRY

S220
No 87 of 2011

BETWEEN:

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

AND

JOHN WILLIAM BOOTH
First respondent

AMACA PTY LIMITED
(ACN 000 035 521)
(UNDER NSW ADMINISTERED WINDING UP)
Second respondent

APPELLANT'S SUBMISSIONS

PART I: INTERNET PUBLICATION

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

PART II: STATEMENT OF ISSUES

2. Can causation in tort be established by reference to an increase in risk of injury, even a small increase in risk? Did any increase in risk attributable to Amaba cause the first respondent's injury?

3. Is the evidence sufficient to permit acceptance of a particular theory as to how inhaling asbestos causes mesothelioma?

PART III: SECTION 78B OF THE *JUDICIARY ACT 1903*

4. Notice under s78B of the *Judiciary Act 1903* is unnecessary.

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PART IV: REASONS FOR JUDGMENT

5. Both decisions remain unreported. The citations are: at trial – *John William Booth v Amaca Pty Limited* [2010] NSWDDT 8; on appeal – *Amaba Pty Limited v Booth* [2010] NSWCA 344.

PART V: NARRATIVE OF FACTS

The proceedings

6. The first respondent contracted pleural mesothelioma in 2008. Pleural mesothelioma is a cancer of the lining of the lung which is usually caused by inhaling asbestos. The first respondent had been exposed to asbestos in four different ways during his lifetime, and amongst those he was exposed to asbestos while working on brakes in his job as a motor mechanic.

7. The first respondent sued each of Amaca Pty Ltd and Amaba Pty Ltd. Amaca was formerly known as James Hardie & Coy Pty Limited and manufactured brakes from before 1953 and until 1962. Amaba was previously known as Hardie-Ferodo Pty Limited and manufactured brake parts from 1962 until about 1983. The first respondent claimed that negligence of each caused his mesothelioma. Amaca and Amaba defended the proceedings raising issues including causation. After a trial in the Dust Diseases Tribunal, the primary judge, Judge Curtis, found in favour of the first respondent and awarded damages. In particular, the primary judge found that the first respondent's work on both Amaca and Amaba's products caused his mesothelioma. An appeal to the Court of Appeal (Basten JA; Beazley and Giles JJA agreeing) was dismissed.

8. In this appeal it is Amaba's case that, *first*, the evidence was incapable of supporting that conclusion on causation; and, *secondly*, if orthodox principles were applied, the first respondent's case on causation had to fail.

The facts

9. The first respondent was born in 1937. He spent 26 years working as a motor mechanic – from 1953 until 1969, and again from 1973 until 1983. Amongst

the many different tasks he undertook as a motor mechanic, the first respondent removed and replaced brakes on cars and trucks. The linings in brakes contain a proportion of asbestos. Some of the tasks of replacing brakes release asbestos into the atmosphere.

10. The first respondent was exposed to asbestos in four circumstances. One (as described above) was during his work as a mechanic. Another occurred in his childhood, while he was assisting his father in cutting and handling asbestos cement sheets. A third occurred on one day in 1959 when the first respondent was working as a truck driver and was involved in handling bags of pure asbestos. And the fourth exposure – and it is common ground that this is the most significant exposure – occurred throughout his lifetime while the first respondent suffered the same ordinary exposure to asbestos common to all Australians, and which gives rise to a risk of contracting mesothelioma known as "*the background risk*".

11. The background risk of mesothelioma is a key consideration in these proceedings. It is a risk probably generated by breathing the very small levels of asbestos constantly present in every urban atmosphere. The risk is well documented; repeated studies show that after careful investigation between 15% and 30% of mesothelioma sufferers are unable to identify any previous exposure to asbestos. The medical scientists identify the risk faced by this group as the background risk.

12. The risk of contracting pleural mesothelioma can be calculated, and it was calculated for the purpose of this case. The risk is typically expressed in terms of the incidence of the disease for every million persons in the Australian population, over an average lifetime of 70 years (expressed as a risk "*per million, per lifetime*"). The scientific evidence established that the background risk – that is the risk of an Australian contracting mesothelioma in the absence of a recollection of a specific exposure to asbestos – is in the range of 70 to 140 per million, per lifetime[1].

