

Before the High Court

Mind the ‘Evidential Gap’:[†] Causation and Proof in *Amaca Pty Ltd v Ellis*^{††}

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Abstract

In *Ellis* the plaintiff sought compensation for the deceased’s lung cancer on the basis of his work-place asbestos exposure. But a far greater risk had been posed by the deceased’s smoking. Martin CJ, dissenting, concluded that causation was not proven to the requisite standard. The trial judge and the majority on appeal relied upon a number of arguments and doctrines in finding causation, however, none appears very sound. Their rejection of the epidemiological evidence is unwarranted. Contrary to the majority’s claim, *Ellis* is not a case of alternative sufficient causes. There is uncertain support for the trial judge’s presumption of causation from breach, and strong rebutting evidence. The House of Lords’ exposure to risk principles have not yet been adopted in Australia, and, in any event, would be inapplicable to *Ellis*. The most authoritative basis for causation may be that asbestos exposure made a material contribution, cumulatively with the deceased’s smoking. However, this operation of the material contribution doctrine should be rejected by the High Court. It does too much work with too little evidence, with potentially arbitrary results.

1. Introduction

Paul Steven Cotton died of lung cancer. At trial, the plaintiff, the executor of his estate, obtained compensation from the three defendants.¹ Two, South Australia and Millennium Inorganic Chemicals Ltd, were employers who had exposed the deceased to asbestos dust in the workplace. The third, Amaca Pty Ltd, had manufactured some of the asbestos products. Causation was a central issue. The asbestos exposures were relatively light, and the deceased had been a heavy smoker all of his adult life. However, the trial judge still held the appellants liable.²

[†] *McGhee v National Coal Board* [1973] 1 WLR 1 (‘*McGhee*’), 7 (Lord Wilberforce).

^{††} [2009] HCA Trans 77 (special leave to appeal granted).

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1 *Ellis v South Australia* [2006] WASC 270 (‘*Ellis (No 1)*’).

2 There was a 10% reduction in Amaca’s common law damages for contributory negligence, though no reduction in the damages of the other defendants for breach of statutory duty or contract: *Ellis (No 1)* [2006] WASC 270, [811].

In the Western Australian Court of Appeal, Steytler P and McLure JA upheld liability.³ However, Martin CJ provided a strong dissent. The deceased's smoking posed a far greater risk of lung cancer than the asbestos exposure and Martin CJ held that causation had not been proven on the balance of probabilities. The majority, however, held that the lung cancer was the cumulative product of both carcinogens and that their effects could not be separated. The asbestos made a material contribution to the cancer.

The High Court granted all three defendants special leave to appeal on 1 May 2009.⁴ The case raises complex questions about the interrelationship between epidemiological evidence of risk and the law of causation and proof. Is it necessary for 'but for' causation to be proven on the balance of probabilities? Or is does the material contribution doctrine override the 'but for' test where the exposures operate cumulatively? Should the plaintiff be assisted by the presumption of causation from breach? Should mere exposure to risk be sufficient to satisfy the causation requirement? These are issues on which existing High Court authority is unclear, State courts disagree, and there have recently been dramatic developments in the House of Lords.

The High Court should approach these questions without taking refuge in the mantra that it is 'all ultimately a matter of common sense'.⁵ Common sense is not immutable and monolithic. To quarantine it from analysis 'seems a counsel of despair'.⁶ It may be true that 'questions of cause and consequence are not the same for law as for philosophy and science',⁷ but these other disciplines provide a useful foundation for legal judgment. As this case vividly illustrates, to assess causation without properly engaging with the scientific and philosophical issues they raise can invite fictional inferences,⁸ and factual and legal indeterminacy.⁹

2. *The Epidemiological Data and the 'But For' Test*

Epidemiological evidence is crucial to understanding causation in exposure cases like *Ellis*.¹⁰ Epidemiology is the statistical study and analysis of the incidence of disease. For example, the rate of lung cancer sufferers among people aged 40 and

3 *South Australia v Ellis* (2008) 37 WAR 1 ('*Ellis* (No 2)'). The contributory negligence discount was increased to 50%: *Ellis* (No 2) (2008) 37 WAR 1, [498].

4 *Amaca Pty Ltd v Ellis* [2009] HCA Trans 77.

5 *March v E & M H Stramare Pty Ltd* (1991) 171 CLR 506 ('*March*'), 515 (Mason CJ), quoting *Fitzgerald v Penn* (1954) 91 CLR 268, 277 (Dixon CJ, Fullagar and Kitto JJ).

6 H L A Hart and Tony Honoré, *Causation in the Law* (2nd ed, 1985) 26.

7 *March* (1991) 171 CLR 506, 509 (Mason CJ), quoting *National Insurance Co of New Zealand Ltd v Espagne* (1960) 105 CLR 569, 591 (Windeyer J).

8 An invitation declined in *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 ('*Fairchild*'), 68 (Lord Bingham), 71 (Lord Nicholls), 106–107, 110–111 (Lord Rodger).

9 *March* (1991) 171 CLR 506, 532–533 (McHugh J); *Fairchild* [2003] 1 AC 32, 72 (Lord Hoffmann); *Chester v Afshar* [2005] 1 AC 134 ('*Chester*'), 161 (Lord Hope); *Travel Compensation Fund v Tambree* (205) 224 CLR 627–8 (Gummow and Hayne JJ).

10 It was not much of an exaggeration for Martin CJ in *Amaca Pty Ltd v Hannell* (2007) 34 WAR 109 (hereafter *Hannell*) at 121 to suggest that 'epidemiological studies are currently the only basis upon which an assessment of causative effect can currently be undertaken'.

above, with no known specific exposures,¹¹ is about 14 out of 100,000.¹² The rate among heavy smokers, like the deceased in *Ellis*, is about 220 per 100,000. This increase is measured by the notion of ‘relative risk’. The relative risk of lung cancer given tobacco exposure is $RR_T = \frac{220}{14} = 15.5$

If such a smoker then contracted lung cancer, the relative risk can be used to calculate the probability that tobacco was the cause rather than background carcinogens. The apportionment is achieved by dividing the total risk between the two possible causes, tobacco and background factors. RR_T represents the total relative risk given the tobacco exposure. This incorporates both the background risk, with base level 1, and the additional risk from tobacco, $RR_T - 1$. Expressed as fractions, the attributable risk¹³ for tobacco is $(RR_T - 1)/RR_T$, and the attributable risk for background exposure is $1/RR_T$.¹⁴ These add up to one and can be interpreted as the probability that one or the other was the cause. In general, it will be more probable than not that a specific exposure is the cause, rather than background exposure, where the specific exposure more than doubles the risk: $RR > 2$.¹⁵ With the figure given above, $RR_T = 15.5$, there is a 93.5% probability that smoking caused the individual’s lung cancer, against a 6.5% probability that the cancer was caused by exposure to background carcinogens.

In *Ellis* the picture is more complicated as there were two specific exposures, tobacco and asbestos.¹⁶ Expert evidence was provided of the relative risks of tobacco, asbestos, and, most problematically, the two in combination. These figures and their interpretation were highly contested. One of the appellant’s

11 As Martin CJ points out in *Hannell* the ‘background’ group would include those subject to a specific exposure without realizing or remembering it: Id 116-117.

12 MJ Thun, LM Hannan, LL Adams-Campbell, P Boffetta, JE Buring *et al*, ‘Lung Cancer Occurrence in Never-Smokers: An analysis of 13 cohorts and 22 cancer registry studies’, (2008) 5(9) *PLoS Med* 1357 at 1364.

13 See Sander Greenland, ‘Relation of Probability of Causation to Relative Risk and Doubling Dose: A Methodological Error that has become a Social Problem’ (1999) 89 *American Journal of Public Health* 1166, 1167.

14 The trial judge’s explanation of this formula bears more resemblance to Laplace’s rule of succession than it does to the proper derivation: *Ellis (No 1)* [2006] WASC 270, [374], [383].

