HIGH COURT OF AUSTRALIA

FRENCH CJ,
GUMMOW, HAYNE, HEYDON, CRENNAN, KIEFEL AND BELL JJ

Matter No P13/2009

AMACA PTY LTD (ACN 000 035 512) APPELLANT

AND

TERESA ELLIS AS EXECUTOR OF THE ESTATE OF
PAUL STEVEN COTTON (DEC) & ORS RESPONDENTS

Matter No P14/2009

THE STATE OF SOUTH AUSTRALIA APPELLANT

AND

TERESA ELLIS AS EXECUTOR OF THE ESTATE OF
PAUL STEVEN COTTON (DEC) & ORS RESPONDENTS

Matter No P12/2009

MILLENNIUM INORGANIC CHEMICALS LTD
(ACN 008 683 627) APPELLANT

AND

TERESA ELLIS AS EXECUTOR OF THE ESTATE OF
PAUL STEVEN COTTON (DEC) & ORS RESPONDENTS

Amaca Pty Ltd v Ellis
The State of South Australia v Ellis
Millennium Inorganic Chemicals Ltd v Ellis
[2010] HCA 5
3 March 2010
P13/2009, P14/2009 & P12/2009

ORDER

In each matter:

1. *Appeal allowed with costs.*

2. *Within 14 days of the date of this judgment the appellant should file and serve any proposed amended notice of appeal.*

3. *Within 28 days of the date of this judgment either the parties should file agreed minutes of the consequential orders they seek to have this Court make, or, in default of agreement, each party should file, and serve on the opposite party, written submissions setting out the consequential orders which that party submits that this Court should make, together with any submissions which the party makes in support of, or in opposition to, any proposed amendment of the notice of appeal.*

On appeal from the Supreme Court of Western Australia

Representation

G M Watson SC with J C Sheller for the appellant in P13/2009, the second respondent in P14/2009 and the third respondent in P12/2009 (instructed by Minter Ellison Lawyers)

M L Abbott QC with S J Doyle for the appellant in P14/2009 and the second respondent in P13/2009 and P12/2009 (instructed by Crown Solicitor for the State of South Australia)

D F Jackson QC with N J Owens for the appellant in P12/2009 and the third respondent in P13/2009 and P14/2009 (instructed by Lavan Legal)

B W Walker QC with J R C Gordon for the first respondent in each appeal (instructed by Slater & Gordon)

CATCHWORDS

Amaca Pty Ltd v Ellis
The State of South Australia v Ellis
Millennium Inorganic Chemicals Ltd v Ellis

Negligence – Causation – Balance of probabilities – Deceased was a smoker – Deceased exposed to respirable asbestos fibres in course of employment – Deceased died of lung cancer – No scientific or medical examination can say whether deceased's lung cancer caused by inhalation of tobacco smoke, respirable asbestos fibres, both, or neither – Use of epidemiological evidence – Whether epidemiological evidence founded inference of causation – "Synergistic" effect of smoking and exposure to asbestos fibres – Whether proved that exposure to asbestos more probably than not caused or contributed to deceased developing lung cancer – Relevance of material contribution.

Words and phrases – "epidemiology", "material contribution", "more probable than not", "relative risk", "synergistic effect".

1 FRENCH CJ, GUMMOW, HAYNE, HEYDON, CRENNAN, KIEFEL AND BELL JJ. Paul Steven Cotton died of lung cancer. He was a smoker, and it is well established that smoking can cause lung cancer. Breathing in asbestos fibres can also cause lung cancer. The determinative issue in each of these three appeals is whether the plaintiff (the executor of his estate) had established at trial that it was more probable than not that exposure to respirable asbestos fibres was a cause of Mr Cotton's cancer. Although a respondent to each of the appeals in this Court, it will be convenient to continue to refer to Ms Ellis, the executor of Mr Cotton's estate, as the plaintiff.

2 No scientific or medical examination can say why Mr Cotton developed lung cancer. His cancer was not, as some are, a cancer peculiarly associated with exposure to asbestos. The evidence at trial showed that most sufferers of lung cancer have smoked, some have been exposed to asbestos, some have smoked and been exposed to asbestos, and some have neither smoked nor been exposed to asbestos. The evidence also showed that not everyone who smokes, not everyone who has been exposed to asbestos, and not everyone who has smoked and been exposed to asbestos, will develop lung cancer.

3 Mr Cotton had smoked on average somewhere between 15 and 20 cigarettes a day for a bit over 26 years before he was diagnosed with lung cancer.

4 Mr Cotton had been exposed to respirable asbestos fibres in the course of his employment, first with the Engineering and Water Supply Department of the State of South Australia ("the EWSD") between 1975 and 1978, and later, with Millennium Inorganic Chemicals Ltd ("Millennium") between 1990 and his death in 2002. His exposure to asbestos when working with the EWSD resulted from working with asbestos cement pipes manufactured by Amaca Pty Ltd (formerly James Hardie & Coy Pty Ltd – "Amaca").

5 When, as here, medical and scientific examination cannot say whether exposure to respirable asbestos fibres was a cause of Mr Cotton's cancer, the medical practitioner and scientist have little choice but, as one witness said at trial, to "take it into consideration in looking at what *might* have caused his lung cancer". In their inquiries, the uncertainty about cause means that they cannot "exclude it from the end result".