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[1] Professor Berry gave unchallenged evidence to this effect: Ex DX14; Professor Henderson agreed with Professor Berry: T112.16; Dr Leigh said the rate was 133 per million, per lifetime: Ex PX21. There was evidence that the rate could be as high as 330 per million, per lifetime

13. The calculation of the first respondent's risk of contracting mesothelioma from a specific exposure to asbestos depended upon two factors – the likely dose of fibre he inhaled, and the type of asbestos fibre he inhaled. The primary judge made his own calculations of the cumulative dose of asbestos from the various exposures. There are different types of asbestos – crocidolite (blue), amosite (brown) and chrysotile (white) – which carry different risks of mesothelioma: accordingly to a leading authority, the ratio of risk is broadly 500 for crocidolite, 100 for amosite, and 1 for chrysotile. Amaca's and Amaba's brakes contained only the least dangerous type of asbestos – chrysotile.

14. Taking into account his findings, the primary judge found that apart from the background risk, the first respondent's risk emanating from all of the other identified sources (ie: the childhood exposure; the truck driving; and the work with brakes) was 35 per million, per lifetime[2]. The primary judge then found that of this, the risk from all brake work (ie: from Amaca's, Amaba's and the brakes from other manufacturers) was 30.6 per million, per lifetime[3].

15. The primary judge then calculated the specific risk from each of Amaca's and Amaba's products. He found that 30% of the brakes which the first respondent handled came from sources other than Amaca or Amaba[4]. Taking this into account, the primary judge found that the asbestos for which Amaca was responsible increased the background risk of mesothelioma by 10% and Amaba by 20%, and that an increase in risk of this magnitude "*materially contributed*" to the first respondent's contraction of mesothelioma[5].

16. There was medical evidence regarding the causation of mesothelioma. The primary judge found that this evidence supported a conclusion that all exposures to chrysotile asbestos, other than trivial or *de minimis* exposures, materially contribute to the cause of mesothelioma[6]. In practical effect this amounted to acceptance of a medical theory usually described as the "*cumulative effect theory*". On Amaba's submission not only did the medical evidence fail to support such a theory, it contradicted it.

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2 DDT [135]
3 DDT [137]
4 DDT [165]
5 DDT [166], [167] and [172]. There is an error in the primary judge's calculations, and (as explained below) the increase in risk was only 4% and 8% respectfully
6 DDT [62]

PART VI: APPELLANT'S ARGUMENT

17. There are two aspects to the concept of causation as it applies in these proceedings. The first relates to the primary judge's specific findings of causation which where based upon his finding that each of Amaca and Amaba was responsible for a small increase in the risk of injury. This departs from principle and constitutes an error of law. The second aspect of causation is the extent to which the primary judge used the medical evidence as a basis upon which to construct a controversial biological theory – the "*cumulative effect*" theory. There was no evidence to support that theory and the finding in this respect also discloses an error of law.

Causation by reference to an increase in risk

18. It has never been accepted in Australia that causation in tort may be established because some act or omission increased a risk of injury; it has always been essential that the particular risk is found to have come home: see, for example, *Roads & Traffic Authority v Royal* (2008) 82 ALJR 870 per Kiefel J at 898 [144].

19. Here the primary judge decided causation by reference to a small increase in risk. The relevant part of his judgment is headed "*Conclusions on Causation*", and divided into two sections – causation "*Generally*"[7] and causation "*Specifically*"[8]. The primary judge found that Amaca was responsible for "*10% of the additional fibre burden beyond background*"[9] and that Amaba was responsible "*20% of the additional fibre burden*"[10] and each *materially contributed*" to the injury[11].

20. This is sufficient to dispose of the key finding of causation. As this Court explained in *Amaca Pty Limited v Ellis* (2010) 240 CLR 111 at 123 [13] causation is not established unless the facts positively suggest that it was more probable than not that the negligence of either Amaca or Amaba was a cause of the injury. Findings of an increase in risk as low as 10% and 20% compel the

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7 DDT [161]-[162]
8 DDT [163]-[172]
9 DDT [166]; the reference to an "*additional fibre burden*" is the same thing as an additional "*lifetime risk*" – see DDT [137]
10 DDT [167]
11 DDT [172]

conclusion that it was improbable that either Amaca or Amaba caused this injury.

21. In this sense, the result in these proceedings can be contrasted with the result delivered on a strikingly similar set of facts in *Sienkiewicz v Greif (UK) Ltd* [2011] 2 WLR 523, where the deceased's inhalation of asbestos had increased the risk of her contracting mesothelioma by 18%[12] when compared with the background risk. In those circumstances it was accepted that the claim for damages had to fail unless there was some amelioration of the existing causation rule – that is a, by further extension of the "*Fairchild exception*".