15 This of course is purely on the basis of the relative risk evidence. Where there is other evidence of causation, the relative risk should be considered along with that. Compare *Seltsam Pty Ltd v McGuinness* (2000) 49 NSWLR 262 (‘*Seltsam*’), 280–285 (Spigelman CJ). Wholly inconsistent with the relative risk approach is the very recent decision in *Sydney South West Area Health Service v Stamoulis* [2009] NSWCA 153 (‘*Stamoulis*’). Medical negligence delayed the diagnosis of cancer, increasing the risk of metastasis by about 10%. This was based upon epidemiological evidence of an increased risk from 38% to 42%. Unfortunately, metastasis did occur. Ipp JA, with whom Beazley and Giles JJA agreed, reasoned that prior to the negligence, metastasis was improbable. The negligence delayed the diagnosis, the risk went up, and then metastasis occurred. The delay must have tipped the scales. This comes close to reasoning *post hoc ergo propter hoc*. Ipp JA gave no consideration to the relative risk argument that, on these figures, there is a 90% probability that the metastasis was the consequence of the pre-existing tumour, uninfluenced by the delayed diagnosis. Apparently, Ipp JA’s reasoning was not the result of his distrust of statistics: see below n 23. His reasoning was essentially the same both on a statistical and individualistic interpretation of the 10% figure: *Stamoulis* [2009] NSWCA 153, [146], [150].

epidemiologists, Mr Rogers, in the present case arrived at the attributable risks in Table One.¹⁷ The cancer was most probably caused by tobacco alone (93.31%), but could have been caused by background exposure (6.44%). The probability that the cancer was caused by asbestos alone or in combination with tobacco was rated very low (0.26%). The plaintiff’s expert, Professor de Klerk, gave the attributable risks in Table Two.¹⁸ In the Western Australian Court of Appeal Martin CJ, dissenting, expressed a preference for Rogers’ figures.¹⁹ But even on de Klerk’s figures, there is still a 77% probability that the deceased would have contracted lung cancer without the asbestos exposure. This led Martin CJ, applying the ‘but for’ test, to hold that the plaintiff had not established causation on the balance of probabilities.²⁰

Background	6.44
Tobacco (alone)	93.31
Asbestos (alone)	0.02
Tobacco and asbestos (synergistic)	0.24
Total	100

Table One: Rogers’ Attributable Risk

Background	10
Tobacco (alone)	67
Asbestos (alone)	3
Tobacco and asbestos (synergistic)	20
Total	100

Table Two: de Klerk’s Attributable Risk

16 In fact, there were two separate asbestos exposures, one each from the two employers, but, for present purposes, this complication can be put to one side.

17 *Ellis (No 2)* (2008) 37 WAR 1, [202].

18 *Ellis (No 2)* (2008) 37 WAR 1, [324].

19 *Ellis (No 2)* (2008) 37 WAR 1, [141], [202].

20 *Ellis (No 2)* (2008) 37 WAR 1, [252].

3. *Additive and Multiplicative Epidemiological Models*

On appeal the majority in *Ellis v Amaca Pty Ltd*²¹ (*‘Ellis (No 2)’*) gave a number of reasons for rejecting the figures derived from the epidemiological models, and Martin CJ’s ‘but for’ interpretation of them. At the most general level, the majority dismissed the evidence as irrelevant: ‘[e]pidemiological evidence is purely statistical, probabilistic evidence, that says nothing about the actual cause of a disease in a specific case.’²² This should be recognised as an exaggeration. As Lord Nicholls recently observed in *Gregg v Scott*,²³ ‘[t]he value of the statistics will of course depend upon their quality ... But to reject all statistical evidence out of hand would not be acceptable ... When there is nothing better courts should be able to use these figures and give them such weight as is appropriate in the circumstances.’²⁴

The majority in *Ellis (No 2)* provided other grounds for rejecting the epidemiological models. One was the claim that ‘the statistical analyses performed by the epidemiologists wrongly assume that tobacco smoke and asbestos are independent causes of lung cancer.’²⁵ They agreed with the trial judge that the models are not appropriate ‘for dependent or related causes acting in combination.’²⁶ But this misunderstands epidemiology in general and the epidemiological evidence given in the case. Models can be constructed to reflect different assumptions about the interaction between causal agents. Two common variations are additive and multiplicative models.²⁷ The first of these corresponds with the causal agents operating independently. The latter incorporates a positive synergistic interaction.

Consider, first, the additive model. As explained above, the relative risk for tobacco is RR_T , to which tobacco contributes $RR_T - 1$, and background risk, 1. Similarly, for asbestos, the relative risk is RR_A , with asbestos contributing $RR_A - 1$, and background risk, 1. Where there are both kinds of exposure, the additive model assumes that the total risk is simply a sum of the risks from the three sources taken separately. The contributions to the total relative risk and the attributable fractions for the additive model are given in Table Three.

The multiplicative model assumes that, on top of these contributions, there is a further contribution from synergistic interaction of tobacco and asbestos. The size

21 (2008) 37 WAR 1.

22 *Ellis (No 2)* (2008) 37 WAR 1, [304] (emphasis added). The majority made an identical statement in *Hannell* (2007) 34 WAR 109, 188. See also *Seltsam* (2000) 49 NSWLR 262, 274 (Spigelman CJ); *Stamoulis* [2009] NSWCA 153, [135]–[138] (Ipp JA).

23 [2005] 2 AC 176 (*‘Gregg’*).

24 *Gregg* [2005] 2 AC 176, 187. See also *Gregg* [2005] 2 AC 176, 215–216 (Lord Phillips); *Ellis (No 2)* (2008) 37 WAR 1, [240] (Martin CJ); *Seltsam* (2000) 49 NSWLR 262, 276 (Spigelman CJ); *Hannell* (2007) 34 WAR 109, 121–123 (Martin CJ).

25 *Ellis (No 2)* (2008) 37 WAR 1, [496] (emphasis added). See also *Hannell* (2007) 34 WAR 109, 198 (Steytler P and McLure JA), 160 (Martin CJ).

26 *Ellis (No 1)* [2006] WASC 270, [380], quoted with approval in *Ellis (No 2)* (2008) 37 WAR 1, [334], see also [336].

27 See Amy Berrington de González and David R Cox, ‘Additive and Multiplicative Models for the Joint Effect of Two Risk Factors’ (2005) 6 *Biostatistics* 1.

of this is calculated as the multiplicative product of the individual contributions. The parameters of the multiplicative model are provided in Table Four.

In *Ellis*, contrary to the suggestions of the trial judge and the majority on appeal, the epidemiologists took account of the synergistic effect of tobacco and asbestos, and provided figures based upon a multiplicative model.²⁸ The risk attributable to asbestos under these models, either alone or in combination with tobacco, is many times more than the risk attributable to asbestos under the additive model. For example, on de Klerk’s figures, $RR_T = 7.7$ and $RR_A = 1.3$, the attributable risk for asbestos increases from 3.75% on the additive model to 23% (asbestos alone or in combination with tobacco) on the multiplicative model.²⁹ With Rogers’ figures, $RR_T = 15.5$ and $RR_A = 1.0026$, the corresponding increase is from 0.02% to 0.26%.³⁰

	contribution to RR	attributable fraction
background	1	$\frac{1}{RR_T + RR_A - 1}$
tobacco	$RR_T - 1$	$\frac{RR_T - 1}{RR_T + RR_A - 1}$
asbestos	$RR_A - 1$	$\frac{RR_A - 1}{RR_T + RR_A - 1}$
total	$RR_T + RR_A - 1$	1

Table Three: Additive Model

28 The relative risk and attributable risk figures of the various experts conform to the parameters of Table Four, other than those of Dr Leigh which diverge slightly: *Ellis (No 2)* (2008) 37 WAR 1, [324] (de Klerk); [331] (Berry); [202], [333] (Rogers); [326]–[329] (Leigh). However, the derivation of the models is poorly explained in the judgments, and de Klerk’s explanation appears to be the source of the confusion between additive and multiplicative models. De Klerk states his calculations reflect the assumption that the causes are ‘independent and therefore multiplicative’: [325]. The majority then go on to suggest, wrongly, ‘the statistical multiplicative model assumes that asbestos and tobacco smoking are independent carcinogens’: [326].

