Allianz Australia Ltd v Sim - [2012] NSWCA 68

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Court of Appeal
New South Wales

Case Title: Allianz Australia Ltd v Sim; WorkCover Authority (NSW) v Sim; Wallaby Grip (BAE) Pty Ltd (In liq) v Sim

Medium Neutral Citation: [2012] NSWCA 68

Hearing Date(s): 15, 16 November 2011

Decision Date: 04 April 2012

Jurisdiction:

Before: Allsop P at 1;
Basten JA at 54;
Meagher JA at 182

Decision:

In each of the three matters:

(1) Appeal dismissed.

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(2) Appellant to pay Mrs Sim's costs.

[Note: The Uniform Civil Procedure Rules 2005 provide (Rule 36.11) that unless the Court otherwise orders, a judgment or order is taken to be entered when it is recorded in the Court146s computerised court record system. Setting aside and variation of judgments or orders is dealt with by Rules 36.15, 36.16, 36.17 and 36.18. Parties should in particular note the time limit of fourteen days in Rule 36.16.]

Catchwords:

COSTS - indemnity costs - offer of compromise - whether Dust Diseases Tribunal Regulation 2007 (NSW), Pt 6 , only operates to the extent that it is not inconsistent with the Uniform Civil Procedure Rules - whether offeror had provided all documents necessary to enable the offeree to consider offer under the Uniform Civil Procedure Rules - whether offer was open for a reasonable time

EVIDENCE - admissibility - expert evidence - whether appropriate to consider all of the evidence to determine whether expert evidence should have been admitted - whether pathologist able to give evidence as to the causal connection between exposure to asbestos dust and lung cancer, given current state of medical knowledge

TORTS - causation - whether increase in risk can be equated with factual causation - whether liability requires satisfaction of the necessary condition test - whether open to Tribunal to accept pathologist's opinion as to link between exposure to asbestos dust and lung cancer

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Legislation Cited:

Civil Liability Act 2002 (NSW), ss 3B, 5D
Civil Procedure Act 2005 (NSW), ss 10, 11
Dust Diseases Tribunal Act 1989 (NSW), s 32
Dust Diseases Tribunal Regulation 2007 (NSW) , cll 84, 87; Pt 6
Evidence Act 1995 (NSW), ss 55, 76, 79
Law Reform (Miscellaneous Provisions) Act 1946 (NSW), s 6
Supreme Court Act 1970 (NSW), s 75A
Uniform Civil Procedure Rules 2005 (NSW), rr 1.5, 1.7, 20.26, 36.16, 51.53 ; Schs 1, 2
Workers Compensation Act 1987 (NSW), ss 151, 151AB, 236

Cases Cited:

Adelaide Stevedoring Co Ltd v Forst [1940] HCA 45; 64 CLR 538
Amaba Pty Ltd (Under NSW Administered Winding Up) v Booth; Amaca Pty Ltd (Under NSW Administered Winding Up) v Booth [2010] NSWCA 344; (2011) Aust Torts Rep ¶82-079
Amaca Pty Ltd (under NSW administered winding up) v Booth [2011] HCA 53; 86 ALJR 172
Amaca Pty Ltd v Ellis [2010] HCA 5; 240 CLR 111
Amaca Pty Ltd v King [2011] VSCA 447
Anderson v Minneapolis, St P & S Ste M Ry, 146 Minn 430; 179 NW 45 (1920)
Barker v Corus UK Ltd [2006] UKHL 20; [2006] 2 AC 572
Bendix Mintex Pty Ltd v Barnes (1997) 42 NSWLR 307
Bonnington Castings Ltd v Wardlaw [1956] AC 613
Chappel v Hart [1998] HCA 55; 195 CLR 232
Clark v Ryan [1960] HCA 42; 103 CLR 486
Corey v Havener, 182 Mass 250; 65 NE 69 (1902)
Dasreef Pty Ltd v Hawchar [2011] HCA 21; 243 CLR 588
EM Baldwin & Son Pty Ltd v Plane (1998) 17 NSWCCR 434
Evans v Queanbeyan City Council [2011] NSWCA 230
Fairchild v Glenhaven Funeral Services Ltd [2002] UKHL 22; [2003] 1 AC 32
Fairchild, Barker v Corus UK Ltd [2006] UKHL 20; [2006] 2 AC 572
Gett v Tabet [2009] NSWCA 76; 254 ALR 504
HG v The Queen [1999] HCA 2; 197 CLR 414
Jones v Dunkel [1959] HCA 8; 101 CLR 298
Makita (Australia) Pty Ltd v Sprowles [2001] NSWCA 305; 52 NSWLR 705

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March v E & MH Stramare Pty Ltd [1991] HCA 12; 171 CLR 506
Merck Sharp & Dohme (Australia) Pty Ltd v Peterson [2011] FCAFC 128; 196 FCR 145
Michie v Great Lakes Steel Div, 495 F2 d 213 (6th Cir), cert denied, 419 US 997 (1974)
Norton Australia Pty Ltd v Streets Ice Cream Pty Ltd [1968] HCA 61; 120 CLR 635
Resurfice Corp v Hanke 2007 SCC 7; [2007] 1 SCR 333
Seltsam Pty Ltd v McGuiness [2000] NSWCA 29; 49 NSWLR 262
Sienkiewicz (Administratrix of the Estate of Enid Costello Deceased) v Greif (UK) Ltd [2011] UKSC 10; [2011] 2 AC 229
Strong v Woolworths Ltd [2012] HCA 5
Tabet v Gett [2010] HCA 12; 240 CLR 537

Texts Cited:

American Law Institute (ALI), Restatement (Third) of Torts: Liability for Physical and Emotional Harm, (2009) Vol 1 380-1 (s27, Illustration 3)
Michael S Moore, Causation and Responsibility (OUP, 2010), p 84
Professor Jane Stapleton in "Factual Causation" (2010) Fed L Rev 467 at 475-476
Professor Jane Stapleton, "Two Causal Fictions at the Heart of US Asbestos Doctrine" (2006) 122 Law Quarterly Review 189
Professor Jane Stapleton, "Factual Causation and Asbestos Cancers", (2010) 126 LQR 351 at 355, 356
Professor Richard W Wright "Causation in Tort Law" (1985) 73 Cal L Rev 1735, pp 1791-1792

Category:

Principal judgment

Parties:

2011/16442:
Allianz Australia Ltd - Appellant
Lorraine Fay Sim (as legal personal representative of the Estate of the Late David Sim) - First Respondent
WorkCover Authority (NSW) - Second Respondent
Wallaby Grip (BAE) Pty Ltd (In liq) - Third Respondent

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2011/21072-002:
WorkCover Authority (NSW) - Appellant
Lorraine Fay Sim (as legal personal representative of the Estate of the Late David Sim) - First Respondent
Allianz Australia Ltd - Second Respondent
Wallaby Grip (BAE) Pty Ltd (In liq) - Third Respondent

2011/21072-004:
Wallaby Grip (BAE) Pty Ltd (In liq) - Appellant
Lorraine Fay Sim (as legal personal representative of the Estate of the Late David Sim) - First Respondent
Allianz Australia Ltd - Second Respondent
WorkCover Authority (NSW) - Third Respondent

Representation

- Counsel:

Counsel:

T G R Parker SC with D T Miller SC - Allianz Australia Ltd
G Little SC with J L Sharpe - WorkCover Authority (NSW)
B W Walker SC with D T Miller SC - Wallaby Grip (BAE) Pty Ltd (In liq)
P C B Semmler QC with S Tzouganatos - Lorraine Fay Sim (as legal personal representative of the Estate of the Late David Sim)

- Solicitors:

Solicitors:

Ellison Tillyard Callanan - Allianz Australia Ltd
Thompson Cooper Lawyers - WorkCover Authority (NSW)
Hicksons Lawyers - Wallaby Grip (BAE) Pty Ltd (In liq)

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	Turner Freeman Lawyers - Lorraine Fay Sim (as legal personal representative of the Estate of the Late David Sim)
File number(s):	2011/16442; 2011/21072
Decision Under Appeal	
- Court / Tribunal:	Dust Diseases Tribunal
- Before:	Curtis J
- Date of Decision:	24 December 2010
- Citation:	Lorraine Fay Sim v Allianz Australia Ltd [2010] NSWDDT 19
- Court File Number(s)	DDT 158 of 2009
Publication Restriction:	

HEADNOTE

[This headnote is not to be read as part of the judgment]

Mr Sim worked with asbestos for many years and in the employ of various employers. He contracted asbestosis and subsequently lung cancer. He died on 6 July 2009. Mrs Sim ("the plaintiff"), the legal personal representative of his estate, commenced proceedings in the Dust Diseases Tribunal ("the Tribunal") against four of these employers or their insurers. The WorkCover Authority of NSW ("WorkCover") incurred liability on behalf of the defunct insurer of the second and third employers.

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The Tribunal admitted expert evidence from Professor Henderson, Associate Professor Bryant and Dr Yates. The evidence asserted that it could not be established that the tortious exposure during employment with each employer was a necessary causal element in the chain of events leading to the lung cancer. The Tribunal accepted this evidence. Nevertheless, the Tribunal held that the employers' negligence had caused Mr Sim's lung cancer.

The Tribunal:

(a) found that the appellants were jointly and severally liable in respect of Mr Sim's lung cancer and awarded damages of $317,561.85,

(b) found that WorkCover was liable in respect of the second employer pursuant to the *Workers Compensation Act 1987* (NSW), s 151AB , and

(c) ordered that WorkCover pay the plaintiff's costs on an indemnity basis from 14 May 2010, following an offer of compromise which was not accepted by WorkCover.

The issues for determination on appeal were:

(i) whether the Tribunal erred in admitting the expert evidence because the opinions of the experts were not "wholly or substantially based" on their "training, study or experience",

(ii) whether each of the four employers was implicated or whether all could escape because it could not be established that the tortious exposure during employment with each was a necessary causal element in the chain of events,

(iii) whether the Tribunal erred in drawing an adverse inference from the appellants' failure to call expert medical evidence on the question of causation,

(iv) whether the Tribunal erred in finding that WorkCover was liable in respect of the second employer pursuant to the *Workers Compensation Act 1987* (NSW), s 151AB , and

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(v) whether the Tribunal erred in ordering that WorkCover pay the plaintiff's costs on an indemnity basis from 14 May 2010.

The Court held, dismissing the appeal:

In relation to (i)
(per Basten JA, Allsop P and Meagher JA agreeing)

1. It is appropriate to look at all of the evidence to see whether, by the end of the trial, any error in admitting the written reports of the experts had been overcome by subsequent evidence: [67]

2. The cumulative theory of causation was in issue, about which an expert could helpfully opine. The challenge to the admissibility of the expert evidence must be rejected: [116]-[122]

Clark v Ryan [1960] HCA 42; 103 CLR 486 distinguished.

(per Allsop P, Meagher JA agreeing)

3. Professor Henderson's evidence was not inadmissible to prove the causal relationship between the exposures and the lung cancer. His evidence drew upon his specialised knowledge. The material crosses the line at which lay analysis can discern any inadequacy of the requirements of *Evidence Act 1995* (NSW), s 79 : [26]-[28]

Dasreef v Hawchar [2011] HCA 21; 243 CLR 588 applied.

4. Professor Henderson rationally explained his opinion by reference to facts, assumptions and specialised knowledge, and was not expressing an opinion about heightened risk: [31]

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Bendix Mintex Pty Ltd v Barnes (1997) 42 NSWLR 307; *Evans v Queanbeyan City Council* [2011] NSWCA 230 distinguished.

5. As Professor Henderson is a pathologist, if he otherwise has the basis to express an opinion, he is concerned with the science of the causes and effects of diseases: [34]

Amaca Pty Ltd v King [2011] VSCA 447 approved; *Amaca Pty Ltd (under NSW administered winding up) v Booth* [2011] HCA 53; 86 ALJR 172 applied.

In relation to (ii)
(per Basten JA, Allsop P and Meagher JA agreeing)

6. Increase in risk is not to be equated with factual causation: [98]

Amaca Pty Ltd (under NSW administered winding up) v Booth [2011] HCA 53; 86 ALJR 172 applied.

7. In circumstances in which the negligence of each defendant was insufficient to effect the damage, but the combined force of two was sufficient, each should be held liable in respect of an indivisible outcome: [139]-[140], [145]

Bonnington Castings Ltd v Wardlaw [1956] AC 613 applied; *March v E & MH Stramare Pty Ltd* [1991] HCA 12; 171 CLR 506; *Amaca Pty Ltd v Ellis* [2010] HCA 5; 240 CLR 111; *Amaca Pty Ltd (under NSW administered winding up) v Booth* [2011] HCA 53; 86 ALJR 172 discussed.

(per Allsop P, Meagher JA agreeing)

8. Under general law principles, the counterfactual satisfaction of the but for test is not essential: [49]

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March v E & MH Stramare Pty Ltd [1991] HCA 12; 171 CLR 506; *Amaca Pty Ltd (under NSW administered winding up) v Booth* [2011] HCA 53; 86 ALJR 172; *Strong v Woolworths Ltd* [2012] HCA 5 applied.

In relation to (iii)
(per Basten JA, Allsop P and Meagher JA agreeing)

9. No error of law was demonstrated in the manner in which the Tribunal dealt with the failure of the appellants to adduce expert evidence: [126], [128]

In relation to (iv)
(per Basten JA, Allsop P and Meagher JA agreeing)

10. There does not appear to have been any issue raised before the Tribunal that the date of Mr Sim's employment with the second employer extended past the date of expiration of the relevant insurance policy, nor was it suggested that the insurance policy terminated before the date of sale of the business: [163]

In relation to (v)
(per Basten JA, Allsop P and Meagher JA agreeing)

11. It may be that the provisions of the Dust Diseases Tribunal Regulation 2007 (NSW), Pt 6 , only operate to the extent that they are not inconsistent with the equivalent provisions in the Uniform Civil Procedure Rules : [168]

12. No error of law was shown in the Tribunal's conclusion that the plaintiff made a valid offer of compromise and was entitled to indemnity costs: [173], [175], [180]

JUDGMENT

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1. **ALLSOP P**: I have read the reasons of Basten JA. I agree with them and with the orders he proposes. I would add the following reasons.

2. First, the scope of the appeal on a question as to the admission or rejection of evidence under the *Dust Diseases Tribunal Act 1989* (NSW), s 32 was not the subject of debate. No one argued that in such an appeal this Court was prevented from examining factual questions relevant to the evidential question at hand. That was the correct approach. Some questions of evidence require factual analysis and conclusion, such as a view on the expertise of a witness. In order for this Court to be able to deal with (and correct, if necessary) the rulings on evidence by the Tribunal, it must be able to address any relevant factual questions. It is unnecessary, however, to consider the scope otherwise of the operation of the *Supreme Court Act 1970* (NSW), s 75A .

3. The appeal was conducted at two principal levels: first, whether the relevant parts of the expert evidence of Professor Henderson and Drs Bryant and Young were admissible; and secondly, if so, whether the evidence could support the primary judge's finding of a causal link sufficient to found responsibility in each of the defendants for Mr Sim's death from cancer.

4. The first level of argument depended on the validity of two propositions that were closely related. The principal vice of Professor Henderson's evidence (and, through it, of that of Drs Bryant and Young) was the absence of rational explanation drawn from his expertise about the causal connexion in Mr Sim's individual case between the asbestos fibres inhaled through the breach of duty by each defendant and Mr Sim's lung cancer. The related vice was that Professor Henderson had no expertise in drawing causal conclusions from primary material facts. The conclusion was one of legal cause and not for Professor Henderson to draw, however eminent and well-qualified he might otherwise be, as a pathologist. For the reasons given by Basten JA and in what follows, both these propositions should be rejected.

5. The second level of argument that worked on the assumption of the admission (and lawful admissibility) of the relevant evidence was directed to the inadequacy of the material to supply any evidential foundation for the causal conclusions drawn by the primary judge. This was approached in argument by reference to the reasons of the primary judge. With respect, though giving a helpful elaboration of the essentials of the argument, and to the essential elements of the asserted error of the primary judge, the matter is to be assessed by reference to the whole of the evidence. That is not the only difficulty. If the evidence is of no probative value in the proof of the causal responsibility of any of the defendants (even if it "passes muster" as opinion evidence as to something because of the presence of adequate explanation sourced in relevant expertise) it must be because it lacks probative value for the purposes of the *Evidence Act 1995* (NSW), s 55 .