10 22. The primary judge committed three additional errors in arriving in his specific conclusions on causation.

23. *First*, there was the failure to consider or apply the "*but for*" test. It appears that the primary judge simply overlooked the issue, despite the fact that specific submissions were addressed to this issue. Although the Court of Appeal recognised this omission by the primary judge, it did not correct it, and (although it is not clear) may have held that the "*but for*" test did not apply[13]. If the "*but for*" test was applied, when accompanied by the extent of the increase of risk, the claim would have failed against each of Amaca and Amaba.

24. The second additional error made by the primary judge relates to the way in
20 which he applied Lord Reid's *dictum* in *Bonnington Castings v Wardlaw*[14]. As this Court explained in *Amaca v Ellis*[15], that *dictum* does not apply in circumstances like these; it can apply to divisible injuries which are the outcome of a gradual process to make a tortfeasor liable for a contribution to the overall injury. The *dictum* does not apply in cases, like this, of an indivisible injury. Since then, in *Sienkiewicz v Greif*, Lord Phillips has given a similar explanation for *Bonnington Castings*[16].

25. The third additional error made by the primary judge came about because he was wrong in his assessment of an increase of risk of 10% and 20% – the correct figures are 4% and 8%. There were two errors in the mathematics:

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12 [2011] 2 WLR 523 at 527 [4]
13 CA [110]-[114]
14 DDT [170]. The Court of Appeal specifically approved the primary judge's reasoning on this point at CA [170]
15 At 136 [66]-[68]
16 At 530-1 [17]

(a) The first stemmed from the use of a background risk of only 70 cases per million, per lifetime. The primary judge gave no reasons for doing this. The range was 70 to 140 per million, per lifetime. Given the onus, the primary judge should have used the upper end of the range to test the increase in risk, not the lower end of the range.

(b) The second derived from the use of a false comparator. In the case of Amaba, the primary judge calculated the extent to which asbestos from its products increased the risk, and then compared that to the lower end of the range of the background risk[17]. This is an error: the primary judge should have compared the risk due to Amaba's product with the risk from all other sources – ie: the background risk + the risk created by other asbestos exposures + the risk created by asbestos from all other brake products for which Amaba was not responsible, including Amaca's products.

26. The Court of Appeal declined to look at this issue in the context of an appeal limited to a point of law because "*if this exercise involves some arithmetical error, it was an error of fact, not law*"[18]. With respect, a legal conclusion dependent upon a mathematical calculation must be vitiated if the calculation is wrong.

Another reason to doubt the existence of a risk

27. There is another reason to suggest that the first respondent was at little or no risk of mesothelioma from his work as a motor mechanic – since 1988, 22 epidemiological studies have been published, but no epidemiological study has been able to demonstrate that motor mechanics are at an increased risk of mesothelioma from their brake work[19]. This has been explained by the fact that the type of fibre used in manufacturing brake components is chrysotile, the least dangerous kind of asbestos, and the bonding processes and materials may render the asbestos component harmless[20].

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17 DDT [166]-[167]
18 CA [132]
19 For example, Professor Henderson agreed that this was so: T118.49 – 118.50
20 See Dr Leigh T470.50 – 471.19

28. The primary judge discarded this consideration, and was in error to do so. To explain the error something needs to be said about epidemiology and its role in this case.

(a) Epidemiology is the study of disease in populations. It was through epidemiology that scientists were able to discern that inhaling asbestos causes mesothelioma. Further studies were able to discern a marked difference in the potency of the different fibre types;

(b) There are different kinds of epidemiological studies, which have different values as evidence amongst medical scientists. Of the 22 epidemiological studies in respect of motor mechanics, 19 were case-control studies and three were meta-analyses;

(c) A case-control study collects a group of people with the particular disease and matches those with a control group being of people of similar age etc, who do not suffer the disease. Investigations then made of the control group to find out how many of those have, for example, worked with asbestos. From this an epidemiologist can deduce the relative risk of the disease associated with asbestos[21];

(d) A meta-analysis is a method of combining the results of several studies to produce a more precise estimate, summarising all of the available information[22]. This is the most important kind of study;

(e) Case-control studies and meta-analyses are able to express the risk of contraction of a particular disease in mathematical terms. The presence of a risk is described by reference to the concept of "*relative risk*" (which is commonly abbreviated to "*RR*"). A relative risk of RR = 1.0 is a null result, and a positive or negative result – ie an increase or decrease in risk – would be demonstrated by numbers which are higher or lower than RR = 1.0. For example, a result of RR = 2.0 indicates that the risk has doubled;