29 *Ellis (No 2)* (2008) 37 WAR 1, [324]. It is interesting to note that some epidemiologists then apportioned the combined attributable risk between the separate causes, producing figures for the contribution of asbestos very similar to those of the additive model. See *Ellis (No 1)* [2006] WASC 270, [369] (Berry), [323] (Rogers); *Ellis (No 2)* (2008) 37 WAR 1, [332] (Berry). Even Dr Leigh gave figures for such a breakdown: *Ellis (No 1)* [2006] WASC 270, [357], [369]; *Ellis (No 2)* (2008) 37 WAR 1, [329].

30 *Ellis (No 1)* [2006] WASC 270, [315].

	contribution to RR	attributable fraction
background	1	$\frac{1}{RR_T \cdot RR_A}$
tobacco	$RR_T - 1$	$\frac{RR_T - 1}{RR_T \cdot RR_A}$
asbestos	$RR_A - 1$	$\frac{RR_A - 1}{RR_T \cdot RR_A}$
tobacco and asbestos	$(RR_T - 1)(RR_A - 1)$	$\frac{(RR_T - 1)(RR_A - 1)}{RR_T \cdot RR_A}$
total	$RR_T \cdot RR_A$	1

Table Four: Multiplicative Model

And yet the majority agreed with the trial judge that the models displayed a ‘major, and significant, fallacy’.³¹ According to their understanding of the medical and epidemiological evidence, the combined-effect risk should have been the largest, but in the epidemiological models it is the second smallest.³² The trial judge indicated this was an inevitable though ‘enigmatic’ effect of mathematics.³³ Since a probability has a maximum of one, the product of two probabilities can be no larger than either of its factors, and usually smaller.

The trial judge is right about the multiplication of probabilities. But, as explained above and shown in Table Four, the multiplicative model is based on relative risks, not probabilities. The risk contribution of two exposures acting together is the product of their individual risk contributions. An exposure contributing a greater risk than background exposure will have a risk figure greater than one. In *Ellis*, the tobacco-alone risk was greater than one, but the asbestos-alone risk was less than one. The combined-effect risk therefore was larger than the asbestos-alone risk, but smaller than the tobacco-alone risk. This does not display a fallacy. It simply reflects the fact that the asbestos exposure in this case was relatively slight. Had both single-agent risks been greater than background risk, then the combined-effect risk, on the multiplicative model, would have been the largest.³⁴

31 *Ellis (No 1)* [2006] WASC 270, [380], quoted in *Ellis (No 2)* (2008) 37 WAR 1, [334] (Steytler P and McLure JA) with approval, and [209] (Martin CJ) with disapproval.
32 The trial judge suggested combined group was the ‘smallest’, but as the figures in the text show it is considerably larger than the asbestos-only group: *Ellis (No 1)* [2006] WASC 270, [380].
33 *Ellis (No 1)* [2006] WASC 270, [374].
34 See Per Gustavsson et al, ‘Low-Dose Exposure to Asbestos and Lung Cancer: Dose-Response Relations and Interaction with Smoking in a Population-Based Case-Referent Study in Stockholm, Sweden’ (2002) 155 *American Journal of Epidemiology* 1016, 1019.

A final misunderstanding is revealed by the majority's comment that 'epidemiological evidence ... [is] irrelevant to the central issue ... of whether the carcinogens [are] ... alternative causes or cumulative causes ... That is a medical question.'³⁵ Actually, epidemiology can say a great deal about this question by comparing the predictions of models with the incidence in actual populations. For example, a recent article in *Biostatistics* considers the sample sizes required 'to detect departures from an additive model ... in the multiplicative direction and from a multiplicative model ... in the additive direction'.³⁶ And the abstract of a recent article in the *American Journal of Epidemiology* notes how actual incidence related to additive and multiplicative predictions:

This population-based case-referent study investigated the lung cancer risk associated with occupational exposure to asbestos, focusing on dose-response relations and the interaction with tobacco smoking. ... The joint effect of asbestos and smoking was estimated to be 1.15 ... times that predicted from the sum of their individual effects and 0.31 ... times that predicted from their product, indicating a joint effect between additivity and multiplicativity.³⁷

Clearly, epidemiological models can take account of the synergistic effect of tobacco and asbestos. The epidemiological evidence in *Ellis* was not premised on the carcinogens operating independently. The trial judge and the majority should not have disregarded the epidemiological evidence on this basis.

4. Cumulative Material Contribution versus 'But For' Causation

The trial judge and majority misunderstood the construction and operation of the epidemiological models, and overstated the models' limitations. Nevertheless it is true that such models only provide a limited perspective on the aetiology of disease. For a more complete view, the pathology of the disease must also be considered. The trial judge and the majority relied upon the evidence of the pathological development of lung cancer as a further basis for rejecting the 'but for' interpretation of the epidemiological models.

The epidemiological models operate on a population of lung cancer sufferers that are assumed to have been subject to similar exposures to the deceased. This population is then partitioned into four separate groups according to the type of exposure that is supposed to have caused the lung cancer — background, tobacco-only, asbestos-only, and asbestos and tobacco combined. But at the pathological level, the causes may not partition so neatly. Asbestos exposure may have contributed to the lung cancer of some sufferers in all the groups. The trial judge and the majority relied upon the evidence of Dr Leigh who testified:

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

35 *Ellis* (No 2) (2008) 37 WAR 1, [310]. See also [337].

36 de González and Cox, above n 27, 4.

37 Gustavsson et al, above n 34, 1016.

than not, that in this situation, the lung cancer was the singular result of the two factors acting together.³⁸

Martin CJ indicated that this evidence was ‘contrary to the position of all other experts’.³⁹ But as the majority pointed out, ‘[o]nly Dr Leigh is a specialist physician and epidemiologist. The other epidemiological experts do not have medical qualifications.’⁴⁰

The force of Dr Leigh’s evidence is that lung cancer could well have a cumulative aetiology. In broad terms, this is one of three basic causal mechanisms.⁴¹ First, there are divisible diseases where every dose has the potential to cause damage. The total damage is in proportion to the total number of doses, and each tortfeasor can be held liable in proportion to the number of doses they are responsible for.⁴² Asbestosis and pneumoconiosis are divisible diseases.

Lung cancer is an indivisible disease. Once it takes hold, its development and extent bears no relation to the number of doses that the sufferer was exposed to, and subsequent doses are superfluous.⁴³ In broad terms there are two possibilities as to how an indivisible disease starts.⁴⁴ One is that the disease may be the effect of a single dose. Each dose is an alternative possible cause, capable of operating independently. The risk of disease is in proportion to the total exposure.

The final possibility, where the causes operate cumulatively, is more complex. More than a single dose is required for the onset of the disease. For example, the disease may only commence when an initial threshold of damage is passed, with all doses having the potential to contribute to the damage. In such a case, even very slight exposures may have made some contribution to the onset of the disease.

The alternative and cumulative causal mechanisms correspond to a degree with the two epidemiological models discussed above. The additive model’s linear relationship between exposure and risk is consistent with single doses operating independently and in the alternative. However, a multiplicative incidence is indicative of a synergy. The risk of combined exposures is greater than the sum of the parts, ruling out an independent alternative aetiology.

Where an independent alternative aetiology is operating, it is relatively unproblematic to take the attributable fraction as the probability of causation. The cause was a single dose. All else being equal,⁴⁵ the likelihood that a particular exposure was *the* cause of the disease will be in proportion to the number of doses

38 *Ellis (No 1)* [2006] WASC 270, [354], [466]; *Ellis (No 2)* (2008) 37 WAR 1, [335].