6 The courts' response to uncertainty arising from the absence of knowledge must be different from that of the medical practitioner or scientist. The courts cannot respond to a claim that is made by saying that, because science and

medicine are not now able to say what caused Mr Cotton's cancer, the claim is neither allowed nor rejected. The courts must decide the claim and either dismiss it or hold the defendant responsible in damages. As Dixon J rightly said[1], albeit in a context very different from this:

> "There are few, if any, questions of fact that courts cannot undertake to inquire into. In fact it may be said that under the maxim *res iudicata pro veritate accipitur* [a thing adjudicated is received as the truth] courts have an advantage over other seekers after truth. For *by their judgment they can reduce to legal certainty questions to which no other conclusive answer can be given*." (emphasis added)

The plaintiff recognised that the courts were asked in this case to reduce to legal certainty a question of causation to which no other conclusive answer can be given.

7 The plaintiff sued the State of South Australia (in respect of the alleged defaults of the EWSD), Amaca and Millennium in the Supreme Court of Western Australia. Each defendant was an appellant in this Court.

8 At trial, each of the State of South Australia, Amaca and Millennium was found[2] by the trial judge (Heenan J) to have been negligent and to have breached a duty or duties owed to Mr Cotton and his dependants. All three were found liable in negligence. South Australia and Millennium were also found to have breached terms implied in the contract of employment each had made with Mr Cotton and to be in breach of statutory duty. The terms implied in each contract (and the relevant statutory duty) obliged each employer to take reasonable care for the safety of Mr Cotton. Judgment was entered for the plaintiff against each of South Australia, Amaca and Millennium.

9 By majority (Steytler P and McLure JA, Martin CJ dissenting), appeals to the Court of Appeal of Western Australia by each of South Australia, Amaca and

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1 *Bank of NSW v The Commonwealth* (1948) 76 CLR 1 at 340; [1948] HCA 7.

2 *Ellis v The State of South Australia* [2006] WASC 270.

Millennium, against the judgments entered at trial, were dismissed[3]. By special leave, each of South Australia, Amaca and Millennium appeals to this Court.

10 Neither the existence of duty nor breach of duty was in issue in this Court. Argument of the appeals in this Court proceeded, for the most part, on the assumption that the relevant claims were framed in negligence. The central question was whether it had been shown to be more probable than not that exposure to asbestos was a cause of Mr Cotton's death. More particularly, in each case, had it been shown to be more probable than not that the negligence of the defendant in question was a cause of Mr Cotton contracting lung cancer?

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

12 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 the 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*[4], *Fairchild v Glenhaven Funeral Services Ltd*[5] and *Barker v Corus UK Ltd*[6] or by the Supreme Court of Canada in *Resurfice Corp v Hanke*[7].

13 These reasons will demonstrate that each appeal should be allowed. Causation was not established in the plaintiff's case against any defendant. The

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3 *South Australia v Ellis* (2008) 37 WAR 1.

4 [1973] 1 WLR 1; [1972] 3 All ER 1008.

5 [2003] 1 AC 32.

6 [2006] 2 AC 572. See also *Sienkiewicz v Greif (UK) Ltd* [2009] EWCA Civ 1159.

7 [2007] 1 SCR 333.

evidence did not establish facts which positively suggested that it was more probable than not that the negligence of any defendant was a cause of Mr Cotton's cancer.

14 To support that conclusion it will be necessary to examine what evidence was led at trial, what the trial judge decided and what the majority in the Court of Appeal decided. It will also be necessary to examine the way in which, in argument in this Court, the plaintiff sought to describe what was held in the courts below, and sought to analyse the evidence. Those steps will necessarily require examination of detail. But in the end, the plaintiff's case, that it was more probable than not that Mr Cotton's being exposed to respirable asbestos fibres was a cause of his cancer, fails because no more was established than that, although exposure to asbestos *may* have been a cause of his cancer, it was not a probable cause.

Proceedings at trial

15 The plaintiff's case at trial was, and at all subsequent stages of this litigation has been, that resolution of the issue of causation in her claim against each defendant depended on inference. Her case was that the evidence founded the inference that it was more probable than not that the negligence of each of the defendants was a cause of Mr Cotton's lung cancer. That is, the plaintiff's case, that causation should be inferred, depended upon establishing one or more facts which "positively suggest[ed], that is to say provide[d] a reason, special to the particular case ... for thinking it likely that in that actual case ... a specific state of affairs existed"[8]. The "specific state of affairs" alleged to exist in this case was "but for" causal connection between the relevant defendant's exposing Mr Cotton to respirable asbestos fibres and his contracting the lung cancer from which he died.

16 The evidence relied on as establishing the facts which "positively suggest[ed], that is to say provide[d] a reason ... for thinking it likely" that the defendant's negligent exposure of Mr Cotton to respirable asbestos fibres was a cause of his cancer was evidence of epidemiological studies, which recorded and drew conclusions from the results of studies of large groups or populations.

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8 *Jones v Dunkel* (1959) 101 CLR 298 at 305; [1959] HCA 8.

