

# CAUSATION IN MALIGNANT DISEASES CASES

## lung cancer and mesothelioma

By Tanya Segelov

Courts in Australia have recognised asbestos as a cause of two malignant diseases: lung cancer and mesothelioma.

Causation is most contested in lung cancer cases. While asbestos is capable of causing lung cancer, it is only one of a large number of causes, the most significant being smoking. Given a latency period of 20 years plus and the prevalence of smoking in Australian society among blue collar workers in the 1960s and 1970s, a significant majority of those diagnosed with lung cancer who were exposed to asbestos were also heavy smokers.

The issue of causation in mesothelioma cases is different. The only known cause of mesothelioma in Australia is exposure to asbestos. Rather than two or more competing causes, the issue of causation arises where there have been several different exposures to asbestos, defendants having attempted to argue along the lines of the UK decision of *Fairchild v Glenhaven Funeral Services Ltd*<sup>1</sup> that a plaintiff must prove which exposure to asbestos was causative of the mesothelioma.

### LUNG CANCER

It was traditionally believed that to have an asbestos related lung cancer, a person must also suffer from asbestosis (the necessary precursor hypothesis).

In *McDonald v The State Rail Authority (NSW) & Ors*,<sup>2</sup> O'Meally P held that carcinoma of the lung may be attributable to asbestos exposure in the absence of asbestosis where the exposure was sufficient to have caused asbestosis (the fibre burden hypothesis).

Following *McDonald*, the argument agitated in lung cancer cases shifted from 'the necessity for asbestosis to be present',

to the 'level of asbestos exposure necessary' to attribute the cancer to asbestos exposure.<sup>3</sup>

### Asbestos and tobacco

Lung cancer and the issue of asbestos and tobacco exposure came before the High Court in *Amaca Pty Ltd v Ellis*.<sup>4</sup> *Ellis* was an appeal from the Western Australian Court of Appeal, which upheld the trial judge's verdict by a two:one majority.<sup>5</sup> The claim related to the late Paul Steven Cotton. Mr Cotton began smoking at the age of 17 in 1973 and smoked continuously for over 26 years, smoking between 15 and 20 cigarettes per day. In terms of his exposure to asbestos fibres, he worked for the Engineering and Water Supply Department in South Australia from between 1975 and 1978 (2.5 years), laying asbestos cement pipes manufactured by Amaca (formerly James Hardie & Coy Pty Ltd) and, on occasions, carrying out repair and maintenance work on such pipes. Mr Cotton also alleged intermittent exposure to asbestos in a second period, with a different employer,<sup>6</sup> as a result of insulation around ovens in a factory in which he worked.

The trial judge<sup>7</sup> found that Mr Cotton's cumulative dose of asbestos exposure was between 5 and 10 fibre/ml years and that the relative risk for exposure to asbestos for the causation of lung cancer was in the order of 1.1 to 1.2.

The majority of the Court of Appeal<sup>8</sup> (Steytler P and McLure JA with Martin CJ dissenting) accepted that smoking and asbestos exposure were not independent but cumulative causes of lung cancer. In these circumstances, they found that epidemiological evidence had no direct application to the question of causation, because it was based on a false >>



assumption – that asbestos exposure and tobacco were competing causes. The majority held that once it had been determined that all asbestos exposure operated cumulatively, the only remaining issue was whether each period of occupational exposure made a material contribution to Mr Cotton's lung cancer. In determining this question, the Western Australian Court of Appeal concluded that the relative risk ratio would best serve as a general guide.<sup>9</sup>

The High Court unanimously rejected the reasoning of the Western Australian Court of Appeal.

There is some difficulty in interpreting the High Court decision, as the case seems to have been argued on a limited basis. The judgment states:<sup>10</sup>

'It was the plaintiff's case in this Court (and in the courts below) that causation was to be determined by applying a "but for" test: would Mr Cotton have contracted lung cancer but for the negligent exposure to asbestos?'