39 *Ellis (No 2)* (2008) 37 WAR 1, [208].

40 *Ellis (No 2)* (2008) 37 WAR 1, [337].

41 Of course, this is a simplistic and one-dimensional perspective on disease aetiology. There are many variations within the three categories, and there are also totally different schemes of categorisation, such as deterministic and probabilistic: see below n 68–75.

42 *Barker v Corus UK Ltd* [2006] 2 AC 572 (*‘Barker’*), 587–588 (Lord Hoffmann), 606 (Lord Rodger).

43 Just as subsequent sperm are superfluous once the egg has been fertilised: *Hannell* (2007) 34 WAR 109, 159.

44 See *Bryce v Swan Hunter Group plc* [1988] 1 All ER 659, 665; *McGhee* [1973] 1 WLR 1, 3–4; *Ellis (No 2)* (2008) 37 WAR 1, [311].

it contained. An exposure of less than half the total number of doses is unlikely to have made any contribution to the causation of the disease. However, where the aetiology is cumulative and synergistic, the equation between attributable fractions and the probability of causation is contestable. No longer is there a search for *the* causal dose. The disease was the result of a number of doses operating together, and even a relatively small exposure may have made some contribution to the disease.

The majority in *Ellis (No 2)* cited *Bonnington Castings Ltd v Wardlaw*⁴⁶ as authority that where causes operate cumulatively, any exposure above *de minimis* will constitute a material contribution to the injury, and will satisfy the causation element.⁴⁷ On this view, the ‘but for’ analysis of the epidemiological data is displaced rather than being invalidated. Given a cumulative causal mechanism, it cannot be said that asbestos exposure had no impact on the lung cancer sufferers in the tobacco-only and background-risk partitions. However, the partitions still have ‘but for’ significance. Dr Leigh, having indicated the possibility of a cumulative aetiology, added: ‘It is, however, true that exposure to either factor alone is capable of causing lung cancer.’⁴⁸ The tobacco-only and background-risk partitions indicate the number of sufferers that would have contracted lung cancer even without the asbestos exposure. But on the *Wardlaw* approach, these figures are disregarded. Material contribution is an alternative to ‘but for’ causation.⁴⁹ As the majority in *Ellis (No 2)* noted, where *Wardlaw* applies, ‘satisfaction of the “but for” test of factual causation is not required when a factor makes a material cumulative contribution to the contraction of an indivisible disease.’⁵⁰

Lord Rodger recently observed that the effect of the *Wardlaw* approach is that ‘[t]he “but for” or sine qua non test of causation gives way to this considerably more generous test based on the defendant’s material contribution to the victim’s injury.’⁵¹ But this generosity will often have an unsure foundation.

45 Where the causal mechanism involves an extended latency period, early onset may point more strongly to an earlier exposure. See *Amaca Pty Ltd v Moss* [2007] WASCA 162 (‘*Moss*’), [42], [68].

46 [1956] AC 613 (‘*Wardlaw*’), 621 (Lord Reid). The case may well be decided differently today as pneumoconiosis is recognised as a divisible disease. See *Fairchild* [2003] 1 AC 32, 46–47 (Lord Bingham); Jane Stapleton, ‘Lords A’Leaping Evidentiary Gaps’ (2002) 10 *Torts Law Journal* 276, 283.

47 *Ellis (No 2)* (2008) 37 WAR 1, [308]–[309]. See also *Barker* [2006] 2 AC 572, 602 (Lord Rodger). The exposure in *Moss* [2007] WASCA 162 was *de minimis*.

48 *Ellis (No 1)* [2006] WASC 270, [354]; *Ellis (No 2)* (2008) 37 WAR 1, [208], [335].

49 With perfect information the two approaches could conceivably be reconciled. Having regard to the precise time between the exposures and the onset of the disease, one could, in theory, associate a particular disease with particular exposures. However, knowledge is never sufficiently complete for this to occur. Compare Greenland, above n 13, 1168. In *Ellis* the deceased contracted lung cancer at a relatively young age, and there was some suggestion that this supported an attribution to combined carcinogens. Martin CJ considered that the trial judge had not relied on this style of argument: *Ellis (No 2)* (2008) 37 WAR 1, [338], see also [348]–[349] (Steytler P and McLure JA), contrast [241], [253], [257] (Martin CJ).

50 *Ellis (No 2)* (2008) 37 WAR 1, [309]. See also [315], [338].

51 *Barker* [2006] 2 AC 572, 602. See also *Fairchild* [2003] 1 AC 32, 100 (Lord Rodger).

Epidemiological and medical evidence as to the precise causal mechanism will often be limited and contestable.⁵² Even where causation appears to operate cumulatively rather than from a single dose, this does not mean that every single dose makes a contribution to every single disease. To place such significance on the distinction between the two causal mechanisms appears arbitrary, particularly where the defendant is responsible for such a small exposure, as in *Ellis*, and lung cancer probably would still have ensued without the asbestos exposure.⁵³

5. *Necessary and Sufficient Causes*

The majority in *Ellis (No 2)* claimed that its interpretation and application of *Wardlaw* was ‘conventional’, and had been approved by the High Court on a number of occasions, including *March v E H & M Stramare* and *Chappel v Hart*.⁵⁴ However, the High Court in these decisions did not use the ‘material contribution’ doctrine to override a failure of ‘but for’ causation. Instead, the doctrine was being used to weigh up the force of different causes that have passed the ‘but for’ test. In other words, these decisions concerned ‘scope of liability’, while *Ellis* concerns ‘factual causation’.⁵⁵

In *March* the plaintiff would not have crashed his car but for the defendant having obstructed the road with his truck. But nor would the crash have occurred but for the plaintiff’s alcohol consumption and excessive speed. The court was required to make a comparative assessment of the two ‘but for’ causes. The plaintiff in *Chappel* would not have suffered her throat injury but for the defendant surgeon’s failure to warn her of the risk of this occurring. She would have gone ahead with the surgery, but on another day, with another surgeon, and the low-risk infection would probably not have taken hold.⁵⁶ A further ‘but for’ cause was obviously the infection itself — an unfortunate random occurrence. The material contribution doctrine in such cases is a guide in comparing the force of the

52 In *McGhee* [1973] 1 WLR 1, 4, Lord Reid said: ‘In the present case the evidence does not show – perhaps no one knows – just how dermatitis of this type begins. It suggests to me there are two possible ways. It may be that an accumulation of minor abrasions ... is a necessary precondition ... Or it may be that the disease starts at one particular abrasion and then spreads ...’ With reference to mesothelioma, Lord Bingham noted in *Fairchild* [2003] 1 AC 32, 43: ‘the condition may be caused by a single fibre, or a few fibres, or many fibres: medical opinion holds none of these possibilities to be more probable than any other...’ See also *Fairchild* [2003] 1 AC 32, 71 (Lord Hoffmann), 85–86 (Lord Hutton), 96, 111 (Lord Rodger). In *Hannell* (2007) 34 WAR 109, the majority at 198 interpreted the evidence as establishing cumulative causation while Martin CJ at 155–156 and 159–161 thought it supported the single fibre theory.

53 Compare *Ellis (No 2)* (2008) 37 WAR 1, [216].

54 *Ellis (No 2)* (2008) 37 WAR 1, [308], quoting *Hannell* (2007) 34 WAR 109, 198, citing *March* (1991) 171 CLR 506, 514, 532; *Bennett v Minister of Community Welfare* (1992) 176 CLR 408 (‘*Bennett*’) 419, 428; *Chappel v Hart* (1998) 195 CLR 232 (‘*Chappel*’), 244–245.

55 *Civil Liability Act 2002* (WA) s 5C(1); Jane Stapleton, ‘Legal Cause: Cause-in-Fact and the Scope of Liability for Consequences’ (2001) 54 *Vanderbilt Law Review* 941.