6. Central to the success of the argument at this second level was the proper approach to causation and, in particular, the meaning in the Australian common law of "material contribution" in such expressions of principle as in *March v Stramare (E & M H) Pty Ltd* [1991] HCA 12; 171 CLR 506 at 514, and cf *Norton Australia Pty Ltd v Streets Ice Cream Pty Ltd* [1968] HCA 61; 120 CLR 635 at 643. It was submitted, as I ultimately understood the argument, that it was necessary for proof of "material contribution" in any tortious causal analysis (at least in negligence) that the plaintiff prove that were it not for ("but for") the defendant's breach the damage would not have occurred. For the reasons given by Basten JA

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and in what follows, this proposition should be rejected. I should say at this point that the reasons of the primary judge commenced (at [2]) with the positing of the relevant questions conformably with this proposition of the appellants:

> "The principal issue for determination is whether the evidence called by Mr Sim satisfies the "but for" test of causation in relation to the causal contribution by any particular period of employment to Mr Sim's disease of lung cancer."

7. The argument in this Court, and in essence his Honour's own reasoning ranged wider than this. This was the case because it can be readily accepted that Professor Henderson disavowed any opinion that without any one of the exposures by reference to periods of employment the cancer would probably not have occurred. Further, there was evidence that the load of asbestos inhaled in each year of employment was probably sufficient to cause cancer in Mr Sim.

The operation and application of the *Evidence Act* , s 79

8. Section 79 is an exception to the general rule in the *Evidence Act* , s 76 of the inadmissibility of opinion evidence. In *Dasreef Pty Ltd v Hawchar* [2011] HCA 21; 243 CLR 588, the judgment of French CJ, Gummow J, Hayne J, Kiefel J and Bell J emphasised (at 603-604 [35]) that the conditions necessary for admissibility of an expert opinion under s 79 are: that the witness has specialised knowledge based on training, study and experience and that the opinion is wholly or substantially based on that specialised knowledge, and, of course, that the opinion is one relevant to a fact in issue. Their Honours also said (at 604 [37]) that ordinarily the expert's evidence must explain how the field of specialised knowledge in which the witness is expert by reason of training, study or experience and on which the opinion is based applies to the facts assessed or observed so as to produce the opinion, paraphrasing Heydon JA in *Makita (Australia) Pty Ltd v Sprowles* [2001] NSWCA 305; 52 NSWLR 705 at 743-744 [85] and referring to Gleeson CJ in *HG v The Queen* [1999] HCA 2; 197 CLR 414 at 427 [39] . As their Honours then recognised in the same paragraph, in the ordinary run of cases this may not be an onerous task.

9. **Following paragraph cited by:**

Berhero Pty Ltd v Hinds (30 August 2023) (Rees J)

80. As Allsop P also noted in *Allianz Australia Ltd v Sim* [2012] NSWCA 68, the requirements of section 79 “should not be elevated into something more than they are: procedural rules to limit evidence to that which is rational and coherent and properly arising from expertise and directed to areas in respect of which the court needs assistance”: at [9] .

The recognition of the requirement to explain how the opinion is drawn from the available facts and how it is based on the specialised knowledge of the witness should be understood as the enunciation of a procedural rule for the admission of evidence as available to be

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considered by the tribunal of fact. As such it should (unless the statutory words demand to the contrary) be understood and applied as a facultative rule to promote the just and efficient resolution of disputes. This is achieved by not permitting, through loose application of the requirements of the statute, voluminous or unsourced opinions unfairly to vex tribunals and other parties in litigation. It is also achieved by recognising that the two requirements of the section and the explanation that is ordinarily required as to how the opinion applies to the facts should not be elevated into something more than they are: procedural rules to limit evidence to that which is rational and coherent and properly arising from expertise and directed to areas in respect of which the court needs assistance. The section nowhere calls for, and its purpose is not, the limitation of expert evidence to that which is scientifically or logically complete, in its assumptions, in its appreciation of intervening events and in explaining how an event or circumstance arises. In some scientific explanations, the absence of particular knowledge as to how an event occurs at a particular stage of an otherwise known process may destroy the ability to form a reliable opinion about the process, including a causal relationship within the process. One could well imagine that views may differ among experts as to the importance (or not, as the case may be) of the absence of the particular knowledge. It would then be a matter for resolution by considering and weighing competing expert views as to whether what was known, and what was not known, enabled an impugned expert opinion to be reliably or responsibly drawn. Of course, if the impugned expert opinion so lacks apparent basis in specialised knowledge or contains no explanation as to how the specialised knowledge applies to the facts such that the lay mind of the tribunal can confidently assess such matters without the assistance of competing expert opinion, the tendered evidence will be rejected. There is a real limit to such a course being taken when the debate about the legitimacy of the opinion as being drawn from the specialised knowledge and about the adequacy of the explanation as to how the specialised knowledge applies to the facts appears to involve evaluation and judgment requiring the very expertise under consideration. At that point, bearing in mind the practical procedural purpose of s 79 , it may be difficult to see any other course consonant with practical common sense and running an efficient trial than to admit the evidence, perhaps conditionally as being initially satisfied of its relevance.

10. Here, two full days of argument were taken, minutely examining the linguistic and logical context of a significant body of medical and scientific evidence proffered by an expert of world standing in his field. This exercise was in order to persuade three appeal judges, who lacked any working expert scientific skills (and lacking the knowledge of forensic training first instance judges sometimes obtain in a bespoke way in preparation for the running of a trial concerning a specialised subject matter) that the opinions proffered were not based on the deep body of specialised knowledge of the witness or not sufficiently explained as to be more than reasonless assertions unrelated to that which could be legitimately drawn from the facts, using the relevant specialised knowledge. All this without a word of contrary expert material; and without the slightest suggestion that the expert's views were tainted by bias, interest or considerations of credit, in any way whatsoever. The conclusions could be drawn, it was submitted, from lay reading of the specialised knowledge, the facts and the opinions. It is difficult to conclude that a provision such as s 79 should work like this by reason of the words of the statute or judicial exegesis of the method of its application.

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11. The first matter to consider is the specialised knowledge of Professor Henderson. His report of 4 June 2009 contains evidence as to his training, study and experience. The evidence, in particular at Joint Blue Book Vol 5 of 5 pp 1931-1932, suffices for present purposes to identify his eminence in his field of the pathological study of asbestos related disorders.

12. In the report of 4 June 2009, his view was expressed "at a high order of confidence" that Mr Sim's "probable/presumptive lung cancer is attributable to his total cumulative and heavy occupational exposure to asbestos (with clinical asbestosis), by way of a significant causal contribution." That was a conclusion that did not seek to differentiate the causal contributions of the respective defendants who were the sequential employers of Mr Sim. No significant attack was made on the conclusion that Mr Sim suffered from lung cancer and that that lung cancer was, on the balance of probabilities, caused by asbestos exposure.

13. What was necessary for legal liability of the appellants was evidence that inculpated one or more of the employers such that it could be said that the exposure in the respective employment period caused or materially contributed to Mr Sim's lung cancer.

14. In Professor Henderson's later report of 19 April 2010, he expressed the conclusion that the exposures in each of the employment periods made a significant and substantial causal contribution to the development of his lung cancer. He said:

> Joint Blue Book Vol 5 of 5 pp 2105-2106
> "It is known that asbestos can induce lung cancer, the causal relationship being one of a linear dose-response effect with no threshold. That is, the greater the inhaled 'dose' the greater the risk of lung cancer and the causal contribution by asbestos for lung cancer induction. Lung cancer differs qualitatively from asbestosis and falls into the class of malignant neoplastic diseases. It is considered to be a 'singular' injury, in that once it has come into being, further exposure to its causal factors (e.g., cigarette smoke; asbestos) cannot significantly worsen the injury (cancer) and, conversely, withdrawal of the causal factors cannot reverse the cancer.
>
> ...
>
> Because asbestosis itself represents a dose-response outcome of asbestos exposure, it follows that each of Mr Sim's asbestos exposures related to each of his employments with Australian Asbestos, Asbestospray Corporation and Bells Asbestos made a significant and substantial causal contribution to the development and severity of his asbestosis, as a matter of high probability.
>
> ...
>
> Because the likelihood and pathogenesis of lung cancer are governed by a dose-response relationship, it also follows on a probabilistic basis that each of Mr Sim's asbestos exposures related to each of his employments with Australian Asbestos, Asbestospray Corporation and Bells Asbestos made a significant and substantial causal contribution to the development of his lung cancer.
>
> The basis for my opinions expressed above is covered in the review published in 2004."

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15. The "review published in 2004" referred to in the last sentence quoted above was in evidence. It was authored by Professor Henderson and three others, Klaus Rodelsperger and Hans-Joachim Woitowitz from the Department of Occupational and Social Medicine at Justus Liebig University, in Giessen in Germany, and James Leigh of the Centre for Occupational and Environmental Health, School of Public Health, University of Sydney. It is too long to quote extensively, beyond the following extracts that deal with the cumulative exposure model, the capacity of asbestos to produce damage to the DNA and the lack of necessity for asbestosis or fibrosis for the inducing of cancer:

> Joint Blue Book Vol 5 of 5 p 2110
> "The authors conclude that (i) the prevailing evidence strongly supports the cumulative exposure model; (ii) the criteria for probabilistic attribution of lung cancer to mixed asbestos exposures as a consequence of the production and end-use of asbestos-containing products such as insulation and asbestos-cement building materials - as embodied in The Helsinki and AWARD Criteria - conform to, and are further consolidated by, the new evidence discussed in this review; (iii) different attribution criteria (e.g. greater cumulative exposures) are appropriate for chrysotile mining /milling and perhaps for other chrysotile-only exposures, such as friction products manufacture, than for amphibole-only exposures or mixed asbestos exposures; and (iv) emerging strong evidence on genetic susceptibility/resistance factors for lung cancer risk as a consequence of cigarette smoking, and potentially also asbestos exposure, suggests that genotypic variation may represent an additional confounding factor potentially affecting the strength of association and hence the probability of causal contribution in the individual subject, but at present there is insufficient evidence to draw any meaningful conclusions concerning variation in asbestos-mediated lung cancer risk relative to such resistance/susceptibility factors.
>
> ...
>
> ibid p 2119
> There is increasing evidence that the capacity of asbestos to induce oxidative damage to DNA is an important mechanism for asbestos-mediated carcinogenesis and for fibrosis; there is a well-recognised dose-response effect for both asbestos-related cancers and fibrosis, but there is no proven sequential or obligatory mechanistic linkage between fibrosis and carcinogenesis. This issue has been summarised by Nelson *et al.*: 'Both fibrosis of the lung and cancer of the lung are dose-related occurrences ... consequently ... [the] majority of cancers will occur in those people who have the highest exposure ... [and who] ... will be the most likely to have asbestosis, regardless of whether the process that produces lung cancer has anything to do with fibrosis. ... Only if the biologic process that gives rise to fibrosis itself also directly induces genetic changes important for the production of lung cancer (or creates conditions that enhance the likelihood of these mutations in relevant cells) can it be *necessary* for interstitial lung disease to be present for asbestos to cause lung cancer. [Little] direct evidence that this occurs has been presented to date. Thus, it can be said that ... there is no direct evidence that there is any necessity for asbestosis to be present for a lung cancer to be caused by [asbestos]' (p.478; italics in the original).
>
> ...

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ibid p 2132
Detailed discussion of the molecular and genetic aberrations inducible by asbestos in experimental animals and cultured cell lines lies outside the scope of this review ... However, asbestos is known to be genotoxic and clastogenic, with the capacity to induce DNA strand breaks, anaphase-telophase abnormalities and sister chromatid exchanges in cell lines *in vitro* - where fibrosis cannot be implicated - and free radicals generated from the surface of asbestos fibres or macrophages are implicated in these aberrations. Both crocidolite and chrysotile have been shown to disturb cell division, producing binucleated cells, which may lead to aneuploidy or polyploidy. Asbestos fibres can also induce oncogene expression - such as *c-fos* and *c-jun* proto-oncogenes - in cultured rodent mesothelial cells. Asbestos-related adenocarcinoma of lung is also associated with *p53* and *k-ras* mutations.

...

Ibid p 2135
On the basis of prevailing evidence, the cumulative exposure model for lung cancer induction by asbestos appears to conform to modern approaches to assessment of causality, with coherence of data across multiple different types of investigation that include dose-response data from epidemiological studies and case-referent studies based on lung tissue fibre measurements; the evidence also encompasses a variety of pathological observations that include the separate and combined clastogenic and mutagenic effects of asbestos and tobacco smoke on cell lines *in vitro* and on bronchiolar epithelium *in vivo*. In terms of generalisability, the cumulative exposure model appears to have explanatory-predictive value: after the 25 fibres/mL-year standard was introduced in Germany - where attribution is primarily an administrative exercise, so that decision-making is less likely to be skewed than by adversarial court-based systems of compensation - the excess lung cancer to mesothelioma ratio has shown close agreement with the same ratio obtained from multiple epidemiological investigations."

(Endnotes omitted.)

16. Professor Henderson wrote a later report dated 31 August 2010 in response to the views of Professor Berry (whose report was exchanged, but who was not called). The report is to be read as a defence by Professor Henderson of his opinions as to the causal relationship of all the exposures of Mr Sim by the three appellants, a matter contested in the report of Professor Berry. The whole section entitled "Causation in a claimant" on pp 7-10 of Professor Henderson's report is directly relevant. (See Joint Blue Book Vol 5 of 5 pp 2177-2180.) In it, Professor Henderson seeks to explain why differentiation between employers, such that one can be and another not be causally responsible, cannot be done. He explained that this was the case because, if lung cancer occurs because of exposure to asbestos (as on the probabilities it did here), the induction of asbestos fibres into the lung and respiratory structure that were there retained all played a necessary, cumulative and incremental part in the development of the disease. At the risk of unnecessary lengthening of these reasons I set out the following from this report:

Joint Blue Bok Vol 5 of 5 pp 2178-2180
"The point is that lung cancer is considered to develop by way of a multi-stage model

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of carcinogenesis, analogous to that which applies to the development of mesothelioma when caused by asbestos, but in most cases of asbestos-related lung cancer cigarette smoking is an important causal co-factor.

In this context, it is worth noting the following:

Because of the apparent capacity of asbestos fibres to participate at several stages of cancer (*lung cancer; mesothelioma*) induction (please see the attached document *MM_Overview_Schematic*, reproduced from reference 24), **all exposures to asbestos in an individual patient must be considered cumulatively to play some part in causation of the cancer in question.**

Current understanding of the pathobiological mechanisms for lung cancer and mesothelioma development indicates that epithelial/mesothelial cells are being initiated, initiated cells promoted, and altered cells proliferating at different times, apparently and at least in part as a result of free radical generation, with oncogenes and tumour suppressor genes being activated and inactivated. At some stage, the proliferative airway/mesothelial cells are thought to become resistant to apoptosis. At the same time, fibres are cleared at different rates and, if exposure is continuing, they continue to be deposited in the lung. [At this point he gave a reference for a detailed exposition of the mechanisms of mesothelioma induction.]

All these processes at a cellular level are probabilistic: i.e., the probability of fibre /cell interaction (or, more accurately, fibres/cells interactions) depends on the number of fibres and the number of cells involved at any point in time and space. Hence, simplistically, the greater the number of asbestos fibres, the more free radicals (generated from the fibres or their interactions with cells such as macrophages) and the greater probability of initiated, promoted or proliferative cells at any given time point.

From a current understanding of the multistage model for cancer induction by asbestos, it follows that each of all exposures to asbestos - recalled or unrecognised - contributes incrementally and cumulatively to the singular disease outcome lung cancer or mesothelioma (provided that the latency interval between any one exposure and the subsequent diagnosis of the cancer is not too short for a carcinogenic effect).

From the preceding discussion it is evident that the risk and causal contributory effects of asbestos towards lung cancer or mesothelioma induction are dependent upon the cumulative exposure to asbestos, modified by asbestos fibre types, and in years following commencement of the exposures. Provided that the latency interval is appropriate (i.e. more than 10 years following the beginning of any exposure), each of any identified above-background exposure makes a causal contribution towards the induction of the lung cancer or mesothelioma, the proportional causal contribution being modified by the three factors mentioned in the preceding sentence. **Therefore, it follows that when there are multiple exposures with an appropriate latency interval, each one of those exposures makes a causal contribution towards lung cancer induction. It also follows that one cannot point to any one exposure as being responsible for the lung cancer entirely, with**

exculpation of the others. Furthermore, one cannot point to any one exposure and exculpate it and to blame all of the others. All exposures contribute to the final outcome (i.e., the lung cancer).