17 Epidemiological evidence was adduced from Professor Musk (a respiratory physician), Professor de Klerk (an epidemiologist), Dr Leigh (a consultant occupational physician and epidemiologist) and Professor Berry (an epidemiologist and biostatistician). All four witnesses had longstanding interest and experience in the study of asbestos-related diseases.

18 The evidence of four other witnesses, Professor Wan, Dr Kendall, Mr Kottek and Professor Fox was specifically mentioned by the trial judge in his consideration of causation. Professor Wan gave evidence directed principally to the levels of Mr Cotton's exposure to asbestos and the connection between asbestos exposure and lung cancer. Dr Kendall, a respiratory physician, gave evidence of his opinion about the causes of Mr Cotton's cancer. Dr Kendall said that he could not say whether or not Mr Cotton's cancer was due to smoking or inhalation of asbestos products or some other cause. "Rather", he said, "I think it is a combination of all three." Mr Kottek, an occupational and environmental health consultant, gave evidence about the levels of Mr Cotton's exposure to asbestos when he was working, concluding that he "would have faced at least some increase (certainly more than minimal) in the risk of lung cancer over and above that which he would have faced from smoking alone". Professor Fox, of the Department of Clinical Haematology and Medical Oncology at the Royal Melbourne Hospital and the University of Melbourne, gave evidence of his opinion about the cause or causes of Mr Cotton's cancer.

19 Although expressly relied on by the trial judge, little reference was made on the hearing of the appeals in this Court to the evidence of Professor Wan, Dr Kendall, Mr Kottek or Professor Fox. Attention was focused upon the epidemiological evidence of Professor Musk, Professor de Klerk, Dr Leigh and Professor Berry.

The epidemiological evidence at trial

20 Reduced to its essentials, the epidemiological evidence showed that many sufferers of lung cancer had smoked tobacco, a few had been exposed to asbestos; some of those who had been exposed to asbestos had also smoked; some had been neither smokers nor exposed to asbestos. The observations made about the relationship between exposure to the known carcinogens of smoking and asbestos and the development of lung cancer were used and interpreted by epidemiologists in a number of different ways.

21 First, the data were used to provide measures of "relative risk". Relative risk was defined by Professor Berry (by reference to a Dictionary of Epidemiology[9]) as the "ratio of the risk of disease or death among the exposed to the risk among the unexposed". The measures of relative risk given in evidence described the size of the increase in the risk that a healthy person would contract lung cancer that was to be attributed to exposure to smoking, to asbestos, or to both smoking and asbestos. Second, the data were used to provide a measure of the extent to which the exposure to asbestos increased the chance that a smoker who was exposed to asbestos would contract lung cancer. Third, the data were interpreted as showing that the numbers of lung cancer sufferers who were smokers and had been exposed to asbestos were higher than would have been expected if the incidence of lung cancer in smokers was added to the incidence in those exposed to asbestos. This last observation was expressed as tobacco and asbestos having "a synergistic effect" and as asbestos exposure "multiplying" the risk of lung cancer due to smoking by a quantity greater than one.

22 These short descriptions of the ways in which the epidemiological data were used should not be understood as denying the complexity of the analyses that yielded the results described. Nor should the brevity of the descriptions be permitted to obscure the level of detail at which the data were analysed and expressed. To take but one example of that detail, account was taken in the relevant analyses not only of the extent to which Mr Cotton had smoked but also of the levels of exposure to respirable asbestos fibres that were to be expected in each of his particular employments.

23 There were, of course, differences in the ways in which the several witnesses expressed their opinions. There were differences between them about the particular values that should be given for the various measures of risk and probability about which they spoke. But neither the differences in expression, nor the differences in values stated, should obscure the very substantial measure of agreement between the witnesses on matters which bore upon the question of causation.

24 Professor de Klerk, Professor Musk, Professor Berry and Dr Leigh all gave evidence about the relative risk of exposure to smoking, to asbestos, to both smoking and asbestos, and to neither smoking nor asbestos. From values of

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9 Last (ed), *A Dictionary of Epidemiology*, 3rd ed (1995) at 145.

relative risk it was possible to derive, and Professor de Klerk, Professor Berry and Dr Leigh did derive, measures of the "attributable fraction" among the exposed. The "attributable fraction" among the exposed (usually expressed as a decimal or as a percentage) expressed the probability that the cancer in question was caused by exposure to the carcinogen or carcinogens in question.

25 The relative risk assigned to exposure to smoking or to exposure to asbestos varied with the level of exposure. Because the witnesses used different levels of exposure in their calculations, they derived different relative risk values. Those different values were as follows:

	Relative risk from smoking	Relative risk from exposure to asbestos
Professor de Klerk	7.7	1.3
Professor Musk	20	1.1 to 1.2
Professor Berry	15	1.1
Dr Leigh	8	1.16

It will be observed that each witness gave a relative risk from smoking that was many times greater than the relative risk from exposure to asbestos.

26 Using the figures for relative risk that he adopted, it was Professor de Klerk's opinion that the probability that Mr Cotton's lung cancer was due to smoking only was 0.67 (or 67%); the probability that cancer was due to asbestos only 0.03 (or 3%), due to asbestos *and* smoking 0.2 (or 20%), and due to neither smoking nor asbestos 0.1 (or 10%).

27 Professor Musk offered no written opinion assigning equivalent probabilities or attributable fractions and was not asked to offer an opinion on those matters in his oral evidence.