56 David Hamer, ‘Chance would be a Fine Thing: Proof of Causation and Quantum in an Unpredictable World’ (1999) 23 *Melbourne University Law Review* 557, 568–569. See also *Rosenberg v Percival* (2001) 205 CLR 434, 465 (Gummow J); below n 94; *Chester* [2005] 1 AC 134, 142, 144 (Lord Steyn), 154–155, 161 (Lord Hope).

different ‘but for’ causes. The defendant’s breach can be treated as having made a material contribution notwithstanding other ‘but for’ causes ‘having played an even more significant role in producing the loss or damage’.⁵⁷ This is very different from the majority’s use of the material contribution doctrine in *Ellis* (No 2). Asbestos exposure in that case was treated by the majority as having made a material contribution even though it was not a ‘but for’ cause.

Other statements of the High Court present an obstacle for the majority’s claim in *Ellis* (No 2) that ‘but for’ causation need not be established. While the High Court has rejected the ‘but for’ test as an ‘exclusive test’,⁵⁸ this again reflects the test’s over-inclusiveness, and the need to decide whether ‘but for’ causation should necessarily attract legal responsibility.⁵⁹ While not ‘exclusive’, the High Court has recognised that ‘the “but for” test, applied as a negative criterion of causation, has an important role’,⁶⁰ setting the ‘outer limits’,⁶¹ ‘screening out and eliminating from further consideration factors which made no difference to the outcome’.⁶² A negative operation of this kind would rule out causation in *Ellis*.

But there is one well recognised ‘exception’ to the negative operation of the ‘but for’ test: ‘the unusual case where the damage is the result of the simultaneous operation of two or more separate and independent events each of which was sufficient to cause the damage.’⁶³ Consider the two hunter case.⁶⁴ A and B both carelessly fire their rifles in V’s direction, hitting V and killing him. Although both shots are sufficient to kill V, neither is a ‘but for’ cause. A’s shot is not necessary to V’s death because, but for A’s shot, B’s shot would have killed V anyway. By the same reasoning, B’s shot is not a necessary cause either. The philosophical and legal solution for these problem cases is to replace the ‘but for’ necessity requirement with a sufficiency requirement.⁶⁵ A’s shot is sufficient for V’s death because, without B’s shot, V would not have died but for A’s shot. By the same reasoning, B’s shot is also recognised as a sufficient cause. The majority in *Ellis*

57 *Roads and Traffic Authority v Royal* (2008) 245 ALR 653 (‘Royal’), 675 (Kirby J), quoting from *Henville v Walker* (2001) 206 CLR 459, 493 (McHugh J), citing *Wardlaw* [1956] AC 613, 620 (Lord Reid). It appears that the two causal contenders in *Henville v Walker* were both necessary causes: 494–495 (McHugh J), 508 (Hayne J).

58 *Royal* (2008) 245 ALR 653, 674 (Kirby J), 687 (Kiefel J); *Chappel* (1998) 195 CLR 232, 243 (McHugh J), 255 (Gummow J), 269 (Kirby J) 281–282 (Hayne J); *March* (1991) 171 CLR 506, 508 (Mason CJ), 522–523 (Deane J), 524 (Toohey J), compare 534 (McHugh J).

59 *Royal* (2008) 245 ALR 653, 687 (Kiefel J).

60 *March* (1991) 171 CLR 506, 515 (Mason CJ). Compare *Bennett* (1992) 176 CLR 408, 413 (Mason CJ, Deane and Toohey JJ).

61 *Royal* (2008) 245 ALR 653, 674 (Kirby J).

62 John Fleming, *The Law of Torts* (9th ed, 1998) 220.

63 *March* (1991) 171 CLR 506, 534 (McHugh J). Compare 516 (Mason CJ), 523 (Deane J).

64 Tony Honoré, *Responsibility and Fault* (1999) 107; Richard W Wright, ‘Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts’ (1988) 73 *Iowa Law Review* 1001, 1026; J L Mackie, *The Cement of the Universe: A Study of Causation* (1980) 44; Jane Stapleton, ‘Choosing what we mean by “Causation” in the Law’ (2008) 73 *Missouri Law Review* 433, 442.

65 Many theorists formalise this as the NESS test. The breach must be a Necessary Element of a Sufficient Set: Honoré, above n 64, 94–120; Hart and Honoré, above n 6, 51–61; Wright, above n 64, 1018–1039; Stapleton, ‘Choosing What We Mean by “Causation” in the Law’, above n 64, 444.

(No 2) invoke this exception, suggesting that, while the asbestos exposure may not have been necessary it was sufficient to cause the lung cancer. They criticise Martin CJ's reliance on the 'but for' test as 'inconsistent with the conventional principle of common law causation that a breach can materially contribute to an outcome notwithstanding that there are other sufficient causes of that outcome.'⁶⁶

But it is questionable whether a case like *Ellis* should be treated as exceptional in this sense. In the recent House of Lords asbestos exposure case, *Barker v Corus*, Lord Hoffmann rejected this style of analysis. He said that the present case was 'of course ... *not* a case of "concurrent joint tortfeasors, where the actions of either would be sufficient by themselves to produce the consequence".'⁶⁷ A low level of asbestos exposure *may* be sufficient to cause lung cancer, but this is not enough. Where necessity cannot be demonstrated, 'strong sufficiency' is required. The condition must be such that 'the consequence invariably follows'.⁶⁸ This is satisfied in the two hunter case. Each shot taken individually is strongly sufficient for the victim's death. Given a shot of that kind, death would invariably follow. But the asbestos exposure in *Ellis* is not strongly sufficient. It merely adds a little to a relatively low risk of lung cancer. At most incidence would increase to a dozen or so per 100,000.⁶⁹ Lung cancer is a rare, not invariable, consequence of this amount of asbestos exposure. Whereas, in the two hunter case, the victim's death is over-determined, the lung cancer in a case like *Ellis* is indeterminate. 'Indeterminacy presents a difficulty' for the sufficiency test.⁷⁰

The problem of indeterminacy is not always insurmountable. Lord Hoffmann in *Gregg* recently suggested that 'the law regards the world as in principle bound by laws of causality. Everything has a determinate cause, even if we do not know what it is.'⁷¹ While, based on our limited knowledge, the risk of lung cancer appeared low, there must have been some factor operating that, in combination with the tobacco and/or asbestos exposures, fully determined the onset of lung cancer. We do not know what this factor was, whether infection, genetic predisposition or something else, but it is not unusual for there to be difficulty identifying every single contributing factor.⁷² It appears possible to resolve the indeterminacy by assuming the existence of this additional factor.

To a point, this strategy appears legitimate and may even be necessary.⁷³ Such an assumption forms part of the statistical 'but for' reasoning outlined above. Had the asbestos exposure not occurred there is a high probability that the lung cancer

66 *Ellis (No 2)* (2008) 37 WAR 1, [338], see also [309]; *Hannell* (2007) 34 WAR 109, 199 (Steytler P and McLure JA); *Krakouer v Western Australia* (2006) 161 A Crim R 347, 367 (McLure JA).

67 *Barker* [2006] 2 AC 572, 588 (emphasis in original) rejecting this view of the trial judge.

68 Honoré, above n 64, 96.

69 Thun et al, above n 12.

70 Honoré, above n 64, 115.

71 *Gregg* [2005] 2 AC 176, 196. Although a majority of the High Court have recognised that 'questions as to the future or hypothetical effect of physical injury or degeneration are not commonly susceptible of scientific demonstration or proof': *Malec v J C Hutton Pty Ltd* (1990) 169 CLR 638, 643. See also *Mallett v McMonagle* [1970] AC 166, 176 (Lord Diplock); Hamer, above n 56, 562–566.