The High Court looked at the question as to whether asbestos alone was the cause of Mr Cotton's lung cancer and not whether his exposure to asbestos in conjunction with tobacco smoke was the cause of his lung cancer. That is, the plaintiff did not seek to rely upon authorities dealing with the question of causation where two or more causes existed which cumulatively caused disease.

The judgment continues:<sup>11</sup>

'The plaintiff expressly disavowed any argument in these appeals that demonstrating only that the exposure to asbestos increased the risk of contracting lung cancer was sufficient to establish causation. It was the plaintiff's case in this court, as it had been in the courts below, that she could succeed only if she showed that Mr Cotton's exposure to asbestos had caused or contributed to (in a sense of being a necessary condition for) his developing lung cancer. This being the way in which the case was presented, it will be neither necessary nor appropriate to consider issues of the kind considered by the House of Lords in *McGhee v National Coal Board*, *Fairchild v Glenhaven Funeral Services Ltd* and *Barker v Corus UK Ltd* or by the Supreme Court of Canada in *Resurfice Corp v Hanke*.' [footnotes omitted]

### Epidemiological evidence

In the High Court, the plaintiff did not rely upon the findings of the Court of Appeal in relation to cumulative exposure, submitting that the use by the Court of Appeal of the word 'cumulatively' was 'unfortunate'.<sup>12</sup> Rather, the plaintiff ran a case on the basis that the inferences from epidemiological evidence could prove that asbestos alone was the probable cause of the late Mr Cotton's lung cancer.

The High Court noted that the epidemiological evidence showed that many lung cancer sufferers had smoked tobacco, a few were exposed to asbestos; some exposed to asbestos had also smoked; and some had neither smoked nor been exposed to asbestos.<sup>13</sup> The High Court noted:<sup>14</sup>

'Observing that by far the largest number of a population of lung cancer sufferers had been either smokers, or smokers and exposed to asbestos, does not, **without more**, provide a foundation for an inference about the probability

that asbestos exposure was a cause of Mr Cotton's cancer.' [emphasis added]

The High Court noted that epidemiological evidence referred to populations and for an inference to be drawn from such evidence, the results of the studies of populations had to be related to the particular case at hand. Later, the court stated:<sup>15</sup>

'Observing that a small percentage of cases of cancer were probably caused by exposure to asbestos does not identify whether an individual is one of that group. And given the small size of the percentage, the observation does not, **without more** support the drawing of an inference in a particular case.' [emphasis added]

The reference to 'without more' in the above two quotations echoes Chief Justice Spigelman's 'strands in the cable' approach in *Seltsam Pty Limited v McGuiness*.<sup>16</sup> In *Seltsam*, his Honour held that it is 'necessary to bring together the strands in the cable and determine whether an inference'<sup>17</sup> about the exposure and illness should be drawn. In that case, the Chief Justice noted:

'In Australian law, the test of actual persuasion does not require epidemiological studies to reach the level of a relative risk of 2.0, even where that is the only evidence available to a court. Nevertheless, the closer the ratio approaches 2.0, the greater the significance that can be attached to the studies for the purpose of drawing an inference of causation in an individual case. The "strands in the cable" must be capable of bearing the weight of the ultimate inference.'<sup>18</sup>

### Synergistic effect

In *Ellis*, the High Court only considered whether or not asbestos on its own probably caused the lung cancer, and not the synergistic effect of exposure and smoking. In doing this, the High Court accepted that the interactive effect between asbestos exposure and smoking (something acknowledged by all experts) could be partitioned. Dr James Leigh, an epidemiologist, gave evidence that:<sup>19</sup>

'While the precise mechanism of interaction between asbestos and tobacco smoke in causing lung cancer is not known, it is not possible in my view to separate their effects in the individual case when both have acted and it is thus more probable than not, that in this situation, the lung cancer was the single result of the two factors acting together.'