(f) The certainty of the assessment of a relative risk is subject to interpretation within mathematical tolerances, which mean that a result is

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[21] Professor Berry T530.14 – 530.22
[22] Professor Berry T534.11 – 534.19

typically verified by reference against a "*95% confidence interval*" (commonly abbreviated to "*CI*"). The 95% confidence interval sets out (with a 95% degree of confidence) the upper and lower range of results. For example, if a study establishes a relative risk of RR = 1.0 with a confidence interval of CI = 0.8 – 1.2 means that the data cannot produce a risk no smaller than RR = 0.8 or greater than RR = 1.2.

29. Despite 22 epidemiological studies which failed to show any increase in risk of mesothelioma amongst motor mechanics, the primary judge found that, in effect, this was the wrong way in which to interpret the studies. There were two reasons why the primary judge arrived at this view.

30. First there was the evidence of Dr Leigh who, the primary judge said, "*gave cogent evidence criticising many of these studies*"[23]. But that statement presents an incomplete picture:

    (a) In his written report, Dr Leigh referred to only one of the 22 epidemiological studies, and then without criticism and only in passing[24];

    (b) In evidence in chief, over objection and without notice, Dr Leigh offered some recalculations in relation to five of the 22 studies. This could be described as criticism, but it was in respect of the least important studies;

    (c) During cross-examination Dr Leigh was asked about the three meta-analyses and was unable to make any substantial criticism of any of them[25]. Dr Leigh said he was unable to make such criticism because he had not "*been hired to do that*"[26].

31. There was no basis for finding that Dr Leigh made a "*cogent*" criticism of "*many*" of the studies.

32. The second reason given by the primary judge was that the studies did disclose an increase in risk. This was on the basis of his reinterpretation of the meta-analysis by one of the researchers – Otto Wong[27]. Wong reported "*there*

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[23] DDT [76]
[24] T277.46 – 277.48
[25] T276.2 – 294.21
[26] T294.20
[27] Ex DX 4 Wong: "*Malignant mesothelioma and asbestos exposure among auto mechanics: Appraisal of scientific evidence*" (2001) Regulatory Toxicology and Pharmacology

is no evidence to support ... an increased risk of mesothelioma ... among garage mechanics". The study found the relative risk of mesothelioma amongst mechanics was only RR = 0.90 – ie no increase of risk at all. However, the confidence interval was CI = 0.66 – 1.23. The primary judge used the upper end of this range to suggest that the result of the Wong study was consistent with an increased risk[28]. That, with respect, is an inappropriate use of statistics.

The alternative: Causation by reference to the "*cumulative effect*" theory

33. The primary judge found that all exposures to asbestos, other than trivial or *de minimis* exposures, materially contribute to the cause of mesothelioma[29]. This was an acceptance of the "*cumulative effect*" theory of causation. Under the "*cumulative effect*" theory (at least as it was interpreted by the primary judge) every asbestos fibre was necessary to produce the mesothelioma, and so fibres from all sources are causally implicated. Under the "*cumulative effect*" theory no defendant escapes liability.

34. The primary judge accepted the "*cumulative effect*" theory because, he said, four medical experts were "*each of the opinion that all asbestos fibres contribute to the development of a mesothelioma*"[30]. With respect, that statement is wrong; in fact, none of the experts said so. The medical witnesses were saying no more than the *risk* of mesothelioma increases with the dose of asbestos. It is an example of terminology differing between disciplines: The significance which a lawyer should place upon the distinction between *risk* and *cause* might be trivial to a medical doctor.

35. There were four experts who gave evidence in relation to this matter – Professor Henderson, Dr Leigh, Professor Musk and Dr Heiner. This is their evidence.

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[28] DDT [78-[79]
[29] DDT [59]-[62]
[30] DDT [52]

Professor Henderson

36. Professor Henderson is a pathologist. He gave a written report on causation in which he said[31]:

> *"So that as cumulative asbestos exposure increases so does the risk of mesothelioma as a consequence. It follows that each pattern/episode of asbestos exposure within an acceptable latency interval contributes causally towards the development of mesothelioma".*

37. This is a reference to risk, not cause: The slippage in terminology is critical – if a risk increases, "*it follows*" it "*contributes causally*".