28 Professor Berry calculated the probabilities as:

"(a)	Due to smoking alone	92%
(b)	Due to asbestos alone	0.1%
(c)	Due to smoking-asbestos combination	0.9%
(d)	Background risk	7%."

Dr Leigh expressed the view that, *if* it were necessary "to partition attributability to smoking and asbestos", an approximate apportionment of 2 to 12% (or 5 to 20% if higher cumulative exposures were taken to account) should be made to asbestos.

29 One aspect of the epidemiological evidence that was given at trial must be kept at the forefront of consideration. Although there were differences between witnesses, all who gave evidence at trial about relative risks due to exposure to asbestos and due to smoking agreed that the risk due to smoking was much greater than the risk due to exposure to asbestos.

30 None of the four witnesses mentioned assigned a probability greater than 23% to the chance that Mr Cotton's cancer was caused by his exposure to asbestos, with or without his also being exposed to smoking. All assigned a much higher probability (not less than 67%) to the cancer being caused by smoking alone. Professor Berry described the relationship between the two risks by saying that it was about 100 times more likely that Mr Cotton's lung cancer was caused by smoking than by asbestos inhalation. While the witnesses differed about the figures, all witnesses agreed that the risk from smoking was many times greater than the risk from asbestos inhalation.

31 It is necessary to say something more about the synergistic or multiplicative effect of smoking and asbestos. That is necessary because an essential premise for the conclusions reached by the trial judge and the majority in the Court of Appeal[10] was that the acknowledged synergistic effect of tobacco smoke and asbestos showed that tobacco smoke and asbestos fibres operated interdependently to cause Mr Cotton's lung cancer. A proposition not different in any fundamental respect lay at the heart of the plaintiff's argument in this Court.

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10 (2008) 37 WAR 1 at 82 [319].

The synergistic or multiplicative effect of smoking and asbestos

32 It is convenient to examine the evidence that was given about the synergistic effect of tobacco smoke and asbestos inhalation by first examining the evidence of Dr Leigh. Argument in this Court proceeded on the footing that no other witness gave evidence that was more favourable to the plaintiff's arguments about synergistic effect.

33 Dr Leigh gave evidence about research into the possible biological mechanisms for the synergistic effect, and noted that some research suggested that tobacco smoke and asbestos act at different stages of the development of cancer. He expressed the opinion that:

> "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 singular result of the two factors acting together*. It is however true that exposure to either factor alone is capable of causing lung cancer." (emphasis added)

34 The meaning and effect of this passage of Dr Leigh's report was much debated in the course of the argument of the present appeals. In terms, the proposition advanced by Dr Leigh in this passage of his opinion was that *when* both smoking and asbestos cause cancer, the cancer is "the singular result of the two factors acting together". That proposition, if taken literally, would not address the question in issue in these cases. The question for decision in these cases is whether asbestos was a cause of the cancer.

35 Reading his opinion as a whole, it is evident that Dr Leigh did not express the view that, if a smoker has been exposed to asbestos and develops lung cancer, the asbestos exposure is, or is probably, *a* cause of that cancer. Dr Leigh expressly acknowledged not only that the precise mechanism of interaction between asbestos and tobacco smoke is not known, but also that it remains possible (for what he described as "legal or administrative" reasons) "to partition attributability to smoking and asbestos under a variety of mathematical risk models". To read the passage from his evidence about "singular result of the two factors acting together" as saying that, if a smoker has been exposed to asbestos and develops lung cancer, the asbestos exposure is necessarily (even probably) *a*

cause of the cancer, would not be consistent with his acknowledgement that the probability of causal connection can be divided ("partition[ed]") between the two carcinogens. As noted earlier, he assigned to asbestos a probability of causal connection of 2 to 12% (or 5 to 20% if higher exposure levels were taken).

36 Two further points reinforce the conclusion just described. First, and most importantly, the plaintiff did not submit that Dr Leigh's evidence should be understood in some way different from the understanding just described. Secondly, reading Dr Leigh's opinion as *not* going so far as to say that asbestos exposure is, or is probably, a cause of Mr Cotton's cancer is not only consistent with his dividing attribution of cause between the two carcinogens, it explains the way in which he stated the premises for making that division. Dr Leigh acknowledged that there is an *argument* that smoking and asbestos exposure are indivisible in the causation of an individual lung cancer. But he did not adopt that argument as his opinion. He said that:

> "Whilst it *can be argued* that asbestos exposure multiplies the risk of lung cancer, whatever the level of smoking, that smoking and asbestos exposure are indivisible in the causation of an individual lung cancer, and that any asbestos exposure multiplies the risk of lung cancer due to smoking by some quantity greater than one it is nevertheless possible ... to partition attributability". (emphasis added)

37 Dr Leigh's approach to the matter was consistent with that of Professor Musk, who accepted in cross-examination that, in considering what might have caused lung cancer in a person, "a medical and scientific way of approaching the question" was that "if there is a carcinogen present, then don't exclude it from the end result", "take it into consideration in looking at what might have caused [the patient's] lung cancer". But as explained earlier in these reasons, the courts' response to the uncertainty that results from absence of knowledge must differ from that of the medical practitioner or scientist.