72 Honoré, above n 64, 99.

would still have appeared. In constructing the counterfactual — what would otherwise have occurred — attributable risk figures are used — 99.74% (Rogers) or 77% (de Klerk) (Tables One and Two) — rather than the very low incidence figures. The attributable risks add up to 100%. Given that the lung cancer occurred, for the purpose of identifying the cause, the possibility of it not occurring is disregarded. But it is difficult to see how this approach could be stretched to the point that asbestos exposure, the lowest risk factor, is identified as sufficient or necessary for the onset of lung cancer. Such a construction may be theoretically possible, but it would involve extreme manipulation, and be conceptually suspect.⁷⁴ It does not provide a plausible basis for a finding of causation in *Ellis*.

6. Presuming Causation from Breach

In *Ellis (No 2)* the majority upheld the trial judge's finding of causation primarily on the basis that the asbestos exposure operated cumulatively and made a material contribution to the lung cancer. Further, the asbestos was said to be a sufficient cause of the lung cancer. These findings have been criticised above.

In another recent asbestos exposure case, *Amaca v Hannell*, the Western Australian Court of Appeal split the same way on causation.⁷⁵ In this case, as well as relying on the arguments considered above, the majority also invoked a proposition of Gaudron J in *Bennett v Minister of Community Welfare*:

[G]enerally speaking, if an injury occurs within an area of foreseeable risk, then, in the absence of evidence that the breach had no effect, or that the injury would have occurred even if the duty had been performed, it will be taken that the breach of the common law duty caused or materially contributed to the injury.⁷⁶

In *Hannell* the majority held that the plaintiff's mesothelioma was within the area of risk created by the breach, causation was presumed,⁷⁷ and there was insufficient

73 This resolution of the indeterminacy was not adopted in the 'but for' reasoning in *Chappel* (1998) 195 CLR 232. Contrast *Chester* [2005] 1 AC 134, 141 (Lord Bingham, dissenting), 147 (Lord Hoffmann, dissenting), 166 (Lord Walker). Special policy considerations may apply to medical failure to warn cases: Tony Honoré, 'Medical Non-Disclosure, Causation and Risk: *Chappel v Hart*' (1999) 7 *Torts Law Journal* 1, 8; *Chester* [2005] 1 AC 134, 145 (Lord Steyn), 160–161 (Lord Hope).

74 Mark Kelman, 'The Necessary Myth of Objective Causation Judgments in Liberal Political Theory' (1987) 63 *Chicago-Kent Law Review* 579, 604. In addition to the issue of indeterminacy, there are a number of other obstacles with the application of the NESS test to *Ellis*. To reduce notionally the amount of tobacco exposure in order to create more space for the asbestos exposure to operate creates the 'subset problem': 603. Theorists disagree as to whether it is legitimate to 'disaggregate' in this way. See Stapleton, 'Choosing What We Mean by "Causation" in the Law', above n 64, 476. Further, issues may arise due to differences in the timing and mechanisms involved in the causal operation of tobacco and asbestos: Wright, above n 64, 1022–1025; Honoré, above n 64, 111–115. In the two hunter case the shots operated identically and simultaneously.

75 *Hannell* (2007) 34 WAR 109. The court was unanimous upholding the defendant's appeal on other grounds.

76 *Hannell* (2007) 34 WAR 109, 194, quoting *Bennett* (1992) 176 CLR 408, 420–421.

evidence to rebut the presumption. The trial judge adopted this reasoning in *Ellis (No 1)*,⁷⁸ but it received no comment by the majority.⁷⁹

Suggestions similar to that of Gaudron J in *Bennett* have been made in other High Court decisions. An early version is Dixon J's statement in *Betts v Whittingslowe*:

breach of duty coupled with an accident of the kind that might thereby be caused is enough to justify an inference, in the absence of any sufficient reason to the contrary, that in fact the accident did occur owing to the act or omission amounting to the breach of statutory duty.⁸⁰

Such statements have been applied widely, to road accident cases as well as exposure cases like *Hannell* and *Ellis*. But questions have been raised regarding the authority, meaning, and logic of such statements. In the recent road accident appeal, *RTA v Royal*, Kiefel J noted that the majority in *Bennett* reserved its opinion on the point.⁸¹ She held that the onus remained on the plaintiff and that it had not been discharged.⁸² Kirby J agreed that the onus remained on the plaintiff, but held that a finding of 'breach ... may open the way for (while not compelling) an inference of causation-in-fact.'⁸³ Dissenting, he upheld the inference of causation in that case. The majority applied the presumption, but swiftly and without enthusiasm, and held that there was sufficient evidence to rebut it.⁸⁴

The majority in *Hannell* drew on Gaudron J's judgment in *Bennett* to explain the reasoning behind the presumption:

[T]here is usually no reason to separate or distinguish the question of breach of a common law duty from that of causation because the duty relates to precautions a reasonable person in the position of the person sued would have taken to prevent a foreseeable risk of harm of the kind suffered and a precaution would not be classified as reasonable unless its performance would, in the ordinary course of events, avert the risk that called it into existence.⁸⁵

However, this overstates the connection between breach and causation. 'Sometimes the common law requires a person to take precautions which would merely reduce, rather than eliminate, a particular risk.'⁸⁶ The fact that a breach is

77 Here I use the term 'presumption' loosely. Below I note the view that the 'presumption' is no more than a permissible inference, and the alternative view that it shifts the evidential burden, but not the legal burden: see n 84 and n 98.

78 *Ellis (No 1)* [2006] WASC 270, [673], [674].

79 Compare *Ellis (No 2)* (2008) 37 WAR 1, [223]–[224] (Martin CJ).

80 (1945) 71 CLR 637 ('*Betts*'), 649. See also *Chappel* (1998) 195 CLR 232, 273 (Kirby J); below n 95.

81 *Royal* (2008) 245 ALR 653, 689, citing *Bennett* (1992) 176 CLR 408, 416.

82 *Royal* (2008) 245 ALR 653, 689. See also *Stamoulis* [2009] NSWCA 153, [127]; *Gett v Tabet* (2009) 254 ALR 504 ('*Gett*'), 556–557; *Flounders v Millar* [2007] NSWCA 238 ('*Flounders*'), [3]–[37]; *Civil Liability Act 2002* (WA) s 5D.

83 *Royal* (2008) 245 ALR 653, 676.

84 *Royal* (2008) 245 ALR 653, 662.

85 *Hannell* (2007) 34 WAR 109, 194, citing *Bennett* (1992) 176 CLR 408, 422.

followed by injury will not necessarily imply causation, particularly in a case like *Ellis* where other factors contributed greater risks. A causation finding on this basis may be little more than speculation and conjecture.⁸⁷

The scope of the presumption can be tightened to reflect this limitation. According to Gaudron J's statement, the presumption operates only when 'an injury occurs within an area of foreseeable risk'.⁸⁸ In *Betts* Dixon J indicated that the injury must be 'of the kind that might thereby be caused'.⁸⁹ In *North Sydney Council v Binks*,⁹⁰ Basten JA, dissenting, held that the car accident was not of a kind that the defendant council's misleading road signs may have caused because that was 'only one of a number of plausible explanations' for the accident.⁹¹ Others were the plaintiff's high blood alcohol level, excessive speed and inadequate attention. Basten JA was critical of the majority's looser reasoning: 'If the "kind" of accident is defined at a sufficient level of generality, the requirement of a causal connection will, in a practical sense, become irrelevant.'⁹²

This differentiation and narrowing strategy may not be available in exposure cases where the various possible causes are of the same kind. Mesothelioma, for example, is not known to have any cause other than the inhalation of asbestos fibres. The question will be which asbestos exposure was the cause, with each exposure creating the same kind of risk of the same kind of injury. In *Ellis* there were different types of exposure. It could, perhaps, be said that the injury had the appearance of smoking-induced lung cancer, rather than asbestos-induced lung cancer. The injury was not 'of the kind that might thereby be caused' by the breach, and causation is therefore not presumed. However, this reasoning appears rather strained and artificial.