10 38. In oral evidence Professor Henderson was asked about the "*cumulative effect*" theory over objection with a leading question[32]. Although Professor Henderson agreed with the proposition contained within the leading question, he qualified that so that it was understood to mean that as "*cumulative exposure increases, so does the risk of mesothelioma*"[33].

39. Under cross-examination Professor Henderson agreed that the biological processes whereby inhaling asbestos causes mesothelioma were not completely understood[34], and gave this evidence[35]:

> *Q. What is understood, what the science establishes, is that inhaling asbestos increases the risk of contracting mesothelioma, do you agree?*
>
> 20 *A. I agree with that. But I would add further comment that risk is not in this context simply a nebulous or theoretical construct. The risk is assessed on the basis of the numbers of cases which result from that type of inhalation. And I have always thought that risk is a bad term but everybody uses it.*

40. And[36]:

> *Q. And what you are really saying is that each episode of exposure adds to a risk?*
>
> *A. That's right.*

41. And in response to a question from the primary judge[37]:

> *Q. In the case of Mr Booth, are you able to say whether or not that particular risk of that last exposure came home?*
>
> 30 *A. No. I'd say particularly the risk from all his exposures came home because the model which I adopt is that of a cumulative exposure dose response, so I think that all of the asbestos fibres that he has inhaled, or at least a proportion of them, will contribute to the risk and to the ultimate development of the mesothelioma."*

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[31] Ex PX 10
[32] T89.11 – 90.33
[33] T90.38
[34] T112.37 – .40
[35] T113.23 – 113.72
[36] T116.46 – 116.47
[37] T117.12 – 117.16

42. And in response to a further question in cross-examination[38]:

> *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.*

43. This evidence contradicts the primary judge's interpretation of the "*cumulative effect*" theory. The evidence supports the proposition that an increased dose of asbestos increases the risk of the contraction of mesothelioma.

Dr Leigh

44. Dr Leigh is an occupational physician and epidemiologist. He is the strongest proponent of what might be called the "*cumulative effect*" theory, or at least something close to it. This seems partly driven by an interest to reform the law in this area[39].

45. In a written report, Dr Leigh said "*all exposure, recalled and unrecalled or unrecognised, would have contributed cumulatively to the risk of mesothelioma*"[40]. That is a conventional statement of the state of medical knowledge. But he also referred to what he described as a "*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. On this basis he said[41]:

> "*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.*"

46. This is the closest the evidence comes to supporting the "*cumulative effect*" theory, but even then it falls short of offering some basis for such an opinion. In any event, it is inconsistent with the rest of Dr Leigh's evidence.

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[38] T117.19 – 117.22

[39] Dr Leigh also has a law degree, and has given evidence that he does not agree with the High Court's decision in *Amaca v Ellis*, or the legal rules by which liability is determined and damages awarded: see, for example, *Evans v Queanbeyan City Council* [2010] NSWDDT 7 at [70], [76]

[40] Ex PX 21

[41] Ex PX 21

47. In oral evidence, Dr Leigh (like Professor Henderson) was asked a leading question over objection, but responded in terms of risk[42]:

> *Q. Perhaps I can put it more directly, do you consider that all exposure contributes cumulatively to the cause of mesothelioma?*
>
> *A. ... all exposure cumulatively contributes to the 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.*

48. In cross-examination Dr Leigh gave this evidence[43]:

> *Q. Is what you're saying really in effect that the exposure on the wharves 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.*

49. And later[44]:

> *Q. ... Dr Leigh, you can't say that except for the brake work Mr Booth wouldn't have got his mesothelioma, can you?*
>
> *A. No, I can't say that.*
>
> *Q. What you're saying is that you can't exclude the brake work?*
>
> *A. Yes.*

50. Dr Leigh's evidence conflates the concepts of *risk* and *cause*. His evidence is that once mesothelioma is contracted not even a single fibre can be ignored[45], but this is because, on his view, once a risk eventuates it must be held to be the product of all factors increasing that risk. That is not medical science, and it does not accord with the legal test for causation.

Professor Musk

51. Professor Musk is a respiratory physician. He gave a written report which described causation in terms of increasing a risk[46].

52. In examination in chief Professor Musk gave this evidence[47]:

> *Q. Do you consider that all exposure to asbestos within an acceptable latency period materially contributes to the mesothelioma in a particular patient?*
>
> ...