The High Court noted that Dr Leigh conceded that it was possible to partition attributability to smoking and asbestos under a variety of mathematical risk models. Various witnesses gave evidence in relation to these models, the High Court observing that no witness assigned more than a 23 per cent chance of Mr Cotton's lung cancer being caused by his exposure to asbestos with or without his smoking history.<sup>20</sup>

A few weeks prior to the decision of the High Court, the New South Wales Court of Appeal handed down a decision in *Workers' Compensation (Dust Diseases) Board of New South Wales v Smith, Munro and Seymour*,<sup>21</sup> which also considered the question of attributability of lung cancer to asbestos exposure. The appeal related to three former waterfront

workers who had all been exposed to asbestos while unloading asbestos cargo and who had contracted lung cancer and died.

The NSW Workers' Compensation Dust Diseases Board (Dust Diseases Board), both at trial and on appeal, argued that while both smoking and asbestos were capable of causing lung cancer, the statistical likelihood in each case was that it was less than 50 per cent likely that asbestos caused the disease. The Dust Diseases Board contended that the likelihood of lung cancer having resulted from the deceaseds' smoking was many times that of their exposure to asbestos dust and, accordingly, none of the deceased could demonstrate that, on the balance of probabilities, the cancer resulted from their exposure to asbestos dust.

The plaintiffs argued that it was reasonably likely that the two agents had interacted, with the result that each probably contributed to the disease in a material respect in a particular individual and although it may be possible to statistically separate the likely contribution of each agent, this was neither necessary nor appropriate for determining that a disease was due to a particular agent.

Although the court was concerned with the statutory requirement under the *Workers' Compensation (Dust Diseases) Act 1942* (NSW), that each worker's death was 'reasonably attributable to the person's exposure to the inhalation of dust',<sup>22</sup> it was accepted that this test involved a determination of whether asbestos dust materially contributed to the carcinoma.

The Court of Appeal found that the trial judge did not ignore the causative effect of smoking; rather, she correctly identified that the test required the court to determine whether it had been shown on the balance of probabilities that exposure to asbestos dust materially contributed to the lung cancer. The Court of Appeal did not accept that there was an error by the trial judge in failing to find a material contribution only when the asbestos inhalation contributed 50 per cent or more to the cause of the cancer. As Basten JA stated:<sup>23</sup>

'One factor can materially contribute to an outcome even though, relative to another factor, it has a minor effect.

All that is required is that the effect be "material". That required an evaluative judgment on the part of the court.'

In *Ellis*, the High Court noted that the biological processes by which tobacco and asbestos interact is unknown.<sup>24</sup> This was based on the evidence of Dr Leigh. The evidence of Professor Henderson in the *Smith, Munro and Seymour* appeals (Professor Henderson did not give evidence in *Ellis*), seems to be somewhat different, in that while conceding that the interactive effect was not medically explained, he discussed various theoretical explanations, two of which had good supportive evidence.<sup>25</sup> Basten JA noted that:<sup>26</sup>

'Further support may be given to the potential inference by expert evidence of a possible explanation of a mechanism, although the operation of the mechanism has not been observed. That may provide an element of "biological plausibility": See Spigelman CJ at [41], [42].<sup>27</sup> Biological plausibility may be strengthened by observation of a known mechanism in laboratory experiments or by

epidemiological studies. These are matters for expert analysis and explanation, not commonsense.'

Based on this evidence, the Court of Appeal upheld the trial judge's finding: that the relative risk of cigarette smoking and asbestos could not be apportioned, describing the apportion modelling as an 'artificial exercise'.<sup>28</sup>

#### Post-Ellis

It is worth noting the decision of *Evans v Queanbeyan City Council & Anor*.<sup>29</sup> This decision of Curtis DCJ of the Dust Diseases Tribunal of NSW, in relation to a lung cancer claim without asbestosis and with a heavy smoking history, is the only such decision post-*Ellis*.<sup>30</sup>

While much has been made of the decision in *Ellis*, it is debatable whether the decision has altered the landscape of lung cancer cases. In *Ellis*, the plaintiff (Cotton) had a large smoking history and only an intermittent exposure to asbestos. This was also the case in *Evans*. In contrast, the three waterside workers in the *Smith, Munro and Seymour* appeals all had heavy exposure to asbestos, with cumulative exposures well in excess of 25 fibre/ml years and with one of the waterside workers ultimately being found by the court to have asbestosis.