I agree with Prof. Berry that **not all asbestos fibres initially inhaled actually participate directly in lung cancer induction. When airborne asbestos fibres are inhaled, a proportion will be filtered out by the upper nasal passages (although humans frequently convert from nose breathing to mouth breathing when exerting themselves). A proportion of the fibres which successfully bypass the upper respiratory passages will be deposited in the bronchial walls, and amphibole fibres such as crocidolite and/or amosite will tend to persist, although some will translocate away from bronchial walls and others will reach the pleura. Nonetheless, those that remain within the airway structures and eventually induce the lung cancer represent a proportional fraction of those fibres first inhaled, and the more fibres that are deposited (when there were multiple exposures over time), the greater the probability of induction of chemical messengers such as reactive oxygen species *and the amounts of such reactive chemicals*, which can damage the DNA of the cells. Put simplistically, the more fibres deposited in airways, the greater the probability of key interactions with airway epithelial cells and the greater amounts of chemical messengers produced by the cells-fibres interactions, with the greater probability that different asbestos fibres interacting with cells over multiple generations of cells, eventually and cumulatively lead to the production of a lung cancer. To reiterate: these cells-fibres effects do not occur between a single asbestos fibre and a single airway epithelial cell: rather, the situation is one of multiple asbestos fibres interacting with multiple airway epithelial cells over multiple generations of those cells (please see preceding discussion and the analogous situation for mesothelioma induction by asbestos, as discussed by Hammar et al).**

In other words because tobacco smoke and asbestos fibres are thought to participate at multiple different points in the causal chain leading to the development of lung cancer, the cancer itself is thought to be the outcome of reactions between a proportion of the inhaled and deposited fibres, and the greater the numbers of those deposited fibres the greater is the likelihood of lung cancer induction."

(Endnotes omitted; emphasis added.)

17. It is evident that Professor Henderson recognised the gaps in scientific knowledge in understanding the process. He used expressions such as "probabilistic". But it is tolerably clear from an overall reading of this evidence that his opinion drawn from his specialised knowledge was that if a lung cancer is caused by asbestos, the fibres induced into and that remained within the airway structures induced the lung cancer, and that he was not in those circumstances limiting himself to a statement of risk.

18. Professor Henderson was examined, and cross-examined, at length. The oral evidence reflected the content of his reports, and, in answer, the attack made (in the Tribunal and on

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appeal) that he simply had no scientific ground for the opinion that in Mr Sim's case all three periods of exposure were causally relevant to the onset of Mr Sim's cancer. The following passages are of assistance in appreciating the debate in the course of evidence.

In chief

19. In relation to his rejection of Professor Berry's opinion, Professor Henderson stated:

> Joint Black Book Vol 1of 2 p 245
> "A - The simple fact is that if one has say four separate asbestos exposures, one cannot fractionate a causal effect or even one stage in the multi step progression to an indivisible outcome like lung cancer, and attribute that stage to one of those four exposures. The point is, and a point that I think is overlooked in the quoted passage of text, that -
>
> Q. From Professor Berry. A - That's right, is that amphibole fibres in particular which are known to be the most carcinogenic forms of asbestos, are characterised by biopersistence, that is once deposited in tissues, they will remain there for year after year after year, and they will interact with cells such as macrophages and they will generate toxic chemicals known as reactive oxygen and reactive nitrogen species, which are known then to be injurious to the DNA of various cells, for example, airway epithelial cells in the case of lung cancer. **So that when you have multiple exposures, one cannot say that one stage is related to exposure 1 and that that exposure or its effects then cease and then one goes on to exposure 2, because the fibres are persistent to the tissue year after year and as additional exposures occur, additional fibres are deposited in the tissues, so that there are more fibres interacting with more cells generating more toxic chemicals increasing the toxicity to the DNA of airway epithelial cells and increasing the probability of a set of mutations in what is a chain of necessary causal preliminary events on the pathway to lung cancer which is an indivisible and singular outcome.** So my problem there is that the quoted passage of text involves speculation on pathogenesis which is at variance with what we know about the biopersistence of asbestos fibres in tissues, and also the multi step model of lung carcinogenesis.
>
> ...
>
> Ibid pp 246-247
> Q. ... A - ... **So the difference is between the non-necessity for this causal chain in the throwing of the darts, but the dire necessity - in fact, it is essential - that one moves from one mutational event to another, ultimately striking the right sequence or combination of mutational and other events necessary to lead to a lung cancer. So there is a necessary causal chain.**
>
> HIS HONOUR
>
> Q. So if we do not look at darts, but 99 to 100 fibres, every fibre does its part . A - **Ever y deposited fibre potentially does its part, yes.**

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MR SEMMLER

Q. Going on to the next page, at the top of page 8 you use the expression, 'The hundred darts do not constitute an obligate chain of events essential for the score of a triple 20.' Does the exposure in four separate periods, as in Mr Sim's case, to asbestos, with four separate employers - does each exposure constitute an obligate chain of events essential for the ultimate development of the cancer. A - **Well, in the development of that particular cancer my answer is in the affirmative; but, yes, that each of those exposures will increase the likelihood that a cancer will develop, but the fibres from the first exposure remain throughout the other periods of exposure and are added to by the fibres inhaled and deposited by the subsequent exposures, so that each has incrementally increased the likelihood that a lung cancer will develop, and in this case that likelihood has been translated into the actual development of lung cancer.**

Q. **When the lung cancer develops and the risk expressed as a likelihood has come home, is it possible then to exclude any of the four periods of exposure to asbestos with each for the employers in the explanation for the reality of the cancer. A - Well, the answer to that is no, one cannot exclude any one and blame all of the others. The point is that all of them have contributed to the load of inhaled and deposited asbestos fibres in lung tissue, or a fraction of those fibres have interacted with the cells to produce the toxic chemicals involved in producing mutations. Those mutations have increased in number as a result of the fact that asbestos fibres can interact with cells at multiple different points along the causal chain, leading up to the development of the lung cancer.** ,... **one cannot point to one and blame it and exculpate the others; and equally, one can't point to one and exculpate it and then blame all of the other three exposures. The point is that all of them have contributed to the fibre burden in lung tissue, all have contributed to the risk which is then translated into the actual occurrence of the lung cancer.**

Q. Yes. And when you say it is translated into the actual occurrence of the lung cancer, you mean that once the risk comes home each of them has caused that cancer. A - That's right, yes."

20. Having been taken to a chapter in a text entitled "Asbestos Risk Assessment, Epidemiology and Health Effects", he was asked and answered as follows:

Ibid pp 247-248
"Q. ... Does the chapter that you have there in your hand - does that provide perhaps a more comprehensive and detailed explanation of the patho-biological mechanisms for lung cancer and mesothelioma development at a cellular level. A - Well, yes, it's a more widespread account. It's dealing with the cellular and molecular events that take place between asbestos and various cells and tissues, including malignant transformation near the end of the chapter; but it also addresses other things like asbestosis.

Q. Yes. Do you accept that that is a reasonable explanation of that phenomenon that you have just described. A - Yes, I think it is a reasonably - not entirely up-to-date, but a reasonably current account of some of the events that are thought to take place

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between deposited asbestos fibres in various cells and the pathways that were thought to lead to asbestosis and the cancer, such as lung cancer or mesothelioma."

21. The evidence in chief continued, by way of the explanation of a diagram contained at p 2429 of Joint Blue Book Vol 5 of 5. Professor Henderson said:

> Joint Black Book Vol 1 of 2 pp 249-250
> "Q. It is because of the potential for persistence of fibres from years earlier and the understanding that they interact at a later time that one cannot exclude an earlier in time exposure. A - That's correct, because it's known that amphibole fibres once deposited in tissues tend to be bio persistent and they remain stable interacting with cells for varying periods of time. There is some evidence about 10% of the fibres may be cleared each year but it means they have a very long half life in tissues so that if you have one episode of asbestos inhalation a proportion of the fibres will be deposited in lung tissues. They will remain there for many years thereafter interacting with cells causing mutations and the like. Another episode of fibre inhalation will be superimposed on that so that one had an incremental increase in the number of fibres. As the number of fibres increased the amounts of toxic chemicals generated by those fibres as a result of interaction with various cells will increase and hence the likelihood of key mutations involved in the multi-stage pathway will occur and those mutations will accumulate ultimately leading to a singular event called the development of a lung cancer or a mesothelioma. So I was just trying to give some broad understanding of some of the steps which are involved."

Cross-examination

22. In cross-examination, Professor Henderson was taxed with the proposition that available medical and scientific evidence did not enable an opinion to be given, in any particular case, as to what causal relationship any particular asbestos exposure had to an assumed lung cancer. All that could be stated, it was put to him, was that there were risk factors of which exposure to asbestos was one and that such exposure heightened the risk of cancer. One could not, it was put to him, translate that into an opinion about the causal responsibility of any particular exposure to a lung cancer. It was put to him that all that science tells us could be summarised in four propositions that are paraphrased as follows:

    (1) There are cohorts of asbestos workers who were subject to substantial exposure who have been studied and who show an increased incidence of lung cancer compared to those who did not have that exposure (Joint Black Book Vol 1 of 2 p 255 P).

    (2) Work has been taken further and a dose response relationship has been derived which is a linear dose response relationship (ibid p 255 Q). (In relation to that relationship Professor Henderson agreed that epidemiology has taken us to the stage of saying, RR [relative risk] equals I plus K times E, and the occupational hygienists may or may not be able to tell us in particular circumstances whether K or E can be determined with any precision (ibid p 257 G)).

    (3) Asbestos is biopersistent (ibid p 257 J).

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(4) Asbestos fibres have been shown to initiate biochemical processes which may lead to cell mutation and also proliferation of cells that have damaged DNA, directly or indirectly, from generation of toxic chemicals from the fibres themselves or from the fibre cell interactions particularly with macrophages (ibid p 257 K-M).

23. As to the fourth proposition the following exchange took place:

> Ibid p 257
> "Q. And that is a not at all uncommon feature of other carcinogens. A - That is correct. For example ionising radiation also develops similar reactive chemicals and some of the carcinogens in tobacco smoke generate similar toxic chemicals like reactive oxygen species.
>
> Q. And those toxic chemicals that you describe, can have either the direct or the indirect effect on the cells, direct in the sense that they may induce mutations in the cells. A - That's correct.
>
> Q. Or indirect in the sense that they may interfere with the body's natural defence mechanisms, [that] ordinarily would control and eliminate rogue cells. A - Well, that may be part of carcinogenesis but it is a much more speculative one, but yes.
>
> Q. What I have just put to you is a possibility but you would regard it as speculative. A - Well, in case of asbestos I would regard it as more speculative.
>
> Q. But it is established in relation to other carcinogens is it. A - Well, yes, some of other carcinogens are known to depress cell immunity.
>
> Q. But in asbestos that is not - sorry, you do not regard that as being - A - Well, I don't regard it as being one of the really important factors. The important interaction is thought to be the fibre macrophage interaction and they may well have secondary effects further down the track on lymphocytes and other arms of the immune system, but the essential factor is the fibre cell or rather fibres cells' interactions with the generation of reactive oxygen species, which are known to be damaging to the DNA."

24. It was then put to Professor Henderson that that was "all that science tells us relevantly." (Joint Black Vol 1 of 2 p 257 X.) Professor Henderson did not accept this. He said:

> Ibid p 258
> "A. **Well, I think it goes beyond that because a number of the steps in a mutational pathway towards cancer have been established, and there is now a - although the process is incompletely understood, there is now good evidence for a number of the mutational steps and one can name individual genes involved.**
>
> Q. Well, can we include all of that as falling within the general statement in the fourth proposition. A - Right.
>
> Q. **Now, understanding the propositions in those broad terms, is what I put to you correct, that those four propositions exhaust what science can tell us about the relationship. A - I don't think they exhaust it**."

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25. Meticulous cross-examination then followed in aid of the propositions as to the limits of science identified. This included the following exchange:

> Ibid pp 263-264
> "Q. Now the creation of genomically unstable cells is something that happens even without asbestos exposure, correct. A - Yes, people develop mutations spontaneously.
>
> Q. So it can happen spontaneously, correct. A - Yes.
>
> Q. Or other carcinogens can be involved. A - Yes.
>
> Q. Such as ionising radiation. A - Yes.
>
> Q. Or cigarette smoke. A - Yes.
>
> Q. And that may happen either at the initial stage when the first, and I call it, the ancestor cell which is the first genomically unstable cell was created or at any of the descendant stages. A - Well, yes, just as the asbestos fibres interact either at the first and as they accumulate the subsequent stages in cancer development.
>
> Q. So you could have one genomically unstable cell that had been started by ionising radiation and then another at later generations that could accumulate a further mutation and that might be the produce of an asbestos fibre. A - **Yes, but all the asbestos fibres then are doing is adding on to a previous mutation and furthering the development of the necessary causal chain of the mutations necessary for cancer development.**
>
> ...
>
> Ibid p 264
> Q. So looking at the ultimate development of the cancer, it is possible to trace it back to a series of finite number of individual events at which first the ancestor cell has become genomically unstable and then further mutations have occurred to its descendant cells until it eventually becomes a cancer cell and then develops into a tumour, correct. A - **W ell, I don't know what you mean by the expression a series of finite steps. All I'd say is that when you have a deposition of inhaled asbestos fibres especially amphibole fibres those fibres will interact with otherwise normal cells, macrophages release toxic chemicals which can then impact on otherwise normally replicating bronchial epithelial cells and induce mutations and the more fibres that are inhaled and deposited the greater the number of the toxic chemicals, the greater the number of the mutations, the greater number of epigenetic events, for example, autocrine cell stimulation, which will lead to this expanded population of cells and the point is that all of these factors accumulate in a necessary obligate causal chain of precancerous events finally leading up to the final event which becomes the cancer.**
>
> ...
>
> Ibid p 271
> Q. The proposition is that when one reads this report as a whole it is clear that the

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words on a probability basis and the sentence to which I have drawn attention connote an exercise for an individual in the application of a formula RR equals 1 plus K times E. A - **Well, I just do not agree with that proposition. I would not use that equation. I would think that the attribution in an individual patient is based on a variety of considerations and including - and a work history of substantial to heavy exposure as in this case or asbestosis however diagnosed and I don't think one needs to invoke the equation RR equals 1 plus K times E. I don't see the relevance of that to this particular exercise.**

...

Ibid p 274
Q. Now, I asked you some questions earlier about the biochemical processes which are thought to lead to the development of cancer. You will agree with me, will you not, that not all of the fibres which lodge in the lung and produce the adverse effects that you are referring to actually contribute to the chain of causal events which lead to the generation of the particular cancer which may later develop**. A - I agree that not all of the inhaled fibres will actually participate in the process of carcinogenesis, that some of the fibres will migrate away and be translocated to pleura. But what I can say is that a proportion which is likely to be proportionate to the dose of each episode of exposure will participate in the process of carcinogenesis, so one is looking at a fraction, a proportion of the inhaled and deposited fibres which will participate in carcinogenesis.**

...

Q. Well, we do not now how many steps there are or may be, how many mutative steps there are or may be between the original unstable - and I am sorry, I have forgotten your phrase - the ancestor unstable cell. A - Well, we don't know the precise number of steps."

...

Ibid pp 276-279
Q. Would you agree with me that the model that you have described involves an assumption that there is a continued accumulation through the generations of further mutations which, if the cell does not automatically die or undergo apoptosis or be eliminated by the body's defence mechanisms, eventually results in accumulating enough mutations that the cell then becomes a cancer cell capable of replicating itself and then the cancer grows from that cell. A - Well, that's a fair representation of the process, as we discussed this morning; and again, the more a person smokes cigarettes the greater the likelihood that the carcinogens in the cigarette smoke will lead to an outcome, yet not everybody who is even a heavy smoker will develop lung cancer. It is increasing the probability that the required chain of sequential causal mutational and epigenetic events will actually occur that lead to the cancer; and the point is, the more that one smokes, the more that one is exposed to asbestos, the greater the probability that this chain of events will come to its fruition, if you like. And the point is that each of the, if you like, cigarette smoke or asbestos exposures will contribute to the pool of asbestos fibres, from the first, second, third and fourth exposures, and as the fibre numbers increase the probability that a cancer will develop increases. Each adds to the

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likelihood that a cancer will develop and eventually that is translated, or comes home, or comes into being as a cancer.

Q. When it does, in your language, the fibre in question - that is, the fibre that has been inhaled as a whole - in your language has made a material causal contribution to the cancer which ultimately results. A - What I am saying is that -

Q. That is the sense in which you have used this phrase, 'material contribution or cause,' is it not. A - I don't think I've used the word 'material,' but if I have it has been a mistake on my part.

HIS HONOUR ...