On one view, Spigelman CJ takes the differentiation and narrowing strategy to an even greater extreme in *Seltsam v McGuinness*.⁹³ The target is McHugh J's proposition in *Chappel v Hart*:

86 Andrew Palmer, 'Causation in the High Court' (1993) 1 *Torts Law Journal* 9, 21.

87 *Hannell* (2007) 34 WAR 109, 122, 124 (Martin CJ); *Flounders* [2007] NSWCA 238, [33] (Ipp JA); *North Sydney Council v Binks* (2007) 163 LGERA 94 ('*Binks*'), 116–117 (Basten JA); *Seltsam* (2000) 49 NSWLR 262, 278–280 (Spigelman CJ); *Bendix Mintex Pty Ltd v Barnes* (1997) 42 NSWLR 307, 316 (Mason P); *Jones v Dunkel* (1959) 101 CLR 298, 304–305 (Dixon CJ); *Luxton v Vines* (1952) 85 CLR 352, 359. However, Hoeben J arguably misapplies these propositions in *Flounders* [2007] NSWCA 238 at [113], stating 'it matters not that there were two reasonably available scenarios in which negligence could be found if it be the case that there remained an equally reasonable scenario which was inconsistent with negligence.' This reasoning suggests that the probability of negligence is 2/3, considerably more probable than not. However, a majority of the High Court has twice rejected Murphy J's reasoning along these lines: *TNT Management Pty Ltd v Brooks* (1979) 23 ALR 345; *West v Government Insurance Office of NSW* (1981) 148 CLR 62.

88 *Bennett* (1992) 176 CLR 408, 420.

89 *Betts* (1945) 71 CLR 637, 649.

90 (2007) 163 LGERA 94.

91 (2007) 163 LGERA 94, 116.

92 *Binks* (2007) 163 LGERA 94.

93 *Seltsam* (2000) 49 NSWLR 262.

If a wrongful act or omission results in an increased risk of injury to the plaintiff and that risk eventuates, the defendant's conduct has materially contributed to the injury that the plaintiff suffers whether or not other factors also contributed to that injury occurring.⁹⁴

According to Spigelman CJ, McHugh J meant 'eventuated', 'not in the sense that X happened and Y had also happened, but that it was undisputed that Y had happened *because* of X.'⁹⁵ It may be objected that Spigelman CJ's interpretation is implausible. It makes a nonsense of McHugh J's statement of the presumption; causation must be proved before it is presumed. And yet, Spigelman CJ's interpretation appears correct. McHugh J was not referring to a presumption that could assist the plaintiff in establishing 'but for' causation. The issue was whether legal responsibility should be attached to a proven 'but for' cause: scope of liability rather than factual causation. As discussed in the previous section, in *Chappel v Hart* the defendant surgeon's failure to warn of the risks of surgery was a 'but for' cause of the throat injury. However, on McHugh J's view, breach did not increase the risk of the injury, and should therefore not be considered to have materially contributed to it.⁹⁶ But while Spigelman CJ's analysis may deprive the causation presumption of McHugh J's support, the propositions of Gaudron J in *Bennett* and Dixon J in *Betts* remain.

The reasoning that argues against the creation of the causation presumption works to rebut the presumption where it is employed. At most, the effect of the presumption should only be to shift the evidential burden to the defendant.⁹⁷ To rebut it, the defendant need only adduce some evidence against causation. The plaintiff will then bear the legal burden and be required to prove causation on the balance of probabilities. In *Royal*, the majority, applying Dixon J's proposition from *Betts*, held '[t]here was ample material in the behaviour of the drivers to create a "sufficient reason to the contrary"'.⁹⁸ The majority in *Binks* were far too demanding in requiring the defendant Council to 'establish' its 'various theories or hypotheses said to exonerate the Council ... [and] to wholly supersede the Council's negligence'.⁹⁹

In exposure cases, in combination with a generous 'material contribution' doctrine, the presumption could provide the plaintiff considerable assistance where the nature of the causal mechanism remains unclear.¹⁰⁰ However, if the case

94 *Chappel* (1998) 195 CLR 232, 244. This was quoted with approval in *Naxakis v Western General Hospital* (1999) 197 CLR 269, 279 (Gaudron J), 312 (Callinan J).

95 *Seltsam* (2000) 49 NSWLR 262, 280 (emphasis in original), cited in *Hannell* (2007) 34 WAR 109, 123–124 (Martin CJ); *Royal* (2008) 245 ALR 653, 689 (Kiefel J), quoted in *Flounders* [2007] NSWCA 238, [20] (Ipp JA). See also *Fairchild* [2003] 1 AC 32, 118 (Lord Rodger).

96 *Chappel* (1998) 195 CLR 232, 250 (McHugh J, dissenting). See also 281, 286 (Hayne J, dissenting). Contrast 241 (Gaudron J), 277 (Kirby J), 281. Gummow J did not express a view.

97 *Chappel* (1998) 195 CLR 232, 247 (McHugh J); *Binks* (2007) 163 LGERA 94, 103–104 (Santow JA); *Hannell* (2007) 34 WAR 109, 126–127 (Martin CJ), 194–195 (Steytler P and McLure JA).

98 *Royal* (2008) 245 ALR 653, 662.

99 *Binks* (2007) 163 LGERA 94, 104.

100 See above n 52.

turns on a 'but for' interpretation of the epidemiological evidence, the presumption may have little effect. Often the evidence will indicate unambiguously whether or not the 'but for' test is satisfied. In *Hannell* the majority provide no basis for their suggestion that 'a statistical conclusion could not be a proper basis for preventing (or rebutting) a prima facie case under the *Bennett* test. ... [The presumption] only assists the plaintiff to jump the evidential gap.'¹⁰¹ More sound is Martin CJ's conclusion that epidemiological evidence attributing a relatively low relative risk to the defendant's breach would be 'sufficient to displace any prima facie case'.¹⁰² In *Ellis*, even if the presumption did operate, there would be a strong argument that it was rebutted.

7. *Liability for Exposure to Risk*

In this article I have sought to clarify the evidence and legal principles relating to the proof of factual causation¹⁰³ in *Ellis*. It might be argued, however, that, regardless of the facts, causation should be considered to be proven as a matter of policy. Should Australia follow the House of Lords in the asbestos exposure case, *Fairchild v Glenhaven*,¹⁰⁴ and abolish the requirement of causation, allowing the plaintiff to recover simply on the basis of having been exposed to risk?¹⁰⁵ What are the implications of this approach for *Ellis*?

Needless to say the *Fairchild* principle is a radical move which should not be taken lightly. In *Fairchild* the court sought to limit narrowly the cases covered by its new approach.¹⁰⁶ Lord Hoffmann subsequently warned that it is 'only natural that, the dyke having been breached, the pressure of a sea of claimants should try to enlarge the gap.'¹⁰⁷ And Lord Bingham acknowledged that 'it would be unrealistic to suppose that the principle ... will not over time be the subject of incremental and analogical development'.¹⁰⁸

A further development took place in *Barker*. The House of Lords held that recovery under *Fairchild* should not be 'all or nothing', but in proportion to the risk exposure.¹⁰⁹ This is a logical development,¹¹⁰ but raises further policy issues and

101 *Hannell* (2007) 34 WAR 109, 199.

102 *Hannell* (2007) 34 WAR 109, 137.

103 Glanville Williams, 'Causation in the Law' (1961) *Cambridge Law Journal* 62, 62–63; Stapleton, 'Legal Cause: Cause-in-Fact and the Scope of Liability for Consequences', above n 55.

104 [2003] 1 AC 32, interpreting and applying *McGhee* [1973] 1 WLR 1.

105 Although Lord Hoffmann points out that risk is a causative concept and denies that the causation element is being abandoned altogether: *Fairchild* [2003] 1 AC 32, 74.