Despite the decision in *Ellis*, it is possible to run lung cancer cases even where there is a heavy smoking history and no asbestosis, provided there is a moderately heavy and prolonged exposure to asbestos. The exposure should >>

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be sufficient to have caused the doubling of risk for lung cancer, at either 25 or 50 fibre/ml years of cumulative exposure to asbestos, depending on the fibre type and nature of the exposure.

Evidence in relation to the interactive effect of asbestos and tobacco and the causation of lung cancer would need to be adduced. The evidence in *Ellis* did no more than establish that it was arguable that such interactive effect existed. In the *Smith, Munro and Seymour* appeals, there was evidence in each case from Professor Henderson, an eminently qualified pathologist with particular expertise in asbestos and lung cancer, that the interactive effect occurred. In each case, Professor Henderson gave evidence that it was his view that the deceased's lung cancer was not a result of either asbestos or tobacco smoke but of both acting together. This direct evidence is to be contrasted with the evidence in *Ellis*, which relied solely upon epidemiological considerations and concepts of relative risk.

It is possible that, in addition to the above evidence further evidence is required to prove the 'something more' referred to by the High Court in *Ellis*. 'Something more' may be evidence that workers with similar asbestos exposure had contracted asbestosis.

### MESOTHELIOMA

Plaintiffs in the Dust Diseases Tribunal had assumed that the issue of causation in mesothelioma claims had been settled for many years following the decision of the New South Wales Court of Appeal in *EM Baldwin & Sons Ltd v Plane*.<sup>31</sup>

#### Cumulative effect

Mr Plane contracted mesothelioma following exposure to asbestos during the course of his employment working for EM Baldwin Pty Ltd. During his employment he was exposed to asbestos brake linings manufactured by Jsekarb Pty Ltd (formerly Hardie Ferodo Pty Ltd). Jsekarb argued that Mr Plane's exposure was not causative of his mesothelioma in circumstances where its product contained commercial chrysotile (white) asbestos and the plaintiff had been exposed to other asbestos containing crocidolite (blue) asbestos.<sup>32</sup>

Professor Henderson was called as an expert for Mr Plane at the trial. His evidence was to the effect that the plaintiff's total cumulative exposure to asbestos caused his mesothelioma (the cumulative effect theory).<sup>33</sup>

The Court of Appeal accepted the evidence of Professor Henderson, noting that he was undoubtedly the pre-eminent expert witness on the pathogenesis of mesothelioma. The Court noted that there was no contrary view proffered by an equally qualified expert and held that:

'...the Tribunal was entitled to accept Professor Henderson's "cumulative effect" theory and was correct to do so and reject Jsekarb's fundamental proposition that the inhalation of more than one amphibole form of asbestos fibre raises a number of separate, independent, possible causes of mesothelioma.'<sup>34</sup>

Over ten years later, Amaba Pty Ltd (formerly Jsekarb Pty Ltd) sought again to argue the question of causation in

proceedings brought by John Booth in the Dust Diseases Tribunal.<sup>35</sup> The defendants were Amaba Pty Ltd and Amaca Pty Ltd in relation to asbestos inhalation from brake linings, manufactured by the two defendants, resulting in his condition of malignant pleural mesothelioma.

Mr Booth worked between 1953 and 1983 as a motor mechanic, with the exception of three years between 1969 and 1971. In addition to occupational exposure, Mr Booth had small home renovation type exposures, as well as 20 minutes unloading hessian bags of asbestos on the Sydney waterfront.

Mr Booth called medical evidence, including from Professor Henderson and Dr Leigh. As in *Plane*, Professor Henderson gave evidence as to the cumulative effect theory; that is, that all exposure both recalled and unrecalled within an accepted latency period contributes causally to the development of mesothelioma. Professor Henderson's evidence described at length the mechanical and chemical/biological (or cellular) basis for this theory. Dr Leigh explained 'the current consensus understanding of the cellular and biological methods by which asbestos fibres cumulatively cause mesothelioma'. He explained that his views were based on a range of data, including toxicological studies in animals, studies in human cells and experimental clinical studies. Professor Henderson and Professor Leigh's opinions were supported by Professor Musk and Dr Heiner, respiratory specialists.