Q. Doctor, can you reply. A - **Well, I try not to use the word 'material,' your Honour. To me that is a legal term, the expressions I use are 'significant and substantial causal contribution', and the point that I am simply trying to make is that the more fibres that are inhaled, the greater the probability that the cancer will develop. So that each of - when you say there were four exposures, each of the exposures will increase the likelihood of a cancer developing. They each increase the likelihood on top of those that have gone before, so that the total likelihood at the end, when it's translated into a cancer, is the total cumulative exposure that has come home to fruition in terms of a cancer.**

Q. In that each additional fibre adds to the chemical soup which is toxic. A - That's correct. Each of the exposures is adding to all of the events and chemicals and mutations which ultimately lead to the cancer, so that when you see a cancer in reality, as expressed in an individual patient, you're looking at the end result of all the risks or likelihoods imposed by each of multiple exposures.

... HIS HONOUR

Q. So that does each exposure create some additional mutations. A - Well, I don't think one can put it quite so precisely, your Honour, because if you have, say, somebody with four exposures to asbestos over, say a period of two or three years each, then the amphibole fibres from the first exposure, a proportion of those will be present in bronchial walls and lung tissue. They will persist. The second exposure takes place and that adds to that pool of asbestos fibres. So does the third and so does the fourth. But the first exposure fibres are still resident, so that the chemical soup and the mutational events are the end result of the cumulative exposure imposed by each of multiple exposures when there is more than one.

Q. So that each exposure creates some additional mutations. A - The likelihood is that each does, that each additional exposure, because it increases the number of fibres, increases the likelihood that the chemical messengers will induce one of the required mutations for the development of a lung cancer. I mean, I can only requote Professor Berry when he says, 'It would be invalid to conclude a lung cancer is caused only by one exposure. There were three other periods of exposure that would have made a contribution to the excess in risk, and there is no scientific basis for saying that only one of those periods caused the cancer.'

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... WITNESS

If one has an exposure which has contributed, say, 60% to the total fibre pool and there are two other exposures, each of which has contributed 20%, I don't think one can say that the cancer which has developed is the outcome, even on a probability basis, from the 60%. It is the total 100% - the 60 plus 20 plus 20 - which is the cumulative exposure which has been brought home in terms of the development of the cancer.

MR PARKER

Q. That is the way you analyse it. A - Well, yes, it is.

Q. And you have said now many times that an additional exposure - or an individual episode of exposure - increases the risk of a cancer ultimately developing. A - Yes.

Q. **But that particular exposure may not in fact play any role in the development of the particular cancer that in fact develops; correct. A - I think - well, I wouldn't say - I don't believe that is correct, because I think it falls into the realm of far-fetched speculation beyond what is available in the biological and scientific evidence. I think that is entirely speculative.**

Q. **Well, most of the cells which develop these mutations ultimately do not go on to grow into a cancer, they either die out or are eliminated by the natural processes that we have discussed; correct. A - Well, that's correct.**

Q. So it's only a fraction of those cells or indeed usually only one that will actually lead to a line which endures down the generations enough and develops the necessary mutations to turn into a cancer. That is right, is it not. A - Well, yes. The majority of the cells will either die out, the mutations will be irrelevant or they will not progress to cancer for a variety of reasons and it requires a complex series of sequential causal steps to cause the cancer but the point is that the likelihood that those essential causal steps in this casual chain will be achieved is increased by the additional asbestos exposures so that one can't point to any one exposure and say well this one on a probably basis has not contributed. That to me is speculation.

Q. Nor can one point to any particular individual one and say on a probability basis this one did contribute. A - Well, as I say **one cannot point to one of, say, four exposures and say that is the exposure responsible, the other three played no role. One cannot equally point to one exposure and say well that played no role it's the other three. The point is, it's all of the exposures in an individual which are ultimately translated into the development of a lung cancer.**

Ibid p 279
Q. You have expressed yourself in your report and in your evidence a number of times as saying all the exposures make a substantial contribution to any cancer which ultimately develops or you have said all the exposures cumulatively cause the cancer which ultimately develops. A. And this is in accordance with current theories on carcinogenesis.

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Q. That is the way you have expressed yourself and in expressing yourself you mean that all of the exposures increase the risk in the way that you have described in your evidence to me. A. That's correct.

Q. **And nothing more. A - Well, I think it means more when the cancer has finally developed because the risk has come home in the form of a cancer.**

HIS HONOUR

Q. But does every additional exposure increase the number of mutations. A - We can't count them because in humans one can't come back and look at the mutations. **The point here is, your Honour, that even the first exposure will produce a series of ongoing mutations. The next exposure will increase the number of mutations and increase the likelihood that one of those mutations is relevant to the development of cancer and so would a third and so would a fourth. One can't neatly fractionate mutations according to each of, say, four exposures because the mutations - the fibres are long resident in lung, new fibres are added and the mutations are ongoing process over 20 to 30 years**."

(Emphasis added.)

26. From the totality of the written and oral evidence of Professor Henderson, illustrated by the above extensive extracts one can see the following:

(1) The consistent and careful attempts by the cross-examiner to have Professor Henderson accept that his expressed views as to necessary causal relevance of retained asbestos fibres in an asbestos induced cancer were no more than hypotheses of heightened risk; and that scientific knowledge did not permit any conclusions in the case of an individual cancer of the causal relevance of any particular exposure.

(2) The rejection by Professor Henderson of those propositions and his opinion for the reasons he gave that scientific knowledge did permit and support his opinion that upon a cancer occurring that could be attributed to asbestos (as here), the retained asbestos fibres were all necessary or obligate parts of the causal chain.

27. In so expressing the matters in (2) Professor Henderson was drawing upon his specialised knowledge. It may be that the better scientific view of the pathology or aetiology of asbestos induced cancer is another quite different hypothesis. For instance, the medical evidence that formed the basis of the findings of fact in *Fairchild v Glenhaven Funeral Services Ltd* [2002] UKHL 22; [2003] 1 AC 32 as to the cause of mesothelioma was summarised by Lord Bingham of Cornhill at 43 [7] as follows:

"... The mechanism by which a normal mesothelial cell is transformed into a mesothelioma cell is not known. It is believed by the best medical opinion to involve a multi-stage process, in which 6 or 7 genetic changes occur in a normal cell to render it malignant. Asbestos acts in at least one of those stages and may (but this is uncertain) act in more than one. It is not known what level of exposure to asbestos dust and fibre can be tolerated without significant risk of developing a mesothelioma, but it is known that those living in urban environments (although without occupational exposure) inhale large numbers of asbestos fibres without developing a mesothelioma. It is

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> accepted that the risk of developing a mesothelioma increases in proportion to the quantity of asbestos dust and fibres inhaled: the greater the quantity of dust and fibre inhaled, the greater the risk. But the condition may be caused by a single fibre, or a few fibres, or many fibres: medical opinion holds none of these possibilities to be more probable than any other, and the condition once caused is not aggravated by further exposure. So if C is employed successively by A and B and is exposed to asbestos dust and fibres during each employment and develops a mesothelioma, the very strong probability is that this will have been caused by inhalation of asbestos dust containing fibres. But C could have inhaled a single fibre giving rise to his condition during employment by A, in which case his exposure by B will have had no effect on his condition; or he could have inhaled a single fibre giving rise to his condition during his employment by B, in which case his exposure by A will have had no effect on his condition; or he could have inhaled fibres during his employment by A and B which together gave rise to his condition; but medical science cannot support the suggestion that any of these possibilities is to be regarded as more probable than any other. There is no way of identifying, even on a balance of probabilities, the source of the fibre or fibres which initiated the genetic process which culminated in the malignant tumour. It is on this rock of uncertainty, reflecting the point to which medical science has so far advanced, that the three claims were rejected by the Court of Appeal and by two of the three trial judges."

28. If the induction of lung cancer were better seen as reflected by the evidence as to mesothelioma as described by Lord Bingham (as the legal submissions of the appellants asserted) that could form the basis of the rejection of Professor Henderson's evidence. But there was no contrary evidence. There was (relevantly) only that of Professor Henderson. I am unable to characterise Professor Henderson's evidence as inadmissible to prove the causal relationship between all these exposures and the lung cancer. In my view, the material crosses the line, by some measure, at which lay analysis can discern any inadequacy of the requirements of s 79 discussed by the joint judgment in *Dasreef v Hawchar* .

29. There was some discussion at the appeal about the statement of opinion on p 9 of Professor Henderson's report of 31 August 2010 as to the latency period:

> Joint Blue Book Vol 5 of 5 p 2179
> "Provided that the latency interval is appropriate (i.e. more than 10 years following the beginning of any exposure), each of any identified above-background exposure makes a causal contribution towards the induction of the lung cancer or mesothelioma, the proportional causal contribution being modified by the three factors mentioned in the preceding sentence. Therefore, it follows that when there are multiple exposures with an appropriate latency interval, each one of those exposures makes a causal contribution towards lung cancer induction. It also follows that one cannot point to any one exposure as being responsible for the lung cancer entirely, with exculpation of the others."

There was no suggestion in the evidence of any dispute or issue concerning these views about the latency period. No submission or cross-examination was directed to this part of Professor Henderson's report. It is not necessary for a witness to dilate upon issues that are not in contest or give explanations that are not necessary.

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30. The evidence of Dr Bryant and Dr Yates may well have been bad in form in the way it simply rolled up agreement with Professor Henderson. That objection, however, was not the burden of the debate. I did not understand any submission to be put that if Professor Henderson's evidence was admissible and adequate to found the Tribunal's conclusions, any defect in the evidence of Dr Yates and Dr Bryant would make any difference to the orders on appeal.

31. The acceptance that Professor Henderson has rationally explained his opinion by reference to facts, assumptions and his specialised knowledge, and the acceptance that Professor Henderson was not expressing an opinion about heightened risk makes irrelevant any criticisms of his analysis based on cases such as *Bendix Mintex Pty Ltd v Barnes* (1997) 42 NSWLR 307 or what I said in *Evans v Queanbeyan City Council* [2011] NSWCA 230. Thus, a response to the historical exegesis in the appellants' submissions of cases such as *Bendix* , *E M Baldwin & Son Pty Ltd v Plane* (1998) 17 NSWCCR 434, *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29; 49 NSWLR 262, *Fairchild* , *Barker v Corus UK Ltd* [2006] UKHL 20; [2006] 2 AC 572 and *Sienkiewicz (Administratrix of the Estate of Enid Costello Deceased) v Greif (UK) Ltd* [2011] UKSC 10; [2011] 2 AC 229 becomes unnecessary.

32. Reliance was placed by the appellants on the scholarly works of Professor Stapleton such as "Factual Causation and Asbestos Concerns" (2010) 126 *Law Quarterly Review* 351. A reading of Professor Stapleton's work on causation in relation to asbestos, such as the above article and "Two Causal Fictions at the Heart of US Asbestos Doctrine" (2006) 122 *Law Quarterly Review* 189 reveals the view that New South Wales Courts have been inconsistent in their treatment of causation. Professor Stapleton noted that after there was a rejection of mere increase in risk as causally sufficient in cases such as *Bendix* , there was a persuasion of courts to accept expert evidence that every exposure to asbestos contributes to the triggering of the process. Professor Stapleton wrote in 126 *Law Quarterly Review* at 355:

> "This approach seems to outstrip the limits of medical knowledge depicted in *Fairchild* , yet when unsuccessful defendants, attacking such evidence, applied for special leave to appeal ... they too were rebuffed: *Jsekarb Pty Ltd v Plane* [1999] HCATrans 368."

33. Professor Stapleton is undoubtedly correct that the evidence accepted by the Tribunal in mesothelioma cases goes beyond what was accepted in *Fairchild* . That may have been a product of errors of fact of the Tribunal in the weighing of expert evidence, though I do not assert that. The point for present purposes is that the appeal to this Court from the Tribunal is limited by s 32 . A mere error of fact, however critical, is unappealable. The question here is whether Professor Henderson's views, which, in the context of mesothelioma, have been the foundation of the Tribunal's factual findings, are admissible in so far as he proffers them in respect of lung cancer. For the reasons that I have expressed, I think they are. They may be wrong. The experience and multidisciplinary training of others, scientists and legal scholars included, may suffice beyond a lay analysis to criticise the acceptability of Professor Henderson's views. With that I do not quarrel. But that was not attempted by the appellants. His views do not suffer the characterisation capable of being made by a judge without expert assistance of unreasoned *ipse dixit*.

34. **Following paragraph cited by:**

Fire & Rescue NSW v Hayman (14 November 2012) (Deputy Bill P Roche)

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> 74. Mr Perry further submitted that Dr Klug used the expression "balance of probabilities" and it was for the Arbitrator to make a determination. Again, this was a surprising submission. It is common practice for medical experts in the Commission to express their opinions in terms of the balance of probabilities and there is nothing wrong with that practice. Expert evidence may be given on factual questions of causation, even where the opinion is expressed in language that is employed in legal analysis in causation (*Allianz Australia Ltd v Sim* [2012] NSWCA 68 Allsop P (Meagher JA agreeing) at [34] [35] , Basten JA at [118] – [121] (Meagher JA agreeing)). It goes without saying that the ultimate decision on liability is for the Commission.

The proposition also put forward that Professor Henderson cannot give expert evidence on causation is, with respect, incorrect. As a pathologist, if he otherwise has the basis to express an opinion, he is concerned with the science of the causes and effects of diseases. In this respect I agree with what was said by Nettle JA, Ashley JA and Redlich JA in *Amaca Pty Ltd v King* [2011] VSCA 447 at [110] ff; and see also *Amaca Pty Ltd (under NSW Administered Winding up) v Booth* [2011] HCA 53; 283 ALR 461 at 475 [49] per French CJ.

35. It is true that the opinions were expressed in language that is employed in legal analysis in causation, but the expressions used were factual phrases capable of expressing meaning in the contexts in which they were used. There was no cross-examination to the effect that these were not Professor Henderson's views but the words given to him by lawyers.

36. In my opinion, Professor Henderson's opinions were admissible and no error was displayed by the Tribunal in admitting them.

Material contribution

37. It is necessary to deal with the second level of argument, that even if admissible, the evidence of Professor Henderson is insufficient to support the conclusion of causation arrived at by the Tribunal.

38. The factual proposition said to support this conclusion was one with which Professor Henderson agreed: it could not be said in respect of each of the appellants that, but for its negligent exposure of Mr Sim to asbestos, he would not have contracted his lung cancer.

39. The legal proposition said to support this conclusion and to be dictated by the High Court's decision in *Amaca Pty Ltd v Ellis* [2010] HCA 5; 240 CLR 111 and the Full Court of the Federal Court's decision in *Merck Sharp & Dohme (Australia) Pty Ltd v Peterson* [2011] FCAFC 128; 196 FCR 145 was as follows: Under the common law of Australia an essential pre-requisite of a conclusion of causation by a factor materially contributing to the relevant event or circumstance is that it be shown that without the factor the event or circumstance would not have occurred. Thus it was submitted that satisfaction of the but for test was essential for the conclusion that each of the three employment exposures contributed to Mr Sim's cancer. That factual premise not being made out, the appellants must succeed.

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40. The phrases "material contribution" or "materially contributes" to a result used in a causal sense are capable of taking on a number of meanings. Care needs to be taken in their use. In this regard, see *Strong v Woolworths Ltd* [2012] HCA 5. For instance, in Canada the phrase "material contribution" has been used as a special or exceptional notion as an alternative to the but for test in connection with the finding in some cases that the defendant has increased the risk of the harm in question: see *Resurfice Corp v Hanke* 2007 SCC 7*;* [2007] 1 SCR 333 at 342-343 [24]-[25] . It can be accepted that the respondent did not advocate and the Tribunal did not conclude that causation was to be concluded by increase in risk alone. Such an approach was rejected in *Ellis*, *Bendix*, *Gett v Tabet* [2009] NSWCA 76; 254 ALR 504 and *Tabet v Gett* [2010] HCA 12; 240 CLR 537 and *Evans* . There was no argument on behalf of Mrs Sim that this clear line of authority should be departed from.

41. The expression "material contribution" has been used authoritatively in Australia as part of the description of relevant causal connection: *March v Stramare* (Mason CJ, with whom Toohey J and Gaudron J agreed). Indeed, the recognition that the primary legal proposition should be rejected can be taken from the first sentence of Mason CJ's judgment in *March v Stramare* that states his disagreement with McHugh J about the but for test (at 508 [1]):

> "Like McHugh J., I would allow this appeal but my reasons for taking this course are rather different from those stated by his Honour as I do not accept that the 'but for' (causa sine qua non) test ever was or now should become the exclusive test of causation in negligence cases."

42. Of course, Mason CJ recognised (at 514 [16]) the often important place of the but for test in causal analysis, but (at 514-515 [16]-[19]) his Honour also discussed its deficiencies in certain kinds of circumstances, in particular where there are conjunctive causal factors. This refusal to accept that proof that the damage would not have occurred but for the defendant's negligent act as always essential was recognised by Gummow J, Hayne J and Crennan J in *Amaca v Booth* at 480 [70] . See also *Amaca v King* at [123] .