Causation at trial

38 The trial judge identified the relevant question in a way which did not direct attention to whether the negligence of a particular defendant was a cause of Mr Cotton's cancer. The trial judge said[11] that the plaintiff would succeed:

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11 [2006] WASC 270 at [641].

> "if the evidence establishes that it is more probable than not that Mr Cotton's lung cancer was caused by asbestos arising from *one or both* of his periods of occupational exposure to that mineral or if it supports the conclusion, on the probabilities, that his cancer was caused to a material extent by the *combined* effects of his periods of asbestos exposure with the effects of his chronic smoking". (emphasis added)

Not only did this framing of the question not distinguish between defendants, it assumed (by its reference to "one or both" periods of exposure) that cumulative exposure to asbestos, by the negligence of more than one defendant, would be sufficient to establish the relevant causal connection between a defendant's negligence and the damage suffered.

39 Having thus identified the question to be answered, the trial judge restated[12] it by asking whether, "on the probabilities, Mr Cotton's two sequential periods of occupational exposure to asbestos, acting in combination with his habit of smoking, contributed to the development and/or progress of his fatal tumour". In his Honour's opinion[13], there was "really no answer to the evidence of Professors Musk, Wan, Dr Kendall, Professor de Klerk, Dr Leigh and Professor Berry that a not insignificant contribution to the *combined* causative effect was due to this asbestos exposure" (emphasis added). The conclusion that both periods of occupational exposure to asbestos made a "not insignificant" or "material" contribution to the onset or the development of Mr Cotton's cancer (or both its onset and development) was said[14] to be supported by "the probabilities [being] that the toxic effect of the carcinogens of tobacco and asbestos had synergistic effect, and that their effects were also cumulative upon previous exposures".

40 To approach the matter in this way diverted attention from whether individual defendants were to be found liable. It treated the relevant question as being: why did Mr Cotton develop lung cancer? But as pointed out earlier in these reasons, the relevant question, to be asked separately in respect of each

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12 [2006] WASC 270 at [687].

13 [2006] WASC 270 at [687].

14 [2006] WASC 270 at [689].

defendant, was: is it more probable than not that the negligence of the defendant was a cause of Mr Cotton's cancer? The difference between the questions is important. The litigation sought to attribute legal responsibility for Mr Cotton's cancer and death. Asking why Mr Cotton developed lung cancer could be understood as asking what aspects of his history would be considered significant, for their purposes, by a clinician, an epidemiologist, or an occupational health expert.

41 No separate attribution of responsibility as between defendants was made by the trial judge. Attempts made by some of the witnesses (particularly Professor Musk, Mr Kottek, Dr Leigh and Professor Berry) to estimate the proportions "by which each exposure may have contributed to the final fatal result" were dismissed[15] by the trial judge as "rather superficial and approximate". The trial judge concluded[16] that, because there were no proceedings between defendants seeking contribution or apportionment, it was unnecessary and undesirable to attempt to estimate those proportions.

42 As Martin CJ rightly pointed out[17] in his dissenting opinion in the Court of Appeal, the absence of contribution or apportionment proceedings was not to the point in considering the question of causation. The trial judge was required to decide the plaintiff's claim against each defendant by deciding whether the negligence of *that* defendant was a cause of Mr Cotton's cancer. Deciding only whether the *aggregate* exposure to asbestos was a cause of Mr Cotton's cancer did not answer the question about the particular responsibility of each defendant.

Causation in the Court of Appeal – the majority opinion

43 The hinge about which the reasoning of the majority turned was their acceptance[18] that the trial judge had found that "the medical evidence, reflected in the acknowledged synergistic effect of tobacco smoke and asbestos, [showed] that tobacco smoke and asbestos fibres operated inter-dependently and thus

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15 [2006] WASC 270 at [690].

16 [2006] WASC 270 at [690].

17 (2008) 37 WAR 1 at 52-53 [193].

18 (2008) 37 WAR 1 at 82 [319].

cumulatively to cause Mr Cotton's lung cancer". The majority of the Court of Appeal examined what they described[19] as "the statistical evidence" led at trial, including the evidence of Professor de Klerk, Dr Leigh and Professor Berry. But to the extent to which that evidence sought to identify the probability that asbestos was a cause of Mr Cotton's cancer, the majority appear to have treated the evidence as not to the point. Their Honours said[20] that:

> "Once it is accepted that smoking and asbestos are not independent but rather cumulative causes of lung cancer, the epidemiological evidence has no direct application to the question of causation at law because it is based on a false assumption."

Rather, their Honours continued[21]:

> "Once it is determined that tobacco smoking and all asbestos exposures operated cumulatively, the only remaining issue is whether each asbestos exposure made a material contribution."

44 The proposition made by the majority in the Court of Appeal, that the epidemiological evidence had no direct application to the question of causation, proceeded from the premise (explored in the immediately preceding and succeeding paragraphs of the reasons[22]) that Mr Cotton's smoking and his exposures to asbestos had operated "cumulatively" to cause his cancer. This, the majority concluded[23], had been established at trial by the evidence of Dr Leigh. Their Honours said[24]:

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19 (2008) 37 WAR 1 at 82 [319].

20 (2008) 37 WAR 1 at 86 [336].

21 (2008) 37 WAR 1 at 86 [336].