106 *Fairchild* [2003] 1 AC 32, 40, 68 (Lord Bingham), 111–112, 118–119 (Lord Rodger) 74, 77 (Lord Hoffmann). Compare *Fairchild* [2003] 1 AC 32, 70 (Lord Nicholls), 91 (Lord Hutton); *Barker* [2006] 2 AC 572, 581–583 (Lord Hoffmann), 597–600 (Lord Scott), 611, 614 (Lord Walker).

107 *Barker* [2006] 2 AC 572, 581.

108 *Fairchild* [2003] 1 AC 32, 68 (Lord Bingham), quoted in *Barker* [2006] 2 AC 572, 596 (Lord Scott).

109 *Barker* [2006] 2 AC 572, 589–590, 594 (Lord Hoffmann), 599 (Lord Scott), 613 (Lord Walker).

110 Contrast *Barker* [2006] 2 AC 572, 603–608 (Lord Rodger, dissenting).

questions as to how the *Fairchild* principle sits alongside the orthodox principles of proof and causation. Can the plaintiff recover for risk exposure without physical injury?¹¹¹ Is the defendant entitled to a discount where causation is proven on the balance of probabilities but less than certain?¹¹² Should there be proportional damages in medical negligence cases where the plaintiff has been deprived of the loss of chance of a better prognosis?¹¹³ Here too the plaintiff often faces an irreducible evidential gap resulting from the limits of medical knowledge. Reflecting the complexity of the issues of policy and principle, the apportionment principle of *Barker* has since been reversed by legislation.¹¹⁴

At some stage it may well be worth considering whether Australian law should follow the developments in *Fairchild* and *Barker*.¹¹⁵ The traditional ‘all or nothing’ approach to causation may bring rough justice to either party in exposure and medical negligence cases where there are fundamental obstacles to the proof of causation. Proportional risk-based compensation may be the fairest way to distribute the risk of error. But these are radical developments, posing many complex issues, and *Ellis* does not provide a suitable vehicle for their consideration.

Even if the *Fairchild* principle were adopted, it is doubtful whether it would apply to *Ellis*. In *Fairchild* it appeared certain that the plaintiff’s mesothelioma had been caused by tortious workplace exposure to asbestos, but the plaintiff could not prove which employer was responsible. In *Barker* the principle was extended slightly to the situation where some of the workplace exposure was during a period of self-employment. But *Ellis* is a very different case again. The workplace exposure to asbestos was accompanied by the deceased’s own tobacco consumption. This raises two key points of distinction from *Fairchild* and *Barker* apparently putting it beyond the scope of the principle.¹¹⁶ First, there is the fact that one of the exposures is unrelated to the workplace. Second, there is the fact that the two exposures are of different types. The *Fairchild* principle is limited to situations where the various exposures work by substantially the same causal mechanism.¹¹⁷ In *Barker*, Lord Hoffmann expressly stated, ‘I do not think that the exception applies when the claimant suffers lung cancer which may have been caused by exposure to asbestos or some other carcinogenic matter but may also

111 *Gett* (2009) 254 ALR 504, 583–585.

112 *Gett* (2009) 254 ALR 504, 583–585.

113 Such loss of chance claims have recently been rejected in *Gregg* [2005] 2 AC 176, and *Gett* (2009) 254 ALR 504. See also *Rothwell v Chemical & Insulating Co Ltd* [2008] 1 AC 281.

114 *Compensation Act 2006* (UK) s 3.

115 Note that s 5C(2) *Civil Liability Act 2002* (WA) does allow, ‘in an appropriate case’, for a breach, not proven to be necessary for harm, to nevertheless be treated as the factual cause of harm. This is also allowed by s 5D(2) *Civil Liability Act 2002* (NSW) although only ‘in an exceptional case’.

116 See above, n 107.

117 This distinguishes *Wilsher v Essex Area Health Authority* [1987] QB 730, 779 (Browne–Wilkinson VC, dissenting); *Wilsher v Essex Area Health Authority* [1988] AC 1074, 1090 (Lord Bridge); *Fairchild* [2003] 1 AC 32, 95 (Lord Hutton), 118–119 (Lord Rodger), contrast 77 (Lord Hoffmann). See also *Barker* [2006] 2 AC 572, 587 (Lord Hoffmann), 599–600 (Lord Scott).

have been caused by smoking and it cannot be proved which is more likely to have been the causative agent.¹¹⁸

These restrictions on the *Fairchild* principle may be viewed as purely pragmatic and open to criticism. Indeed, before adopting it in *Barker*, Lord Hoffmann in *Fairchild* suggested that the causal mechanism restriction was unprincipled.¹¹⁹ But there is a more substantial objection to the application of *Fairchild* in the *Ellis* case. And it is a simple one. In *Ellis* there is a high likelihood that the cancer was caused by the deceased's tobacco consumption, not asbestos exposure. The injustice that the plaintiff faced in *Fairchild* is not present in *Ellis*, and furthermore it is questionable whether there could even be said to be an evidential gap in *Ellis*.

8. Conclusion

Ellis raises the issue whether Paul Steven Cotton's lung cancer was caused by his exposure to asbestos for which the defendants were responsible. Crucial to the resolution of this question is a proper understanding and assessment of the epidemiological evidence, having regard to the legal principles governing causation and proof. The understanding of the evidence shown by the majority and the trial judge is flawed, and their finding of causation is open to criticism. Despite some variation between experts, the epidemiological evidence attributes virtually the entire risk of lung cancer to tobacco and background exposure rather than the workplace asbestos exposure. This strongly suggests that the lung cancer would have ensued even had the asbestos exposure not occurred. As Martin CJ recognised, there is an absence of 'but for' causation.

Contrary to the claims of the trial judge and the majority, the epidemiological models do take some account of the synergistic interaction of the tobacco and asbestos exposures. However, the medical evidence does leave open the possibility that the asbestos exposure contributed to a greater number of lung cancers than the attributable risk figures imply. According to the *Wardlaw* material contribution doctrine, where the breach makes this kind of cumulative contribution, this is sufficient to satisfy the causation element, notwithstanding the absence of 'but for' causation. There is no clear High Court authority determining whether *Wardlaw* should be given such a generous operation. This may be one of the major questions for the High Court to settle on the appeal. Despite the clear acceptance of *Wardlaw* by the House of Lords, the nature of the causal mechanism is frequently a matter of speculation, and the doctrine can operate arbitrarily. In this context, the doctrine may be given a great deal of work, but with very poor materials.

There are further possible bases for a causation finding in *Ellis*, but they also appear weak. There is the majority's suggestion that, though not a necessary cause of lung cancer, the asbestos exposure was a sufficient cause. But the asbestos exposure in *Ellis* was a minor risk factor with insufficient causal strength for this

118 *Barker* [2006] 2 AC 572, 587.

119 *Fairchild* [2003] 1 AC 32, 77.

notion of causation to apply. The trial judge in *Ellis* followed the majority in *Hannell* in applying the *Betts/Bennett* presumption of causation from breach. But the status of this presumption is uncertain. And even if causation is presumed, the epidemiological evidence in *Ellis* provides abundant material for rebuttal.

Finally, there are the recent radical developments to the principles governing causation and proof in the House of Lords. Where *Fairchild* and *Barker* apply, causation is no longer an 'all or nothing' affair. The plaintiff can simply show exposure to a particular level of risk and recover proportional damages. These principles offer significant policy benefits in cases where evidential gaps flowing from the limitations of medical knowledge can lead to a failure of justice. But such principles are also highly unorthodox, and raise significant questions as to their scope and conceptual foundations. *Ellis* is not a suitable case for these issues to be addressed. The evidence in *Ellis* points quite clearly to an absence of causation. There is no significant evidential gap, and no serious threat of injustice. It may have been appropriate for their Lordships in *Fairchild* and *Barker* to have leapt the evidential gap. But the High Court Justices in *Ellis* should look before they leap.