The defendants called no evidence to contradict Mr Booth's medical expert opinions. Judge Curtis noted that although almost ten years had lapsed since the decision in *Plane*, the defendants called no medical evidence to suggest that the 'cumulative effect theory' may not be accepted because of further advances in medical knowledge.<sup>36</sup>

Critically, a number of facts were not in dispute. First, that Mr Booth's mesothelioma was caused by the inhalation of asbestos fibres. Second, that chrysotile asbestos has the capacity to cause mesothelioma. Third, that the brake linings manufactured by Amaba and Amaca contained chrysotile asbestos and, fourth, that Mr Booth inhaled chrysotile asbestos fibres liberated from their products.<sup>37</sup>

#### Asbestos and disease

As in *Ellis*, the defendants in *Booth* sought to attack the scientific or biological understanding of the disease and its attribution to asbestos. The defendants argued that because the biological process whereby the inhalation of asbestos causes mesothelioma is incompletely understood, medical science cannot support the proposition advanced by Mr Booth's expert witnesses that all asbestos inhaled materially contributed to his mesothelioma.

His Honour, rejecting the defendants' submissions, concluded:<sup>38</sup>

'The plaintiff's experts, conceding that some of the steps necessary to form the opinion on purely deductive and scientific grounds are yet to be discovered, are each of the opinion that all asbestos fibres contribute to the development of a mesothelioma. They adopt that theory as most probably according with the actual aetiology

of the disease. Their conclusions are not guesses, but reasonable inferences drawn from the current state of medical knowledge.'

This finding was upheld on appeal.<sup>39</sup> Basten JA opined:<sup>40</sup>

'The civil standard of proof, on the balance of probabilities, permits a yawning gap between complete understanding and sufficient understanding. There may be an even greater gap between that which is "capable" of supporting a finding on the balance of probabilities and that which the appellants would accept "does" support such a finding.'

The defendants argued that the only evidence of causation was the epidemiological evidence, which could not demonstrate causation on the balance of probabilities, unless the relative risk attributable to the particular tortious exposure, as compared with all other exposures, approached 2.0. The primary judge rejected the defendants' argument, noting that:

'proof of causation in this case does not turn upon the epidemiological evidence or upon questionable estimations of total fibre burden. An overwhelming inference of causation may be drawn from the following facts:

1. Mr Booth's mesothelioma was caused by the inhalation of asbestos fibres;
2. Mesothelioma very rarely occurs in persons who have not been exposed to asbestos fibres beyond the background level that pervades urban environments;
3. 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 work of others in his vicinity;
4. The previous exposure, in the course of home renovations and truck loading was, in comparison, trivial.<sup>41</sup>

The Court of Appeal rejected the argument that *Seltsam* stood for the proposition that the Court was bound to accept the epidemiological evidence, noting that in *Seltsam* the plaintiff relied solely on epidemiological evidence, which was not the case here.<sup>42</sup>

### Cumulative effect

*Booth* was not a case where the trial judge found causation on the basis of increased risk. While his Honour made a number of mathematical calculations quantifying the amount of asbestos fibre from products manufactured by Amaba and Amaca above the background level of exposure, the judgment makes clear that he did not consider the mathematical calculations to be 'necessarily compelling',<sup>43</sup> noting that they did not 'accurately reflect the accumulating risk of exposure to asbestos fibres in brake repair work'.<sup>44</sup>

The defendants argued that *Ellis* stood for the proposition that if all exposure had a cumulative effect, a claimant cannot succeed unless he or she demonstrates that the particular exposure resulting from the tort of the defendant is one without which injury would not have occurred. The Court of Appeal noted that the facts of this case and *Ellis* were different in significant respects. As Basten JA observed, '...not only was [Mr Booth's] cancer one which was peculiarly

attributable to the inhalation of asbestos, but the evidence did ascribe a causal connection'.<sup>45</sup>

The evidence upon which the trial judge made his findings went beyond epidemiology and risk assessment. The risk had come home. The defendants called no evidence to prove that in the absence of any particular exposure, Mr Booth would have developed his mesothelioma. In these circumstances, the Court of Appeal accepted that the trial judge's analogy with *Bonnington Castings Ltd v Wardlaw*<sup>46</sup> was apt.<sup>47</sup>

There is nothing new in the judgment of *Booth*. The Court of Appeal held that the plaintiff had to – and did – establish, on the balance of probabilities, that in respect of each defendant, exposure to inhalation of asbestos from its products materially contributed to his disease.