22 (2008) 37 WAR 1 at 85-86 [335], 86 [337].

23 (2008) 37 WAR 1 at 86 [337].

24 (2008) 37 WAR 1 at 86 [337].

> "Whether or not potential causes operate cumulatively is a matter of medical science. Only Dr Leigh [of the witnesses called at trial] is a specialist physician and epidemiologist. The other epidemiological experts do not have medical qualifications."

The plaintiff's arguments in this Court

45 Many of the issues which were agitated at trial, and determined by the trial judge, were not in dispute in this Court. In particular, it is not necessary to examine the controversies at trial, further considered in the Court of Appeal, about such matters as the extent to which Mr Cotton was exposed to respirable asbestos fibres[25], or whether concentrated exposure to fibres over small periods is more toxic than sustained exposure to smaller levels over longer periods[26]. Rather, as noted at the outset, arguments in this Court were confined to the issue of causation.

46 In arguing the appeals to this Court, counsel for the plaintiff did not support the first step in the reasoning of the majority in the Court of Appeal: that "the epidemiological evidence has no direct application to the question of causation". On the contrary, the arguments on behalf of the plaintiff in this Court depended in critically important respects upon the epidemiological evidence.

47 The whole tenor of the plaintiff's argument in this Court (as it appears to have been at trial and on appeal to the Court of Appeal) was first, that causation was established as a matter of inference not direct proof, and second, that the inference of causation was to be drawn from a proper understanding of the epidemiological evidence. Framing the argument in this way entailed rejecting a critical step in the reasoning of the majority in the Court of Appeal. It was not the plaintiff's argument in this Court that Dr Leigh's evidence (or the evidence of any other witness) should be understood as offering an opinion that, independent of epidemiological analysis, it could be concluded that exposure to asbestos *was* a cause of Mr Cotton's cancer.

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25 (2008) 37 WAR 1 at 9-34 [32]-[141] per Martin CJ.

26 (2008) 37 WAR 1 at 18-20 [74]-[78].

48 The plaintiff submitted that the majority's use of the word "cumulatively" (in the expression "tobacco smoke and asbestos fibres operated inter-dependently and thus *cumulatively*") was "unfortunate". In particular, so the plaintiff submitted, it should not be understood as a reference to the kinds of process that lead to a condition like boilermakers' deafness or other forms of progressive disease or injury. Rather, the plaintiff submitted, "cumulatively" should be understood as a reference to an interdependent (because synergistic or multiplicative) operation of the two carcinogens to which Mr Cotton was exposed – tobacco smoke and respirable asbestos fibres.

49 The proposition that there was an interdependent operation of the two carcinogens was an essential premise for the plaintiff's arguments in this Court. It was a premise that was said to be established by the epidemiological evidence. The proposition was reached by the following steps:

(a) Mr Cotton had been exposed to two carcinogens, tobacco smoke and respirable asbestos fibres.

(b) In ways that are not now known, tobacco smoke and asbestos work together to cause cancer "in the usual case".

(c) They must work together because more people who are exposed to both carcinogens contract lung cancer than would be expected from the incidence of lung cancer in those exposed only to smoking and the incidence of lung cancer in those exposed only to asbestos.

From this proposition about interdependence, the plaintiff's argument proceeded to the conclusion that causation was to be inferred by taking three further steps:

(d) There was no alternative explanation or differentiating factor suggested by the medical, social or personal history for Mr Cotton.

(e) The only two explanations for Mr Cotton developing cancer that need to be considered are that his smoking was the sole cause of his cancer, or that the combined effect of asbestos and smoking caused his cancer.

(f) Because exposure to both tobacco smoke and asbestos is more dangerous than exposure to one or the other, it is more probable than not that exposure to both was the cause of his cancer and his exposure to asbestos

was a not insignificant cause of (made a material contribution to) his developing cancer.

50 The plaintiff sought to draw some support for the conclusion that asbestos was probably a cause of Mr Cotton's cancer by pointing out that the age at which he developed cancer was at or near the start of the range of ages at which cancer might be expected to develop in a person who had smoked to the extent he had. But given that Mr Cotton fell within the range of ages (albeit at the younger end of that range) the observation does not support the conclusion asserted.

Causation not established

51 The question to which the plaintiff sought to have an affirmative answer inferred was, in each case: is it more probable than not that the negligence of the defendant was a cause of Mr Cotton's cancer? Observing only that exposure to asbestos *may* have been a cause does not answer that question affirmatively.

52 Three steps in the plaintiff's argument require close consideration: first, that smoking and asbestos *must* work together; second, that the only two explanations of Mr Cotton's cancer that need be considered are smoking as the sole cause and the combined effect of both smoking and asbestos; and third, that because exposure to both is more dangerous than exposure to one or the other, exposure to both was probably the cause of Mr Cotton's cancer.

Smoking and asbestos *must* work together?

53 The proposition that smoking and asbestos *must* work together to cause cancer was not a proposition established by opinion evidence. As explained earlier, Dr Leigh accepted that the proposition was one that "can be argued", but he did not adopt it as his opinion. We were taken to no other opinion evidence to the effect that, if there has been exposure to the two carcinogens, and a person develops lung cancer, the two *must* have worked together.