43. In *Strong v Woolworths* at [26] French CJ, Gummow J, Crennan J and Bell J reiterated what Mason CJ had said in *March v Stramare* at 514, saying:

> Negligent conduct that materially contributes to the plaintiff's harm but which cannot be shown to have been a necessary condition of its occurrence may, in accordance with established principles, be accepted as establishing factual causation..."
>
> (Citations omitted.)

See also at [28].

44. One of the cases to which Mason CJ made reference in *March v Stramare* at 514 as illustrative of the phrase "materially contributes to" was *Bonnington Castings Ltd v Wardlaw* [1956] AC 613. The reasoning of the Law Lords in *Bonnington Castings* did not involve any counterfactual but for analysis; nor was there any overly refined discussion of expert evidence of the necessary elements of a sufficient set of factors to cause the condition. The silica dust inhaled by Mr Wardlaw brought about his condition of pneumoconiosis. Particles of silica small enough to be dangerous if inhaled came either from the annealing process of the castings or by the working of three kinds of machines in the dressing shop or both. There was

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no way of preventing silica dust from one of the machines, a pneumatic hammer, from escaping, nor was any mask effective. The floor grinders had an effective extraction mechanism, so far as it was possible. The only source of silica dust said to be attributable to the negligence of Bonnington Castings was from the swing grinders, because the dust extraction plant was not kept free from obstruction. The evidence revealed (as Lord Reid said at 621) that the disease is caused by the whole of the noxious material inhaled. Thus, if it came from two sources it could not be attributed solely to either. The relevant question posed by Lord Reid (at 621) was incompatible with the but for test:

> "I am in agreement with much of the Lord President's opinion in this case, but I cannot agree that the question is: which was the most probable source of the respondent's disease, the dust from the pneumatic hammers or the dust from the swing grinders? It appears to me that the source of his disease was the dust from both sources, and the real question is whether the dust from the swing grinders materially contributed to the disease. 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. I do not see how there can be something too large to come within the de minimis principle but yet too small to be material."

The negligence of the employers allowed a proportion of silica dust which was not negligible to be inhaled.

45. As to alternative causes Lord Reid said at 622 and 623:

> "I cannot avoid the conclusion that the proportion which came from the swing grinders was not negligible. He was inhaling the general atmosphere all the time, and there is no evidence to show that his hammer gave off noxious dust so frequently or that the concentration of noxious dust above it when it was producing dust was so much greater than the concentration in the general atmosphere, that that special concentration of dust could be said to be substantially the sole cause of his disease.
>
> ...
>
> No doubt the total amount from both sources in the atmosphere was small at any one time, but the combined effect over a period of eight years was to cause the respondent's disease."

46. The dust that was negligently in the atmosphere helped produce the disease. Lord Tucker put it simply (at 623):

> "the silica dust discharged from the swing grinders contributed to the harmful condition of the atmosphere, which admittedly resulted in the pursuer contracting pneumoconiosis, and was therefore a contributory cause of the disease."

47. The conclusion of material contribution to the causal process in *Bonnington Castings* was reached without over-refinement of measurement and without medical or physiological counterfactual analysis, which would have been both impossible and unreal.

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48. **Following paragraph cited by:**

> May v Military Rehabilitation and Compensation Commission (30 June 2015) (Allsop CJ, Kenny, Besanko, Robertson and Mortimer JJ)
>
> 219. These statements have been often applied: in such cases as *Tubemakers of Australia v Fernandez* (1976) 50 ALJR 720; 10 ALR 303 at 307, 310-311, *EMI (Australia) Ltd v Bes* [1970] 2 NSWR 238 at 241 ; *Amaca Pty Ltd v Booth* [2011] HCA 53; 246 CLR 36 at 61-62 [69] ; *Tabet v Gett* [2010] HCA 12; 240 CLR 537 at 588 [149] ; *Australian Iron and Steel Ltd v Connell* [1959] HCA 54; 102 CLR 522 at 535-536 [6] ; *Allianz Australia Ltd v Sim* [2012] NSWCA 68 at [48] ; *Evans v Queanbeyan City Council* [2011] NSWCA 230; *Webb v Repatriation Commission* [1988] FCA 127 at [18] ; *Australian Telecommunications Commission v Barker* [1990] FCA 489; 12 AAR 490 at 493-494 ; *Major Engineering Pty Ltd v Timelink Pacific Pty Ltd (No 2)* [2009] VSCA 83 at [19] . It is also the point made by Latham CJ in *Hume Steel* : 75 CLR 242 at 252.

Science may struggle to give complete expressions of reasons for pathology and aetiology of disease. Still more is it likely to struggle to express opinions about what might have happened had an indefinable or imprecise body of facts not occurred. For the law to demand that exercise in the working out of responsibility for the consequences of tortious wrongdoing would be to demand unreality. As Dixon J said in *Adelaide Stevedoring Co Ltd v Forst* [1940] HCA 45; 64 CLR 538 at 569, cited by Gummow J, Hayne J and Crennan J in *Amaca v Booth* (at 479-480 [69]):

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

49. The requirement for such counterfactual satisfaction of the but for test was one of the matters that Mason CJ rejected in *March v Stramare*, that Gummow J, Hayne J and Crennan J can be taken to have rejected in *Amaca v Booth* at [70] and that French CJ, Gummow J, Crennan J and Bell J rejected in *Strong v Woolworths* at [26]-[28] .

50. Nothing in *Amaca v Ellis* is to the contrary. There the evidence failed to demonstrate on the balance of probabilities that the asbestos exposure caused the cancer. Without acceptance of the synergistic hypothesis as necessarily or probably always occurring, the asbestos exposure rose no higher than one of the possible risk factors, that is, one of the possible causes. In *Bonnington Castings* , the disease was caused by the dust that Mr Wardlaw had inhaled, a not negligible proportion of which was caused by the employer's negligence. Here, the cancer was caused by asbestos, and the evidence, if accepted, was that the cancer was caused by a process that involved the retained fibres from all three periods up to the point of the tumour onset, each exposure contributing to a material degree.

51. Nothing in *Merck Sharp & Dohme* should be taken as contrary to this. There the Full Federal Court rejected the proposition that increased risk was sufficient for a causal connection. To

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the extent that material contribution was discussed it was in this context of increased risk - such as by McHugh J in *Chappel v Hart* [1998] HCA 55; 195 CLR 232 at 244-245 [27] , see *Merck Sharp & Dohme* at 168 [96]. There may be a number of places where the requirement of a causal connection was expressed in terms of the causal requirement of the satisfaction of the but for test eg 169 [99]. The case was not directed to the nature of "material contribution" in the manner disclosed either in *Bonnington Castings* or in the evidence here. The question in *Merck Sharp & Dohme* was whether, beyond heightened risk, it had been shown that the drug had any factual connection with the condition suffered: the same threshold issue as in *Amaca v Ellis* .

52. In any event, even if it laid down an exclusive requirement of a but for test, it would not bind this Court in the face of *March v Stramare* , *Amaca v Booth* and *Strong v Woolworths* .

53. For these reasons and the reasons given by Basten JA, I agree with the orders proposed by Basten JA.

54. **BASTEN JA**:

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55. Mr David Sim died on 6 July 2009. Having worked for many years with asbestos, he contracted asbestosis and subsequently lung cancer. Between 1964 and approximately 1979 Mr Sim was employed by various employers to spray asbestos at commercial and construction sites. For all but a few months of that period, he had four employers. The three appellants in this Court represent the employers responsible for approximately 97% of Mr Sim's total known exposure to asbestos dust.

(1) Background

56. The present proceedings were brought in the Dust Diseases Tribunal by Mr Sim's widow, Lorraine Fay Sim, as the legal personal representative of his estate (she was referred to below as "the plaintiff"). The Tribunal (Curtis DCJ) found in her favour and awarded damages against each of the three defendants for the asbestosis suffered by the deceased in amounts totalling a little over $100,000. The Tribunal also found that the three defendants were jointly and severally liable in respect of Mr Sim's lung cancer, damages being assessed in the sum of $317,561.85. It was the liability in respect of lung cancer which was the subject of dispute in this Court.

57. The first appeal was brought by Allianz Australia Ltd ("Allianz") as the insurer of Mr Sim's first employer, Australian Asbestos Contracting Pty Ltd ("Australian Asbestos"). Allianz was sued pursuant to the statutory provision in s 6(4) of the *Law Reform (Miscellaneous Provisions) Act 1946* (NSW) . Australian Asbestos employed Mr Sim for approximately two years between 1964 and 1966.

58. The second appeal was brought by the WorkCover Authority of NSW ("WorkCover") which incurred liability on behalf of the defunct insurer of the second and third employers, namely Asbestospray Fireproofing and Insulation (NSW) Pty Ltd ("Asbestospray Fireproofing") and the successor to that business, Asbestospray Corporation Australasia Pty Ltd (Asbestospray Corporation"). In respect of the former (second employer), WorkCover raised a separate issue as to its liability pursuant to s 151AB of the *Workers Compensation Act 1987* (NSW) . WorkCover also challenged the Tribunal's order that it pay the claimant's costs on an indemnity basis from 14 May 2010. In addition, WorkCover brought proceedings by way of amended summons seeking judicial review of the judgment of the Tribunal. The summons was not pursued and was dismissed by consent at the commencement of the hearing in this Court: Tcpt, 15/11/11, p 2(5).

59. The fourth employer, Bells Thermalag & Industrial Services Pty Ltd, now known as Wallaby Grip (BAE) Pty Ltd ("Wallaby Grip") relied upon the same grounds as Allianz and adopted the submissions made on behalf of Allianz.

60. The manner in which the case on causation was articulated was addressed, in oral argument, by reference to the following summary of contentions found in the written submissions for Allianz:

> "13.1 The doctors' opinions (or assertions) to the effect that exposure to asbestos in AA's [Australian Asbestos] employment was a cause of Mr Sim's lung cancer were inadmissible. Properly analysed, they involved the application of the doctors' own notions of causation rather than the application of relevant scientific or medical study,

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training or experience. They also lacked a rational exposition of how the assumptions they made led, by the application of relevant expertise, to the conclusions they asserted. They were thus inadmissible under s 79 .

13.2 Properly understood, the evidence as to causation (including the doctors' opinions, if admissible) established no more than that exposure to asbestos in AA's employment had increased the risk of Mr Sim contracting lung cancer. The evidence did not establish that 'all exposure is causative' and did not establish that the lung cancer in fact contracted by Mr Sim was, on the probabilities, caused by exposure in AA's employment.

13.3 Furthermore, the relevant question was whether AA's *negligence* was a cause of Mr Sim's lung cancer. It was clear that Mr Sim would have been exposed to at least some asbestos in AA's employment even if AA had taken all proper precautions. His Honour should have made findings as to the excess, negligent, exposure by AA, and considered the question of causation by reference to that excess exposure, but he did not do so."

61. The appellants treated Allianz as the primary proponent of their cases. Ground 1 in Allianz' amended notice of appeal was replicated in the other notices of appeal and will be referred to as the "common ground". WorkCover raised four additional grounds which will be addressed separately. It is convenient to start with the elements of the common ground, which in their amended form were as follows:

"1. As to the finding (J[210]) that the inhalation of asbestos fibres by the Respondent (**M r Sim**) when in the employ of Australian Asbestos Contracting Pty Ltd, in respect of which entity the Allianz Australia Limited has been sued pursuant to s 6(4) of the *Law Reform (Miscellaneous Provisions) Act* 1946 (herein, **the Appellant**) had materially contributed to the cause of Mr Sim's lung cancer:

(a) the trial judge erred in admitting into evidence the material referred to in the document annexed hereto and marked with the letter "A" headed "First Appellant's Summary Table - Objections. Revised version: 16 November 2011", such being inadmissible under s 79 of the *Evidence Act* 1995 (NSW) .

(b) deleted.

(c) the trial judge erred in point of law in drawing an adverse inference from the Appellant's failure to call expert medical evidence expressing a conclusion as to the causation of Mr Sim's lung cancer (J[190]- [192] and J[209]);

(d) the trial judge erred in point of law in so finding in circumstances where the evidence (to the extent admissible) established no more than that such exposure had increased Mr Sim's risk of contracting lung cancer;

(e) having made a finding of excessive exposure by the Appellant, the trial judge erred in point of law in failing to make necessary findings as to the degree of additional asbestos exposure, if any, attributable to the negligence of the Appellant; and

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(f) the trial judge erred in point of law in so finding as it was not reasonably open on the admissible evidence that Mr Sim would not have contracted lung cancer but for the negligence of the Appellant."

62. Logically, the question of admissibility (ground 1(a)) might have been addressed first; however, by arrangement between counsel, senior counsel for Wallaby Grip commenced the oral argument by addressing the issue identified at par 13.2, set out at [60] above, reflecting ground 1(d). That contention was itself inaptly identified: the question was not what the evidence did or did not establish, but rather whether there was any evidence capable of supporting the relevant findings.

(2) Nature of appeal

63. The last point flows from the statutory conferral of a right of appeal to this Court from a decision of the Tribunal "in point of law or on a question as to the admission or rejection of evidence": *Dust Diseases Tribunal Act 1989* (NSW) ("the *Tribunal Act*"), s 32(1) . In an earlier decision dealing with s 32 of the *Tribunal Act* this Court accepted that, to the extent that s 32 permitted a challenge to the admission of evidence, it was not restricted to identification of legal error, but engaged the powers of the Court on a rehearing, pursuant to s 75A of the *Supreme Court Act 1970* (NSW) : *Amaba Pty Ltd (Under NSW Administered Winding Up) v Booth; Amaca Pty Ltd (Under NSW Administered Winding Up) v Booth* [2010] NSWCA 344; (2011) Aust Torts Rep ¶82-079 at [20] . The issue was not debated in these proceedings. However, it may be that little turns on the proper characterisation because the question of admissibility, in the present case, involves no findings in respect of facts but rather an assessment of whether the expert evidence, admitted by way of exception to the opinion rule, pursuant to s 79 of the *Evidence Act 1995* (NSW) , was capable of supporting the findings made. That involved an assessment of whether, if it were accepted, it could rationally affect the assessment of the probability of the existence of a fact in issue: *Evidence Act* , s 55 . Thus, the real difference between the admissibility question and the substantive grounds of appeal was temporal: in respect of admissibility, the question was whether the Tribunal erred in admitting the evidence by an assessment of the evidence in its own terms, without reference to the later oral evidence and assuming that, if the written reports had been rejected, oral evidence would not have been permitted.

64. As a practical matter, evidence is sometimes admitted subject to the party adducing it satisfying the Court as to its relevance, in circumstances where relevance may depend upon the existence or absence of other evidence or on a more refined understanding of the issues than is possible at the beginning of the trial. That raised a question with respect to the appeal, namely whether the question of admissibility was truly to be determined on a rehearing without regard to the oral evidence of the respective witnesses. It appeared from the answer given by senior counsel for Wallaby Grip to a question from the President at the commencement of argument that such a staged consideration would be artificial and that it was sufficient to address the issues on appeal on the basis of the whole of the evidence: Tcpt, 15/11/11, p 2(35-45) and p 3(1-35).

65. The matter was raised again by the President with senior counsel for Allianz, in the following terms (Tcpt, p 37(15)):

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> "... do you approach it as it were sequentially, that is, that the paper as it stood at the point prior to any oral evidence was inadmissible and that must be examined at that point in time and move forward with each question or are you prepared as it were to embrace the totality of the material that fell out and see whether material in the oral evidence in effect cures what might otherwise have been at an earlier point ... an evidential difficulty and an objection you should have won[?]"

66. Senior counsel responded:

> "We say first that this evidence was inadmissible in the form in which it was first presented before the Court, namely, in written form and his Honour should have excluded it at that point and we do say that's an important matter for the Court because of its obvious potential to affect future trials. So that's the first point we make but we accept ... that what in fact happened was that there was further evidence on the subject and we accept that at a practical level it's necessary to have regard to that material.
>
> Now, that material we say can actually in significant respects [support] the objections we were taking so it's rather like a voir dire in one sense, that we would say one can have regard to that material and see that ... objections which we have taken on the basis that matters that weren't demonstrated in fact later fell out to be demonstrated to the contrary, if I can put it that way."

67. Counsel was asked whether it might be appropriate to look at all of the evidence to see whether, by the end of the trial, any error in admitting the written reports of the experts had been overcome by subsequent evidence so that the written reports might originally have properly been admitted conditionally, rather than unconditionally, but that the condition had been fulfilled. Counsel did not so much demur to those propositions as seek to ensure that no point of waiver was being taken against him. Those propositions should be accepted.