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42 T211.45 – 212.13
43 T263.14 – 263.16
44 T270.33 – 270.36
45 T265.38
46 Ex PX 28
47 T441.22 – 441.30

A. Yes I do, all periods of exposure outside the – more than ten to 15 years ago would have contributed to his risk.

53. This really constitutes a rejection of the "*cumulative effect*" theory: Professor Musk only expressed himself in terms of an increase in risk, not cause.

54. In cross-examination Professor Musk agreed that the biological mechanism whereby inhaling asbestos induces mesothelioma is not well understood[48]. This was his evidence[49]:

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

55. Because Professor Musk would not support the "*cumulative effect*" theory, his own counsel attempted to undermine him in re-examination by asking him to defer to the opinions of Professor Henderson and Dr Leigh[50]. Professor Musk declined to do so: Professor Musk is an extremely experienced researcher, principally responsible for investigating the consequences of crocidolite at Wittenoom in Western Australia. In short, he was the most highly qualified of all of the medical witnesses in the proceedings.

Dr Heiner

56. Dr Heiner is a respiratory physician. He gave three reports, none of which address the "*cumulative effect*" theory. Despite that, the primary judge permitted Dr Heiner to give oral evidence on the subject. In that evidence, Dr Heiner did not endorse the "*cumulative effect*" theory, instead speaking of risk and likelihood[51].

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[48] T443.34 – 444.3
[49] T444.27 – 444.35
[50] T446.2 – 446.28
[51] T402.12 – 402.46

57. In cross-examination Dr Heiner gave this evidence[52]:

> *Q. Is this what you are saying, that what is known it is that inhaling asbestos can, at least in some circumstances, increase the risk of contracting mesothelioma?*
>
> *A. Inhaling asbestos can – can certainly cause mesothelioma, yes.*
>
> *Q. And inhaling asbestos increases the risk, depending upon the 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.*

Conclusion on the medical evidence

58. Properly analysed none of the medical witnesses supported the "*cumulative effect*" theory; rather they were suggesting that an increase in exposure to asbestos will increase the risk of constructing mesothelioma. In this sense, the evidence is compatible with the state of knowledge described by Lord Phillips in *Sienkiewicz v Greif*[53].

59. In any event, the learning which underpins the "*cumulative effect*" theory is insufficient to provide a basis for the acceptance of the theory by a Court. A mere theory, without some general acceptance amongst practitioners in the discipline, is not "*expert opinion*", and should not be acted upon by the Courts. The "*cumulative effect*" theory is not widely accepted. In fact, no basis for it was able to be identified, as can be demonstrated by the following:

(a) Dr Leigh was the strongest proponent of the "*cumulative effect*" theory. Although Dr Leigh has considerable experience in asbestos-related diseases, he is a commentator and not an original researcher. Dr Leigh was reliant upon the information in the leading texts and scientific papers;

(b) When questioned about the authorities upon which he cited, Dr Leigh conceded that he did not know whether researchers have ever published a statement to the effect of the "*cumulative effect*" theory – he could only

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[52] T407.50 – 408.7

[53] See [2011] 2 WLR 523 at 531-2, [18]-[19] and 558-562

say that it was "*inferable*"[54]; or the result of his own "*synthesis*"[55]; or the product of his taking a "*leap*"[56] into the theory of causal mechanisms;

(c) And although invited to take time to find and supply any reference to support his theory, Dr Leigh did not do so[57].

60. In the end, the "*cumulative effect*" theory has not been shown to have any support amongst the leading researchers. There is an insufficient basis for it to be accepted in law.

Conclusion on causation and risk

10 61. Unless there is some alteration to the rules of causation in tort, the first respondent's claim should fail. It is important to bear in mind that the circumstances of the present case are different to those in an ordinary mesothelioma claim where the inference of causation is usually overwhelming. Here there was an exposure to the least dangerous type of asbestos and working with brakes placed the first respondent at a very low risk of contracting mesothelioma. His illness seems to be the product of the rare, but real, background risk.

PART VII: APPLICABLE STATUTORY PROVISIONS

20 62. Section 32 of the *Dust Diseases Tribunal Act 1998*.

PART VIII: ORDERS SOUGHT

63. Appeal allowed.

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

(a) Appeal allowed;

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[54] T473.18
[55] T473.34
[56] T473.43
[57] T473.4 – 475.43

(b) Set aside the Order 1 made by Judge Curtis on 10 May 2010, and in lieu thereof order verdict and judgment for the defendants;

65. Order the appellant to pay the first respondent's costs of this appeal.

Dated: 8 July 2011

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