54 It may be accepted (at least for the purposes of debate) that the synergistic or multiplicative effect suggests that in some cases the two carcinogens will have contributed to the development of an individual patient's cancer. But the proposition which the plaintiff advanced was an absolute proposition of universal application: smoking and asbestos *must* work together and they must have worked together in this case. That proposition was not established.

Consider only smoking or combination of smoking and asbestos?

55 The second of the plaintiff's propositions requiring close consideration is that the only two relevant explanations of Mr Cotton's cancer that need be considered are smoking as sole cause, and the combined effect of both smoking and asbestos. The plaintiff submitted that all other possible causes of Mr Cotton's cancer were to be dismissed from consideration as improbable. This confinement of the field for consideration was said to follow from the way in which the case had developed and been argued at trial.

56 It may greatly be doubted that argument at trial can be understood as having confined the field for debate in the manner asserted. Rather, the plaintiff's assertion is better understood as no more than an observation that the evidence of epidemiological studies established that most who developed lung cancer had been smokers and that the next highest group was formed by those who had been both smokers and exposed to asbestos. But if conclusions are to be drawn from population studies, all of the results obtained for all possible causes of the cancer must be considered. To consider whether one of two circumstances is more "dangerous" than another must not be permitted to obscure examination of the relative dangers of all causes.

57 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. The plaintiff sought to provide that foundation by the third of the steps to which close attention must be given: that because exposure to both smoking and asbestos is more dangerous than exposure to one or the other on its own, it follows that it is probable that exposure to asbestos was a cause of Mr Cotton's cancer.

"More dangerous"?

58 The basis on which the description "more dangerous" was attached to exposure to both smoking and asbestos was not identified in argument. At least in part it seems to have been used as a qualitative description of the synergistic effect examined earlier in these reasons. But the critical question is whether the exposure to asbestos was a cause of Mr Cotton's cancer.

59 To the extent to which the plaintiff's description "more dangerous" was intended to reflect some quantitative measure of the probability that one

carcinogen rather than another was a cause of cancer, it was a proposition not supported by the evidence. None of the witnesses whose evidence is examined earlier in these reasons assigned a probability greater than 23% to the chance that Mr Cotton's cancer was caused by exposure to asbestos (whether alone or in combination with smoking). Professor Berry put that probability as low as 1%; Dr Leigh put it at between 2 and 12% (or 5 and 20% if higher exposure figures were used). Professor de Klerk considered the probability that Mr Cotton's cancer was due to asbestos exposure alone was only 3% and due to exposure to both was 20%. The witnesses who expressed an opinion on the matter agreed that the probability that Mr Cotton's cancer was caused by smoking alone was high (Professor de Klerk said 67%; Professor Berry said 92%).

60 If the description of exposure to smoking and asbestos as "more dangerous" than exposure to one or the other was intended to reflect a quantitative comparison of risk, it is a description that did not accurately reflect the evidence given by the witnesses about the relative risks of smoking compared with the relative risks of exposure to asbestos. And if the description "more dangerous" was intended to convey no more than that those who were exposed to both smoking and asbestos were at greater risk of developing cancer than those who were exposed to only one of those carcinogens, it is necessary to bear steadily in mind that the evidence did *not* establish that smoking and asbestos *must* work together.

61 This third step in the plaintiff's argument was not made good.

62 As explained at the commencement of these reasons, there being no direct evidence about what actually caused Mr Cotton's cancer, it was the plaintiff's case that the epidemiological evidence established facts which "positively suggest[ed], that is to say provide[d] a reason ... for thinking it likely"[27] that, in exposing Mr Cotton to respirable asbestos fibres, the negligence of each defendant was a cause of his cancer. To draw an inference about causation from what was established by the epidemiological studies, it would be necessary to decide whether the particular case under consideration should be treated as conforming to the pattern described by the epidemiological studies. Absent evidence which suggests that the individual may stand apart from the ordinary, there may be sufficient reason to assume conformity, but whether or not that is

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27 *Jones v Dunkel* (1959) 101 CLR 298 at 305.

so, it is important to recognise that the first step that must be taken, if an inference is to be drawn from epidemiological studies, is to relate the results of studies of populations to the particular case at hand. That step is not inevitable[28].

63 In this case, even if it were decided that Mr Cotton's case was not atypical, application of either the relative risk or the probability analyses given in evidence does not found the inference that the plaintiff sought to have made. The probabilities and distributions recorded in the epidemiological studies would point away from the conclusion urged by the plaintiff.

64 Although the witnesses differed about the particular values that should be assigned to the relative risks and probabilities derived from epidemiological studies, it is not necessary in this case to choose between them. It is enough to recognise that, whichever figures are taken as the best expression of the results of epidemiological studies:

(a) the relative risk of Mr Cotton developing his cancer from exposure to respirable asbestos fibres (whether alone or in combination with smoking) was much lower than the relative risk of his smoking as he did;

(b) the probability of Mr Cotton developing his cancer, if exposed to asbestos, was much lower than the probability of his developing his cancer from smoking as he did.

If the relative risks and probabilities derived from epidemiological studies were to be treated as revealing what was a probable explanation of what caused Mr Cotton's cancer, those analyses support two conclusions. First, it is more probable than not that smoking was a cause of (in the sense that it was a necessary condition for) Mr Cotton's cancer. Second, the risks and probabilities associated with asbestos, whether alone or in conjunction with smoking, are low and not sufficient to found the inference which the plaintiff sought to have made: that it is more probable than not that exposure to respirable asbestos fibres was a cause of Mr Cotton's cancer.