68. There may be other ways in which the question of process might be addressed. If the Tribunal had erred in admitting the evidence as originally presented and if the appellants had challenged that decision on an interlocutory basis, even accepting that without such evidence the claimant would fail, there might be a separate question as to whether the claim should be dismissed or there should be a retrial. If there were reasonable grounds for thinking that the claimant might be able to make good any omission in the expert evidence, the Court might decline to interfere on the basis that the decision to admit the expert reports had not occasioned any "substantial wrong or miscarriage", in circumstances where the relevant omission could be rectified: Uniform Civil Procedure Rules 2005 (NSW) (" UCPR "), r 51.53 (1) . If that were a legitimate approach, it must follow that, subject to questions of procedural fairness, the admission of evidence, not challenged on appeal at the time, should be treated as an immaterial error if, as the oral evidence progressed, any omission in the original report was remedied. In that situation, it would be appropriate (and indeed necessary) for the appellate court to consider the question of admissibility by reference to all of the evidence tendered or adduced at trial. Nor was there any complaint of procedural unfairness in the present case, which might otherwise have rendered that course inappropriate.

69. However, as will appear below, little turned on the oral evidence; the issues were adequately identified in, and the findings supported by, the written reports.

(3) The medical evidence

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(a) background

70. It is convenient to address the substance of the appellants' arguments before considering the challenge to the admissibility of the evidence and the alleged error of law.

71. The periods of Mr Sim's employment by the appellants commenced in 1964 and, relevantly in terms of tortious exposure to asbestos dust, terminated on 31 December 1979: third amended statement of claim, pars 4, 6, 6A and 8. On 7 May 1996 Mr Sim was diagnosed by Dr Deborah Yates, a consultant thoracic physician, as suffering from early asbestosis: judgment at [212]. In November 2006, a CT scan confirmed diffuse pleural thickening and asbestosis: at [214] . His condition continued to worsen until January 2009 when Dr Yates diagnosed lung cancer with multiple metastases in the spine: at [217]. He was admitted to hospital on 7 June 2009, where he remained until his death on 6 July 2009: at [218] . Retrospectively, Dr Yates formed the view that his lung cancer formed in early 2008: at [220].

(b) findings with respect to causation

72. The issues with respect to causation were succinctly identified by the trial judge in the following passage:

> "[165] There can be no real dispute that the several employments with each of Australian Asbestos, Asbestospray Fireproofing, Asbestospray Australasia, and Bells caused the severable disease of asbestosis.
>
> [166] Nor can there be any real dispute that the totality of the several periods of asbestos employment caused Mr Sim's cancer. The presence of asbestosis increases the risk of contracting lung cancer by a factor of between 5 and 10 over the background risk. Because Mr Sim stopped smoking 30 years before he died, every medical expert called in the plaintiff's case dismissed smoking as a cause of the cancer.
>
> [167] Nevertheless, the plaintiff cannot succeed against any defendant merely by proving that the aggregate exposure to asbestos caused the cancer (*Amaca Pty Ltd v Ellis* (2010) 240 CLR 111), and each defendant, calling no medical evidence, asserts that the plaintiff has failed to prove that the employment for which it is responsible materially contributed to the contraction of the cancer."

73. After referring briefly to the evidence of the three main witnesses called by the plaintiff, Professor Douglas Henderson (Professor of Pathology at Flinders University), Dr David Bryant (Associate Professor of Medicine at UNSW) and Dr Deborah Yates, the judge identified the principal contentions put forward on behalf of the defendants (now appellants) in the following terms at [183]:

> "(a) Because it is impossible on the current state of scientific knowledge to fully identify the sequence of mutagenic events that caused a lung cancer in an individual, it is impossible to assert that all retained fibres contributed to the particular biochemical reaction which led ultimately to the development of the cancer.
>
> (b) In this circumstance the only rational basis for concluding that asbestos fibre for which a defendant is liable caused or contributed to those events, is ... the laws of

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mathematical probability based on whether the attributable risk fraction for the exposure in question exceed[ed] 50 per cent."

74. The first contention, as summarised, involved a degree of circularity which was unhelpful. The real question was whether enough was known for a scientist with expertise in the relevant discipline to express the view identified in the second part of the proposition. In substance, as explained in oral argument in this Court by senior counsel for Wallaby Grip (Tcpt, p 17(10)):

> "Our point is this: if one's talking about increasing the possibility of a set of mutations, which is what Professor Henderson is talking about, the question remains of any one defendant responsible for some but not all of the aggregate exposure which [on?] the premise is more likely [than] not as an aggregate cause for lung cancer, one still has to ask the question 'how can you say of any part of that component that it did anything other than increase a risk?'. And then if the risk, if the other exposures apart from the exposure by the particular defendant in question, is more than sufficient to explain the lung cancer - which it is in this case - how can you say on the balance of probabilities as opposed to a possibility, that the first of the defendants in question is liable ... Or the second or the third. And that's why it's only in cases where you can say each was necessary though not sufficient. And that means you need to know something about what is sufficient to have caused it and you obviously need to pay close regard to the component of exposure for which [the] defendant is negligently liable - I stress it's not the whole of the exposure necessarily - towards the end of the period it will be. But not at the beginning."

75. The trial judge addressed the argument that the mutagenic process was "not known" in the following terms:

> "[186] As I understand Professor Henderson's evidence, a cell mutates, not because of mechanical impingement upon the cell by an identifiable asbestos fibre, but because of its exposure to free radicals, toxic to DNA. These radicals are released by the response of the immune system, not only to the presence of any particular identifiable fibre or fibres, but to the presence of all retained fibres. Because the ultimate mutation occurs shortly before the development of the cancer, all retained fibres are causally implicated in a cascading chain of mutations.
>
> [187] His opinion is encapsulated in an exchange in cross-examination, where Mr Parker sought to isolate a mutant ancestral cell caused by a particular asbestos exposure as the single cause of the cancer.
>
> [188] Professor Henderson responded by pointing out that, even if it were accepted that an identifiable ancestral cell was generated in a discrete period of employment, further necessary mutations of that cell are modulated by the totality of reactive oxygen and reactive nitrogen species released in the immune system's continuing response to the presence of all retained asbestos fibres, inhaled both before and after the generation of that particular ancestral cell."

76. The trial judge also dealt with the second proposition, namely that the only way in which the plaintiff could succeed was by demonstrating, by reference to a calculation of relative risk,

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that the exposure by one defendant was, on the balance of probabilities, likely to have been the cause of the cancer, when compared with all other exposures. He noted that the submission had been explained in the following manner - at [197]:

> "What this means is that if Mr Sim had never had any asbestos exposure after his employment with Australian Asbestos, a probability analysis would overwhelmingly attribute his cancer to background. The plaintiff cannot be in a better position because, quite independently of his Australian Asbestos exposure, Mr Sim was later exposed to asbestos while employed by other employers."

77. The judge rejected that proposition for three identified reasons, which, in part, were explained as follows:

> "[199] The first is that epidemiological studies are concerned with risk not cause. Once the cancer has developed medical science may attribute that cancer to a causal factor notwithstanding that, prospectively, that factor was not seen as a probable cause.
>
> ...
>
> [203] The second reason is that epidemiological evidence cannot prove that exposure to a toxic agent at a certain concentration *did not* cause a corresponding disease in an instant case.
> ...
>
> [206] A third reason for rejecting the submission is that the incidence of lung cancer rises with increasing doses of asbestos fibre. That circumstance gives rise to an inference that all contributions are causative. Mr Parker cannot advance any scientific basis upon which a particular period of exposure may be exculpated."

78. The appellants take issue with each aspect of the judge's reasoning. One point, however, may be disposed of at this preliminary stage. A complaint that the passage at [203] reversed the onus of proof should not be accepted. The judge was addressing the manner in which the epidemiological evidence was relied on by the appellants to rebut an inference of causation which otherwise arose. Either the inference arose on the plaintiff's case (as his Honour held) or it did not; if it did not, the epidemiology was irrelevant; if it did, the epidemiology might in principle provide a basis for rebutting the inference. I do not read his remarks at [203] as placing a burden on the defendants: rather, he was addressing an argument which they had presented. He disposed of the argument on its merit, in terms to which it will be necessary to return.

(c) thrust of the plaintiff's case

79. In order to address further the thrust of the case for the plaintiff, it is necessary to refer to key passages in the evidence. To know whether the judge's reliance on the evidence was sound, it is sufficient to address the evidence of Professor Henderson: the support obtained from Associate Professor Bryant and Dr Yates do not affect the analysis.

80. Professor Henderson's first report, dated 4 June 2009, was not the subject of any challenge on the appeal. It was directed to the general question as to the correct diagnosis of Mr Sim's condition and, if satisfied that he suffered from a carcinoma of the lung, whether his exposure

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to asbestos dust and fibre "from 1964 to 2002" made a material contribution to the development of the carcinoma. Professor Henderson accepted Dr Yates' diagnosis of asbestosis; he also accepted that the primary site for Mr Sim's metastatic adenocarcinoma was the lung and concluded that "at a high order of confidence" Mr Sim's "probable/presumptive lung cancer is attributable to his total cumulative and heavy occupational exposure to asbestos (with clinical asbestosis), by way of a significant causal contribution": Report, p 17.

81. The focus of the first report was the identification of the primary site of the carcinoma. The diagnosis of an adenocarcinoma derived from a pathology report for a core biopsy taken from a mass lesion in the spine at L2. The report identified "diffuse invasion of bone marrow by metastatic adeno carcinoma where the tumour cells formed groups with a desmoplastic stroma": Report, p 6. The initial question was to determine the primary site of the carcinoma as the lung. That being determined, the second question, answered in the affirmative, was whether "Mr Sim's cumulative asbestos exposure resulting in clinical asbestosis made a substantial causal contribution" to the carcinoma. The opinions set out in Professor Henderson's first report allowed the trial judge to conclude that the cumulative effect of the exposure to asbestos was established as the cause of the lung cancer. That conclusion was not challenged on appeal. The remaining question was whether each of the four employers was implicated or whether all could escape because it could not be established that the tortious exposure during employment with each was a necessary causal element in the chain of events.

82. Professor Henderson provided a supplementary report dated 19 April 2010. That report set out to answer a further question, namely whether each of Mr Sim's periods of employment caused or made a material contribution to the pathogenesis of asbestosis and carcinoma of the lung: Supplementary Report, p 2. Professor Henderson concluded that each of his employments "made a significant and substantial causal contribution to the development of each of his asbestosis and his lung cancer": Supplementary Report, p 5. Referring to Mr Sim's diagnosis of asbestosis, Professor Henderson stated (p 4):

> "Characteristically, asbestosis is an outcome of substantial exposure to asbestos and carries a relative risk of lung cancer of 2-5, or more. The relationship between asbestos exposure and asbestosis is governed by a dose-response model, with evidence of a 'dose' threshold: the greater the exposure, the greater the likelihood of asbestosis and its severity. Asbestosis falls into the class of fibro-inflammatory disorders and once it has developed as such, its severity can be aggravated by continued exposure to asbestos (although it can also progress even after exposure has ceased). The details of Mr Sim's work (insulation work including the spraying of asbestos) as set out in his affidavit indicate that he sustained heavy cumulative exposure to asbestos .... As an approximation only, one could estimate that his inhaled 'dose' of asbestos from each of those employments would have been proportional to the duration of each, and each would have [been] sufficient by itself for the induction of asbestosis."

83. Despite the objection to the whole of the report, no specific complaint was made about the passage set out above. Nor was there complaint about the following passage in relation to lung cancer:

> "It is known that asbestos can induce lung cancer, the causal relationship being one of a linear dose-response effect with no threshold. That is, the greater the inhaled 'dose' the greater the risk of lung cancer and the causal contribution by asbestos for lung cancer

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> induction. Lung cancer differs qualitatively from asbestosis and falls into the class of malignant neoplastic diseases. It is considered to be a 'singular' injury, in that once it has come into being, further exposure to its causal factors (eg, cigarette smoke; asbestos) cannot significantly worsen the injury (cancer) and, conversely, withdrawal of the causal factors cannot reverse the cancer."

84. None of this material was challenged: the heart of the objection was to the conclusion identified in the following passage (p 5):

> "Because the likelihood and pathogenesis of lung cancer are governed by a dose-response relationship, it also follows on a probabilistic basis that each of Mr Sim's asbestos exposures ... made a significant and substantial causal contribution to the development of his lung cancer."

85. The appellants objected to this passage on the basis that the process of reasoning was not exposed. In fact, Professor Henderson identified the basis for that opinion by reference to a paper of which he was a co-author, published in *Pathology* in 2004, which formed part of the tender, with the second report. However, it is not necessary to refer to that article in this context: the reasoning underlying the opinions was more fully set out in Professor Henderson's third supplementary report, addressed below.

86. On 19 May 2010 Professor Henderson produced a second supplementary report. The principal difference in the question asked for the purpose of this report, as compared with the previous report, was to divide the period of employment with Asbestospray between the two separate companies, so that there was not one continuous period of employment from 1966 to 1975, as had been suggested in the earlier material supplied to Professor Henderson. His conclusions were the same as before, namely that exposure during each period of employment (now four rather than three) made a significant and substantial causal contribution to the development of Mr Sim's asbestosis and lung cancer.

87. On 31 August 2010 Professor Henderson provided a third supplementary report. The primary purpose of this report was to comment on a report obtained by Allianz from Professor Geoffrey Berry on 24 August 2010. However, in undertaking that review, Professor Henderson expanded significantly on the process of reasoning upon which he had relied in reaching his earlier conclusions. Accordingly, to the extent that objection to the first and second supplementary reports was based on an absence of reasoning, it is sufficient to address that question by reference to the third supplementary report. (Professor Berry's report was not tendered by the appellants and his opinions, except to the extent they were revealed by Professor Henderson, did not become part of the evidence.)

88. Two themes may be identified in Professor Henderson's response to Professor Berry. The first was the importance of the distinction between epidemiology and aetiology. Epidemiology, at least in the current context, establishes a "relative risk" or "attributable fraction" in respect of a particular carcinogen and an identified disease. As Professor Henderson explained (Third Supplementary Report, p 5) relative risk "deals with the actual number of occurrences of the disease or injury in question for an exposed group, *versus* the actual number of cases of the same disease in those unexposed to the factor(s) under investigation". Its weakness as a predictor of causation in a particular case derives from the application of a result derived from a population to an individual. By contrast, Professor Henderson explained (p 4):

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> "In fact, assessment of causation of disease involves a wider exercise than epidemiology alone, and usually includes knowledge/concepts of the character of the disease in question relative to other diseases (bi-plausibility), and experimental data, as well as clinical/pathology observations, in addition to epidemiology and statistics; as such, this exercise involves a biomedical exercise whereby all relevant data are integrated into a conceptual/actual understanding (although when the evidence is strong enough, causation can be assigned in the absence of some or much knowledge of causal mechanisms - a so-called 'black box' approach).
>
> This multifactorial approach to causation was set forth explicitly and in a commonsense approach by the late Sir Austin Bradford-Hill, in a set of multiple criteria that became known as 'The Bradford-Hill Criteria'."

89. The second focus of Professor Henderson's comments was that "lung cancer is considered to develop by way of a multi-stage model of carcinogenesis, analogous to that which applies to the development of mesothelioma when caused by asbestos". Professor Henderson continued (p 8):

> "Current understanding of the pathobiological mechanisms for lung cancer and mesothelioma development indicates that epithelial/mesothelial cells are being initiated, initiated cells promoted, and altered cells proliferating at different times, apparently and at least in part as a result of free radical generation, with oncogenes and tumour suppressor genes being activated and inactivated. At some stage, the proliferative airway /mesothelial cells are thought to become resistant to apoptosis. At the same time, fibres are cleared at different rates and, if exposure is continuing, they continue to be deposited in the lung. ...
>
> All these processes at a cellular level are probabilistic: i.e., the probability of fibre/cell interaction (or, more accurately, fibres/cells interactions) depends on the number of fibres and the number of cells involved at any point in time and space. Hence, simplistically, the greater the number of asbestos fibres, the more free radicals (generated from the fibres or their interactions with cells such as macrophages) and the greater probability of initiated, promoted or proliferative cells at any given time point.
>
> From a current understanding of the multistage model for cancer induction by asbestos, it follows that each of all exposures to asbestos - recalled or unrecognised - contributes incrementally and cumulatively to the singular disease outcome lung cancer or mesothelioma (provided that the latency interval between any one exposure and the subsequent diagnosis of the cancer is not too short for a carcinogenic effect)."