The defendants sought special leave to appeal to the High Court. The application was heard on 10 June 2011. Leave was granted on ground 2 of the draft Notice of Appeal – the issue of causation. The High Court noted that the parties 'should understand that ground 2 is understood by us as inviting examination of the question whether there was evidence that if a person developed mesothelioma, each and every exposure to asbestos was a contributing cause of the development of that disease'.<sup>48</sup> ■

**Notes:** **1** [2003] 1 AC 32. **2** [1991] 7 NSWCCR 201. **3** See *Judd v Amaca Pty Ltd* [2003] NSWDDT12 (4 July 2003). **4** [2010] HCA 5. **5** *The State of South Australia v Ellis* [2008] WASCA 200 (26 September 2008). **6** The second employer was Millenium Inorganic Chemicals Ltd, between 1990 and 2002 (at 4). **7** EM Heenan J in *Ellis, Executor of the Estate of Paul Steven Cotton (Dec) v The State of South Australia & Ors* [2006] WASC 270 (8 December 2006). **8** [2008] WASCA 200. **9** 'Relative risk' measures the risk of contracting a disease from exposure to a causative factor, compared with those with no exposure; *ibid* at 198. **10** [2010] HCA 5 at 11. **11** [2010] HCA 5 at 12. **12** [2010] HCA 5 at 48. **13** [2010] HCA 5 at 20. **14** [2010] HCA 5 at 57. **15** [2010] HCA 5 at 70. **16** [2000] 49 NSWLR 262. **17** [2010] HCA 5 at 151. **18** [2000] 49 NSWLR 262 at 137. **19** [2010] HCA 5 at 33. **20** [2010] HCA 5 at 30. Compared to not less than 67 per cent probability of smoking alone (at 30). **21** [2010] NSWCA 19. **22** Section 8. **23** [2010] NSWCA 19 at 72. **24** The 'synergistic effect' is discussed at 32-7. **25** [2010] NSWCA 19 at 61. **26** [2010] NSWCA 19 at 128. **27** In *Seltsam*. **28** [2010] NSWCA 19 at 69. **29** [2010] NSWDDT 7. **30** The plaintiff did not succeed in proving causation. **31** [1998] 17 NSWCCR 23. **32** Crocidolite is many times more potent than chrysotile in the causation of mesothelioma. **33** [1998] 17 NSWCCR 23 (BC9807380) at 59-64. **34** [1998] 17 NSWCCR 23 at 95 (BC9807380) at 64. **35** *Booth v Amaca Pty Ltd and Amaba Pty Ltd* [2010] NSWDDT 8. **36** [2010] NSWDDT 8 at 60. **37** [2010] NSWDDT 8. **38** [2010] NSWDDT 8 at 52. **39** *Amaba Pty Ltd (Under NSW Administered Winding Up) v Booth; Amaca Pty Limited (Under NSW Administered Winding Up) v Booth* [2010] NSWCA 344 (10 December 2010). **40** [2010] NSWCA 344 at 116. **41** [2010] NSWDDT 8 at 162. **42** [2010] NSWCA 344 at 90. **43** [2010] NSWDDT 8 at 166. **44** [2010] NSWDDT 8 at 160. **45** [2010] NSWCA 344 at 103. **46** *Bonnington Castings Ltd v Wardlaw* [1956] AC 613. **47** [2010] NSWCA 344 at 107. **48** *Amaca Pty Limited (Under NSW Administered Winding Up) v Booth & Anor; Amaba Pty Limited (Under NSW Administered Winding Up) v Booth & Anor* [2011] HCATrans 152 (10 June 2011).

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