65 It was not shown to be more probable than not that asbestos was a cause of (a necessary condition for) his cancer. It was not shown that exposure to

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28 *State Government Insurance Commission v Laube* (1984) 37 SASR 31 at 32-33.

asbestos made a material contribution to his cancer. Material contribution was not shown because a connection between Mr Cotton's inhaling asbestos and his developing cancer was not demonstrated.

The relevance of material contribution

66 The plaintiff made a deal of reference to the decision of the House of Lords in *Bonnington Castings Ltd v Wardlaw*[29] and, in particular, the statement of Lord Reid[30] that:

> "What is a material contribution must be a question of degree. A contribution which comes within the exception de minimis non curat lex is not material, but I think that any contribution which does not fall within that exception must be material."

Particular attention was given to what was meant by saying that any contribution that was not de minimis must be material.

67 It is important to recognise the context in which this statement was made. The issue in *Bonnington Castings* was whether exposure to silica dust from poorly maintained equipment caused or contributed to the pursuer's 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[31], 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.

68 This description of the issue of causation in *Bonnington Castings* shows how different it is from the issue of causation in this case. The issue in *Bonnington Castings* was whether one source of an injurious substance

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29 [1956] AC 613.

30 [1956] AC 613 at 621.

31 [1956] AC 613 at 621.

contributed to a gradual accumulation of dust that resulted in disease. The issue here is whether one substance that *can* cause injury *did* cause injury. Or, to adopt and adapt what Starke J said in *Adelaide Stevedoring Co Ltd v Forst*[32], was Mr Cotton's cancer "intimately connected with and contributed to" by his exposure to asbestos? Questions of material contribution arise only if a connection between Mr Cotton's inhaling asbestos and his developing cancer was established. Knowing that inhaling asbestos *can* cause cancer does not entail that in this case it probably *did*. For the reasons given earlier, that inference was not to be drawn in this case. Questions of what is a material contribution do not arise.

A paradoxical result?

69 It was submitted that the conclusion that causation was not established in this case entailed a paradox. If consideration of the results of the population studies described in evidence in this matter does not permit the inference that Mr Cotton's cancer was caused or contributed to by exposure to asbestos, no claim by an individual in Mr Cotton's position could succeed. And yet, the argument continued, the population studies showed that exposure to asbestos was a cause of cancer in some cases. How then could it be right to reach a result that entailed the corollary that all individual claims would fail?

70 The answer to the question can be expressed in several different ways. All depend upon the basic and unpalatable fact that no scientific or medical examination can now say, with certainty, what caused Mr Cotton's cancer or lung cancer in any other particular case. As explained at the outset of these reasons, despite this uncertainty, the courts must, and do, "reduce to legal certainty [a question] to which no other conclusive answer can be given"[33]. The courts do that by asking whether it is more probable than not that X was a cause of Y. Saying only that exposure to asbestos *may* have been a cause of Mr Cotton's cancer is not a sufficient basis for attributing legal responsibility. 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

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32 (1940) 64 CLR 538 at 567; [1940] HCA 45.

33 *Bank of NSW v The Commonwealth* (1948) 76 CLR 1 at 340.

the drawing of an inference in a particular case. The paradox, if there be one, arises from the limits of knowledge about what causes cancer.

Conclusion and orders

71 For these reasons each appeal should be allowed with costs.

72 Each appellant's notice of appeal in this Court seeks orders setting aside only the orders of the Court of Appeal made on 26 September 2008. By its orders of 26 September 2008 the Court of Appeal dismissed the appeals to that Court by the State of South Australia and Millennium, but stood over for further consideration the making of orders dealing with Amaca's appeal and the making of orders dealing with the costs of all three appeals to the Court of Appeal. Those matters were dealt with by orders made on 29 January 2009. In addition, each appellant's notice of appeal in this Court seeks the setting aside of orders of Heenan J made on 8 December 2006 but no other order made at first instance. The orders of 8 December 2006 provided, in effect, for judgment for the plaintiff but reserved the costs of the action. The papers made available to this Court do not show whether any later order was made at first instance dealing with those costs.

73 If an appellant seeks consequential orders which deal with orders in the courts below, other than the orders of the Court of Appeal made on 26 September 2008 and the orders made at first instance on 8 December 2006, that appellant will require leave to amend its notice of appeal.

74 Each appellant should now have an opportunity to consider whether it wishes to seek leave to amend. And in any event, all of the parties to the appeals should now have an opportunity to consider what further orders this Court should make beyond allowing each appeal with costs. There should be orders in each appeal in the following terms:

1. Appeal allowed with costs.

2. Within 14 days of the date of this judgment the appellant should file and serve any proposed amended notice of appeal.

3. Within 28 days of the date of this judgment either the parties should file agreed minutes of the consequential orders they seek to have this Court make, or, in default of agreement, each party should file, and serve on the

opposite party, written submissions setting out the consequential orders which that party submits that this Court should make, together with any submissions which the party makes in support of, or in opposition to, any proposed amendment of the notice of appeal.