90. The so-called "latency interval" is a period of at least 10 years. Thus, exposure to asbestos within 10 years of diagnosis of a carcinoma is treated as causally immaterial. In the present case, the carcinoma was diagnosed as occurring in May 2008, more than 30 years after the last relevant exposure. The evidence did not suggest there was an upper limit to the period during which asbestos exposure could contribute to the development of lung cancer.

91. There is one other passage in the report to which reference is necessary, as it was relied upon to illustrate what was said to be an invalid leap from assessment of risk to causation. Professor Henderson's opinion is introduced by the following passage (p 11):

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> "On page 5, Prof Berry responds to the two questions, presumably posed by the instructing solicitors. One of the questions asks whether it is possible to determine scientifically whether Mr Sim's lung cancer (in his individual case) was caused by his exposure to asbestos dust and fibre only whilst employed by Australian Asbestos Pty Ltd between 1964 and 1966? I note that he comments:
>
> > 'In the absence of quantitative exposure information it would be invalid to conclude that the lung cancer was caused only by the exposure whilst employed by Australian Asbestos. There were three other periods of exposure that would have made a contribution to the excess in risk, and there is no scientific basis for saying that only one of these four periods of employment [caused] the lung cancer.'"

(Both the underlining and the inserted words occur in Professor Henderson's document.)

92. Professor Henderson noted that Professor Berry had treated exposure as steady in each year of employment and had therefore suggested that, based on the duration of employment, the contribution to total risk contributed by employment at Australian Asbestos would have been "about 15% 'of the total excess risk'". Professor Henderson expressed agreement with Professor Berry in respect of the material set out and stated, "I would go further, in assuming that one could state the *proportional causal contribution* towards the development of Mr Sim's lung cancer from his Australian Asbestos exposure was roughly about 15%, as opposed to his *risk* of lung cancer, because that *risk* was *translated into an actual outcome as a lung cancer*, ie, the fractional causal contribution from each of the four exposures/employments is roughly proportional to the duration of each in percentage terms". That passage was one of a number relied upon by the appellants in support of their contention that Professor Henderson (and the other experts) had simply restated a legitimate assessment of increase in risk as an entirely speculative claim of causal contribution.

93. For reasons which are identified below, the criticism was not made good. However, before addressing that analysis, it is convenient to refer to the oral evidence of Professor Henderson.

94. Professor Henderson provided some further explanations of his opinions in his evidence in chief. He had stated in his third supplementary report that it was "impossible to fractionate or partition various stages in a multi-stage process into the exposure from one particular employer as opposed to another": at p 7. He was asked to explain why he considered that impossible and stated (Tcpt, 07/09/10, p 240):

> "The simple fact is that if one has say four separate asbestos exposures, one cannot fractionate a causal effect or even one stage in the multi-step progression to an indivisible outcome like lung cancer, and attribute that stage to one of those four exposures. The point is, and a point that I think is overlooked in the quoted passage of text [from Professor Berry] ... is that amphibole fibres in particular which are known to be the most carcinogenic forms of asbestos, are characterised by bio-persistence, that is once deposited in tissues, they will remain there for year after year after year, and they will interact with cells such as macrophages and they will generate toxic chemicals known as reactive oxygen and reactive nitrogen species, which are known then to be injurious to the DNA of various cells, for example, airway epithelial cells in the case of lung cancer. So that when you have multiple exposures, one cannot say that one stage is

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related to exposure one and that that exposure or its effects then cease and then one goes on to exposure two, because the fibres are persistent to the tissue year after year and as additional exposures occur, additional fibres are deposited in the tissues, so that there are more fibres interacting with more cells generating more toxic chemicals increasing the toxicity to the DNA of airway epithelial cells and increasing the probability of a set of mutations in what is a chain of necessary causal preliminary events on the pathway to lung cancer which is an indivisible and singular outcome."

95. With respect to the translation of risk into causative effect, he gave the following further evidence (p 241):

> "Q. ... Does the exposure in four separate periods, as in Mr Sim's case, to asbestos, with four separate employers - does each exposure constitute an obligate chain of events essential for the ultimate development of the cancer?
> A. Well, in the development of that particular cancer my answer is in the affirmative; but, yes, that each of those exposures will increase the likelihood that a cancer will develop but the fibres from the first exposure remain throughout the other periods of exposure and are added to by the fibres inhaled and deposited by the subsequent exposures, so that each has incrementally increased the likelihood that a lung cancer will develop, and in this case that likelihood has been translated into the actual development of the lung cancer.
>
> Q. When the lung cancer develops and the risk expressed as a likelihood has come home, is it possible then to exclude any of the four periods of exposure to asbestos with each of the employers in the explanation for the reality of the cancer?
> A. Well, the answer to that is no, one cannot exclude anyone and blame all of the others. The point is that all of them have contributed to the load inhaled and deposited asbestos fibres in lung tissue, or a fraction of those fibres have interacted with the cells to produce the toxic chemicals involved in producing mutations. Those mutations have increased in number as a result of the fact that asbestos fibres can interact with cells at multiple different points along the causal chain, leading up to the development of the lung cancer. ... In other words, one cannot point to one and blame it and exculpate the others; and equally, one can't point to one and exculpate it and then blame all of the other three exposures. The point is that all of them have contributed to the fibre burden in lung tissue, all have contributed to the risk which is then translated into the actual occurrence of the lung cancer."

96. Allianz characterised this evidence in the following terms in its written submissions in this Court:

> "59 In short, any exposure to fibres (other than numerically insignificant ones) increases the risk of a lung cancer eventuating. This is quite different from saying that if a lung cancer actually eventuates, every exposure actually caused that cancer (in the sense that without that exposure, the cancer would not have appeared).
>
> 60 How then did the experts (and his Honour) justify treating increased risk as equivalent to cause? To the extent that any reasoning at all was offered for this step, it seems to boil down to two arguments: (1) it was appropriate to do so because 'the risk

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came home'; and (2) the proposition that 'all exposure is causative' followed from the existence of a dose-response relationship (ie, the incidence of cancer increases with cumulative dose)."

(4) Analysis of appellants' criticisms

97. Although parts of Professor Henderson's evidence were expanded upon and explained in his oral evidence, as indicated above, the general thrust of his evidence did not vary in significant respects from that set out in his reports, and particularly the third supplementary report. Accordingly, it is convenient to analyse the substantive argument raised by the appellants, to the effect that the evidence failed to demonstrate a causal connection between the exposure to asbestos dust in each period of employment and the carcinoma which resulted from the exposures taken cumulatively and as a whole.

98. The first step in the argument is undoubtedly correct: increase in risk is not to be equated with factual causation. As noted by French CJ in *Amaca Pty Ltd (under NSW administered winding up) v Booth* [2011] HCA 53; 86 ALJR 172 at [41] :

> "Causation in tort is not established merely because the allegedly tortious act or omission increased a risk of injury. The risk of an occurrence and the cause of the occurrence are quite different things."

99. As his Honour further noted, assessment of the risk of an occurrence is "prospective in character": at [42]. Further, the reasoning continued at [43] :

> "An after-the-event inference of causal connection may be reached on the civil standard of proof, namely, balance of probabilities, notwithstanding that the statistical correlation between the first event and the second event indicated, prospectively, no more than a 'mere possibility' or 'real chance' that the second event would occur given the first event."

100. In *Amaca v Booth* , the question was whether Mr Booth had established that his malignant pleural mesothelioma was caused by his exposure to asbestos in working on brake linings over a period of 30 years, and involving two separate defendants. After noting the distinction between a predictive assessment of risk and a retrospective assessment of causation, French CJ stated at [43] :

> "The existence of an association or a positive statistical correlation between the occurrence of one event and the subsequent occurrence of another may be expressed as a possibility, which may be no greater than a 'real chance' that, if the first event occurs, the second event will also occur. The mere existence of such an association or correlation does not justify a statement, relevant to factual causation in law, that the first event 'creates' or 'gives rise to' or 'increases' the probability that the second event will occur. Such a statement contains an assumption that if the second event occurs it will have some causal connection to the first. However, if the association between two events is shown to have a causal explanation, then the conclusion may be open, if the second event should occur, that the first event has been at least a contributing cause of that occurrence."

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101. His Honour then referred to the nine factors identified by Sir Austin Bradford-Hill as matters having the character of circumstantial evidence of a casual relationship: at [44]. These factors had been referred to by Professor Henderson in *Amaca*, and in his report in the present case. Significantly however, the Chief Justice pointed to the distinction between a finding that conduct has increased the risk of injury and a mere statistical correlation between such conduct and such injuries. The former involves, inherently, a causal connection, which the latter need not: at [49]. His Honour concluded at [51] :

> "It is enough for present purposes to say that an inference of factual causation, as against both Amaca and Amaba, was open on the evidence before the primary judge. The cumulative effect mechanism involving all asbestos exposure in causal contribution to the ultimate development of a mesothelioma had been propounded and was accepted by his Honour. It depended upon an understanding of physiological mechanisms. It did not depend upon the epidemiology."

102. The joint judgment of Gummow, Hayne and Crennan JJ reached a similar conclusion. After noting the significance of the "single fibre" theory in the development of the law in England, their Honours noted the contrary view, based on multiple exposures, as explained by Professor Henderson in that case: at [79]. Subject to an attack not made in the present case, their Honours set out and accepted the plaintiff's argument in *Amaca v Booth* in the following terms at [83] :

> "Mr Booth developed his case in the following steps: (1) he had contracted mesothelioma; (2) the only known cause of that disease is exposure to asbestos; (3) the expert evidence at trial, accepted by the primary judge, was that: (a) exposure to asbestos contributes to the disease; and (b) the prospective risk of contracting the disease increases with the period of significant exposure; (4) Mr Booth had two periods of significant exposure; (5) it is more probable than not that each period of exposure made a material contribution to bodily processes which progressed to the development of the disease."

103. The remainder of their conclusions were directed to an alleged failure by the Tribunal to make proper allowance for the epidemiological evidence which the appellants presented; no similar issue arises in the present case, such evidence not having been adduced by the appellants. The appellants' submissions as to the supposed elision from risk to causation paid inadequate attention to the reasoning of Professor Henderson accepted by the trial judge. At no stage was the conclusion supported merely on the basis that the risk had eventuated.

104. Allianz sought to place reliance on the statements by Spigelman CJ in *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29; 49 NSWLR 262 at [118]-[120] . In that passage the Chief Justice was addressing an argument not put in the present case, based on an observation of McHugh J in *Chappel v Hart* [1998] HCA 55; 195 CLR 232 at [27] where his Honour asserted that:

> "Before the defendant will be held responsible for the plaintiff's injury, the plaintiff must prove that the defendant's conduct materially contributed to the plaintiff suffering that injury. ... If a wrongful act or omission results in an increased risk of injury to the

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> 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."

105. There is no need in the present case to explore the scope of McHugh J's remarks: they were made in the course of a dissenting judgment; they related to a case where the breach of duty was the failure to give a warning as to precisely that risk which eventuated, and they occurred in a passage addressing the proposition that the "but for" test of causation is "often not enough to establish a causal connection for legal purposes": at [26]. The passage in the judgment of Spigelman CJ in *Seltsam* related to the relevance of epidemiological evidence which did not establish a relative risk in excess of two, and thus causation on the balance of probabilities.

106. This reasoning was not relied upon in Professor Henderson's evidence which included the following proposition (third supplementary report, p 6):

> "These comments concerning RR extend to the concept of *attributable fraction* (AF) amongst the exposed, because this is based on the RR derived as an *average*. Rockhill et al have emphasised that AF cannot be equated automatically to the probability of causation in an *individual* case (see Henderson et al)."

107. Further, the nature of the epidemiology was quite different in the present case, to the extent that it appeared at all in the evidence. Thus, Professor Henderson estimated Mr Sim's "relative risk of lung cancer from his asbestos exposure sufficient to cause clinical asbestosis, at about 5.0": third supplementary report, p 14.

108. The appellants' second submission was that the trial judge reached a conclusion that all exposure is causative based on the existence of a dose-response relationship, that is a correlation between exposure to asbestos and the incidence of lung cancer. The passage in the reasoning of the Tribunal said to support the inference was but one reason given to reject the proposition identified at [197] of the Tribunal's reasons and set out at [76] above: see third reason at [206] of the Tribunal's reasons, set out at [77] above.

109. More importantly, the complaint both overstates and trivialises the evidence. Neither Professor Henderson nor the trial judge treated all exposure as causative in a practical sense. Each dismissed as irrelevant exposures to asbestos characterised as insignificant. Professor Henderson noted that Mr Sim's condition involved "pathologically advanced asbestosis", being a condition which had reached "stage 4 out of four stages of" the disease: Third Supplementary Report, p 13. He estimated Mr Sim's cumulative asbestos exposure "almost certainly to have been greater than 50 fibres/mL-years - probably substantially in excess of this figure and probably more than a 100 fibres/mL-years": report, p 14. Further, the exposure took place over a period of some 14-15 years. The shortest period of any single employer was the two years during which Mr Sim was employed by Allianz' insured. Significant exposure over a two year period did not require consideration of whether "all" exposure was causative.

110. Nor was it suggested that all exposure contributed to the "chemical soup" described by Professor Henderson as the consequence of inhaling asbestos fibre: Tcpt, 7/9/10, 272(10) and (25). Further, as Professor Henderson acknowledged (Third Supplementary Report, p 9):

> "I agree with Prof Berry that not all asbestos fibres initially inhaled actually participate directly in lung cancer induction. When airborne asbestos fibres are inhaled, a

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> proportion will be filtered out by the upper nasal passages (although humans frequently convert from nose breathing to mouth breathing when exerting themselves). A proportion of the fibres which successfully bypass the upper respiratory passages will be deposited in the bronchial walls, and amphibole fibres such as crocidolite and/or amosite will tend to persist, although some translocate away from bronchial walls and others will reach the pleura. Nonetheless, those that remain in the airway structures and eventually induce the lung cancer represent a proportional fraction of those fibres first inhaled, and the more fibres that are deposited (when there were multiple exposures over time), the greater the probability of induction of chemical messengers such as reactive oxygen species *and the amounts of such reactive chemicals*, which can damage the DNA of the cells."

111. In short, Professor Henderson provided a biochemical explanation for the proposition that substantial periods of exposure, extending over two years or more, all materially contributed to the induction of the cancer. The appellants complained on many occasions of the reliance by the plaintiff on the "ipse dixit" of the experts, meaning unreasoned statements of conclusions. Much the same could have been said of those submissions; while the experts spoke within areas of expertise, the appellants' submissions resorted to tendentious dismissal of the expert evidence, mischaracterised for the purpose.

112. For these reasons, common ground 1(d), asserting error of law in finding material contribution against each defendant when the evidence established "no more than that such exposure had increased Mr Sim's risk of contracting lung cancer" must be rejected.

(5) Admissibility of evidence

113. The appellants asserted that evidence as to causation of Mr Sim's carcinoma was inadmissible, despite the fact that Professor Henderson was a distinguished pathologist with an international reputation and long-standing interest in asbestos caused diseases and that Drs Bryant and Yates were clinicians, eminent in the same area. Each undoubtedly had "specialised knowledge based on [their] training, study or experience" for the purposes of s 79(1) of the *Evidence Act* . So much was accepted: what was disputed was that their opinions in respect of causation were "wholly or substantially based on that knowledge", again within the language of s 79(1) . Just how the appellant sought to make good that challenge requires careful identification.

114. The first proposition raised by the written submissions was that their evidence fell within the class of evidence rejected by the High Court in *Clark v Ryan* [1960] HCA 42; 103 CLR 486. Accepting for present purposes that the terms of s 79 of the *Evidence Act* reflect the general law principles as applicable in 1960, the lessons to be drawn from that case for present purposes are obscure. No relevant principle was derived from the case and the facts are far removed from the present dispute. *Clark v Ryan* involved the tender of evidence from a person with "engineering" experience, who had no specialist qualifications of significance, but had investigated road accidents including those involving semi-trailers. He sought to explain how a semi-trailer had jack-knifed in taking a bend on the Pacific Highway south of Gosford, colliding with the plaintiff's vehicle, which was entirely on the correct side of the road. As explained by Dixon CJ at 490-491 :

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> "If it had been desired to prove how in fact semi-trailers of the kind driven by the defendant Clark do in practice behave, perhaps a witness or witnesses experienced in their actual use might have given admissible evidence, not of opinion, but of the fact. But Mr Foster Joy did not possess that experience. If it had been desired to give technical evidence of the physics involved and of any relevant opinions deduced therefrom, possibly that might have been done by a qualified witness although one may doubt how intelligible to the jury the evidence would have been and what useful purpose it would have served. But it certainly does not appear that Mr Foster Joy was qualified to give such testimony and in fact he did not essay to do so. What in truth occurred was to use the witness to argue the plaintiff's case and present it more vividly and cogently before the jury."

115. To say, as Allianz did, that "the opinions on individual causation offered by the plaintiff's witnesses in the present case are indistinguishable in principle from the opinion offered by Mr Joy in *Clark v Ryan* " is so vapid a proposition as to cast doubt upon the ground of appeal generally, as it formed the primary basis of the written submissions: see par 50. However, there were three attempted refinements on the submission which need to be addressed.

116. First, it was said that because the "general theory as to the aetiology of lung cancer was not in dispute" there was, in effect, nothing left about which an expert could helpfully opine. There are a number of responses to that proposition. First, as has been explained above, the "cumulative theory" of causation was very much in issue and, it appears, remains so in the appellants' view. Thus, in the same written submissions, although in a passage not pursued on the present appeal, Allianz sought to argue that the trial judge was in error in seeking to distinguish the approach of the House of Lords in *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32. Further, the submissions sought to distinguish the factual findings accepted as available to the Tribunal in *Amaba Pty Ltd v Booth* in this Court.

117. A second response is that by purportedly accepting Professor Henderson's evidence as to the aetiology of lung cancer, but not accepting his evidence as to causation in the individual case, the appellants were assuming the correctness of their conclusion, namely that the inferences to be drawn as to causation in the individual case were simply not available on any logical basis. For the reasons already given, that assumption must be rejected.

118. A third response is that the complaint has echoes of an "ultimate issue" challenge. Conscious of the terms of s 80, which provides that evidence of an opinion is not inadmissible only because it is about a fact in issue or an ultimate issue, counsel denied the suggestion. Nevertheless, if it is appropriate at all to draw an inference as to causation in the individual case, the third supplementary report of Professor Henderson demonstrates with clarity why it is that an expert is entitled to take that step. Indeed, although it relied upon no epidemiological evidence, Allianz sought to argue a proposition which itself required scientific justification, namely that only epidemiology could answer the plaintiff's need to establish individual causation. If that required a scientific justification, the answer (provided by Professor Henderson) also required scientific expertise.

119. The second basis on which the appellants sought to bolster their challenge was that "the drawing of inferences of individual causation was [not] the subject of any organised field of scientific or medical study": written submissions, par 52. That extraordinary proposition was,

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in substance, based on the view accepted by one of the clinicians that treatment of lung cancer was unaffected by its cause; that may well be so, but the point is trivial. The submission rejects out of hand all aspects of preventative medicine and public health, the latter, presumably, because it is not concerned with how a particular individual might acquire a disease or other disability. No reference was made to Professor Henderson's evidence, for example, as to the importance of knowing why only 10-15% of cigarette smokers develop lung cancer and only 10-15% of persons heavily exposed to asbestos develop mesothelioma. It also ignores the discussion of genetic susceptibility or resistance that is no doubt partly responsible for some individuals contracting a carcinoma whilst others do not.

120. The third complaint was that the experts expressed their opinions in the language of legal standards, including "the balance of probabilities" and "material contribution", although Professor Henderson disowned "material contribution or cause": Tcpt, 7/9/10, p 271(43).

121. How this complaint fell within the terms of s 79 of the *Evidence Act* was not explained. If the language truly involved legal concepts not available in common speech or having a different meaning from their common meaning, that might have been a matter for cross-examination. Most of the phrases complained of did not fit either of these categories. The phrase "the balance of probabilities" is neither esoteric, nor used by the law with a different meaning from common usage. It requires no explanation for a jury. Indeed, an epidemiologist might use that language as equivalent to a relative risk greater than two. Nor is causation generally a concept known only to the law, although its application may differ depending upon the purpose for which, or the discipline within which, it is deployed. Medical evidence, particularly, is encountered in the courts on a daily basis, using the language of causation. Evidence is not reduced to "legal arguments", as the appellants would have it, for that reason.

122. Fourthly, and perhaps most powerfully, the appellants submitted that their substantive objection to the evidence was also relevant to its admissibility. As explained earlier in these reasons, that submission was undoubtedly available to them, in that evidence which could not support a finding favourable to the plaintiff would not have satisfied the requirements of admissibility. Adopting the same course as the appellants in their written submissions, the substantive argument has already been addressed, and rejected.

123. It follows that the challenge to the admissibility of the evidence of each of the experts, as identified in common ground 1(a), must be rejected.

(6) Inference based on *Jones v Dunkel*

124. The next common ground raising a separate point of law, 1(c), complained that the trial judge had drawn an adverse inference from the appellants' failure to call expert medical evidence on the question of causation. This was said to have occurred in passages in his Honour's reasons at [190]-[192] and [209]. At [190] and [192] the trial judge merely referred to the evidence of the plaintiff's experts, who were described as "well qualified", noting that the defendants called no medical evidence to the contrary of the opinions expressed by the plaintiff's experts. The critical passage is at [209], which was in the following terms:

> "It is significant that no defendant calls expert evidence on the issue of causation. Allianz obtained a report from Professor Geoffrey Berry. WorkCover arranged for Associate Professor A B X Breslin, a highly regarded respiratory physician with extensive experience in asbestos related diseases, to examine Mr Sim. Neither expert is

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called, and I draw the inference that neither would have advanced the case argued by the defendants."

125. The first basis of complaint was no more than maintaining consistency with the appellants' primary argument: that is, if the plaintiff's witnesses were unable to express opinions as to the cause of the carcinoma, there was no sound basis for expecting the appellants' potential witnesses to be able to do so. There was, therefore, no adverse inference to be drawn.

126. That submissions fails at two points. First, it must fall once the foundation is removed. Secondly, Professor Berry was an epidemiologist: the appellants did not suggest that epidemiology might not be relevant to establishing or disproving causation. To the contrary, their primary position was that *only* epidemiology was relevant in the current state of medical knowledge.

127. The second basis of complaint was that Professor Berry's "conclusions" were before the Tribunal because they had been identified by the plaintiff's experts, to whom the report had been provided, who "did not disagree with his conclusions": written submissions, par 79.

128. That submission must be rejected. First, brief extracts were quoted by Professor Henderson, for example, and not necessarily "conclusions". Secondly, Professor Henderson did not agree with all that Professor Berry said, expressing his disagreement in some instances in strong terms.

129. No error of law is demonstrated in the manner in which the trial judge dealt with the failure of the appellants to adduce expert evidence; common ground 1(c) must be rejected.

(7) Assessment of excessive exposure

130. Common ground 1(e) complained that the trial judge had failed to make a necessary finding as to "the degree of additional asbestos exposure, if any, attributable to the negligence of the [appellants]". The qualifier "if any" should be disregarded. The trial judge made findings in respect of breach of duty on the part of each appellant.

131. It is true that the trial judge made no specific finding as to the degree to which the negligent exposure exceeded that which would have eventuated had reasonable care been taken. His Honour held that the failure to provide and supervise the use of adequate masks resulted in Mr Sim's exposure exceeding the "then NHMRC recommended level of less than 5 million particles per cubic foot averaged over a working week": at [129]. Although not pleaded, nor apparently addressed in the course of the hearing, in written submissions to the trial judge Allianz submitted that, on a finding of negligence, the inquiry in respect of causation had to be limited to the effects of the negligent exposure as opposed to the non-negligent exposure: written submissions, 1 November 2010, par 42. (It was not suggested that the other appellants adopted this specific submission.)

132. It is true that the trial judge did not deal with this issue in respect of causation. However, the need to make a precise calculation depended upon the findings based on Professor Henderson's evidence. If the cumulative consequences of continuing exposure were accepted, in the terms suggested by Professor Henderson, it was clear that the extent of the disparity rendered a precise calculation unnecessary. In fact, the fate of this challenge stands or falls with the final common ground.

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(8) Necessary condition test not satisfied

133. Common ground 1(f) asserted that it was not reasonably open on the admissible evidence to conclude that Mr Sim "would not have contracted lung cancer but for the negligence of" each appellant.

134. The premise underlying this ground was that the conduct of each appellant must be a *sine qua non* with respect to the harm suffered. This is sometimes described as the "counterfactual test" of causation because it poses the hypothetical question as to whether the harm would have resulted, if the defendant's tortious conduct were removed from the equation: see, eg, Michael S Moore, *Causation and Responsibility* (OUP, 2010), p 84. It is also known as the "necessary condition" test, in which form it has been adopted in one part of s 5D of the *Civil Liability Act 2002* (NSW) . Its operation in that context does not arise in the present case, to which the Act does not apply: see s 3B .

135. The second proposition relied on by the appellants was that the "commonsense" approach to causation, long favoured by the common law and approved in *March v E & MH Stramare Pty Ltd* [1991] HCA 12; 171 CLR 506 at 515-516 (Mason CJ), should be abjured in circumstances where causation is not capable of commonsense assessment and can only be established by application of scientific principles. For these purposes, the appellants did not seek to set up a necessary condition test as the only relevant test; they accepted that the results of applying that test may be over-inclusive, so that legal policy may restrict the category of those liable, even where the conduct is a necessary condition of the harm suffered. On the other hand, they said that satisfaction of the necessary condition test is a necessary condition of liability.

136. This approach is too limited and does not reflect presently accepted general law principles. In its terms, it rejects reliance on "material contribution" as a sufficient causal connection. In a classic analysis of causation principles in the law of tort entitled "Causation in Tort Law" (1985) 73 Cal L Rev 1735, Professor Richard W Wright undertook a comprehensive survey of causation in tort law, the analysis covering theories from David Hume to the date of publication. In discussing the 'NESS test' (necessary elements of a sufficient set of conditions) as articulated by Hart and Honoré, Wright noted that the courts had not required independent sufficiency for each element in cases of duplicative causation: pp 1791-1792. Referring to two early American cases, *Corey v Havener* , 182 Mass 250; 65 NE 69 (1902) (noisy motorcycles) and *Anderson v Minneapolis*, St P & S Ste M Ry, 146 Minn 430; 179 NW 45 (1920) (merged fires) he noted that the courts did not require the plaintiff in either case to prove independent sufficiency of each contributing factor, but only that each factor contributed to the injury. Professor Wright noted similar results in pollution cases including, more recently, *Michie v Great Lakes Steel Div* , 495 F2d 213 (6th Cir), cert denied, 419 US 997 (1974) .

137. This analysis is apt in the present case: to oversimplify the facts for the purposes of noting the principle, one may assume there were four defendants each of whom contributed (through tortious conduct) 20% of the exposure to asbestos dust which caused the plaintiff's lung cancer; that the contribution of each, taken alone, would not have been shown, on the balance of probabilities, to cause the lung cancer, but that the combination of any two would have been sufficient. Thus the contribution of each individually was neither necessary (because there were three other equivalent contributions) nor sufficient to cause the injury. The example was illustrated in the course of argument by a temporal analysis. Assuming two were

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sufficient, if the first two in point of time had the necessary effect, the third and fourth would have been redundant; alternatively, if the third and fourth were sufficient, it could not be demonstrated on the balance of probabilities that the chain of causation commenced with either the first or second defendant.

138. The appellants contended that if each defendant should in those circumstances be found liable, the conclusion reflected the need, identified in *Fairchild* , to vary the general law principles, even those relating to material contribution, for the defendants, or any of them, to be found liable. It not being appropriate for the Tribunal, nor for that matter this Court, to create a new form of tortious liability, the appellants contended that they should succeed: cf *Tabet v Gett* [2 010] HCA 12; 240 CLR 537; *Gett v Tabet* [2009] NSWCA 76; 254 ALR 504. However, the flaw in the appellants' argument is its foundation in epidemiology. If Professor Henderson's evidence was inadmissible, the appellants succeed on a different basis; if Professor Henderson's evidence was admissible (as held above) then it must be taken into account in the analysis of causation. Indeed, as explained by Lord Phillips PSC in *Sienkiewicz v Greif (UK) Ltd* [2011] 2 WLR 523 at [90] :

> "I see no scope for the application of the 'doubles the risk' test in cases where two agents have operated cumulatively and simultaneously in causing the onset of a disease. In such a case the rule in *Bonnington* [discussed below] applies. Where the disease is indivisible, such as lung cancer, a defendant who has tortiously contributed to the cause of the disease will be liable in full. Where the disease is divisible, such as asbestosis, the tortfeasor will be liable in respect of the share of the disease for which he is responsible."

139. In the 2009 publication of the American Law Institute (ALI), *Restatement (Third) of Torts: Liability for Physical and Emotional Harm*, Vol 1 380-1 (s27, Illustration 3) an example is given of three people who independently but simultaneously and negligently lean against the plaintiff's car, causing it to roll over the edge of a precipice. On the hypothesis presented, the force applied by each was insufficient to effect the damage, but the combined force of two was sufficient. Accordingly, the conduct of none was necessary or sufficient to cause the damage. This constituted one of a number of examples given by Professor Jane Stapleton in "Factual Causation" (2010) Fed L Rev 467 at 475-476. Approving the statement of the ALI, Professor Stapleton accepts that each should be held liable in respect of an indivisible outcome.

140. There is authority in this country to support that approach. Thus, the doctrine of material contribution was applied by the House of Lords in *Bonnington Castings Ltd v Wardlaw* [1956] AC 613, a case involving "pneumoconiosis ... caused by a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years": at 621 (Lord Reid). There were two sources of the dust inhaled by the plaintiff, namely pneumatic hammers and swing grinders. It was conceded by the employer that the dust from the swing grinders should have been intercepted and removed. Lord Reid stated at 621:

> "... I cannot agree that the question is: which was the most probable source of the respondent's disease, the dust from the pneumatic hammers or the dust from the swing grinders? It appears to me that the source of his disease was the dust from both sources, and the real question is whether the dust from the swing grinders materially contributed to the disease. What is a material contribution must be a question of degree. A

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> contribution which comes within the exception de minimus non curat lex is not material, but I think that any contribution which does not fall within that exception must be material."

Viscount Simons (at 618), Lord Tucker (at 623), Lord Keith of Avonholm (at 626) and Lord Somervell of Harrow (at 627) agreed.

141. In *March v Stramare* , Mason CJ (with the concurrence of Deane J, writing separately, Toohey and Gaudron JJ) noted that uniformity of approach to questions of causation might not be possible, given the purpose of allocating legal responsibility for conduct. *Bonnington Castings* (at 620) was referred to as providing support for the following proposition (at 514):

> "Nonetheless, the law's recognition that concurrent or successive tortious acts may each amount to a cause of the injuries sustained by a plaintiff is reflected in the proposition that it is for the plaintiff to establish that his or her injuries are 'caused or materially contributed to' by the defendant's wrongful conduct."

142. More recently, the statement of Lord Reid in respect of material contribution was referred to without disapproval by the High Court in *Amaca v Ellis* [2010] HCA 5; 240 CLR 111. Their Honours stated:

> "[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, 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 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* , 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."

143. While questions of material contribution did not arise in that case, there was no suggestion that, in another case, they could not support a finding of liability: indeed the discussion assumes that they could. Further, in terms of the precise question identified at [68], it was not in dispute in the present case that asbestos did cause Mr Sim's lung cancer: the question related rather to what has been described as "the defendant-identification issue": Stapleton, "Factual Causation and Asbestos Cancers", (2010) 126 LQR 351 at 356.

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144. In *Amaca v Booth* , Gummow, Hayne and Crennan JJ referred to the same issue at [70] :

> "The 'but for' criterion of causation proved to be troublesome in various situations in which multiple acts or events led to the plaintiff's injury, for example, where the development of a particular medical condition was the result of multiple conjunctive causal factors. In such cases what may be unclear is the extent to which one of these conjunctive causal factors contributed to that state of affairs. These situations have been addressed by the proposition stated by Lord Watson in *Wakelin v London and South Western Railway Co* [(1886) 12 App Cas 41 at 47] that it is sufficient that the plaintiff prove that the negligence of the defendant 'caused or materially contributed to the injury'."

Their Honours also referred to the speech of Lord Reid in *Bonnington Castings* at 621 and *Amaca v Ellis* at [67] .

145. The appellants identified no authority for the proposition that substantial successive and cumulative tortious conduct, independently engaged in by several defendants, did not render each liable for the consequential and individual harm, in circumstances where individually, the tortious conduct was neither necessary nor sufficient to cause the harm. The line of authorities referred to above demonstrates that evidence of causation satisfying those conditions is sufficient to establish liability according to general law principles. Accordingly, ground 1(f) must be rejected.

(9) WorkCover Authority - further grounds

(a) further issues

146. Wallaby Grip did not seek to rely upon any grounds beyond the common grounds addressed above. The WorkCover Authority, however, raised four further grounds, in the following terms:

> "2. His Honour erred in point of law in his resolution of the issue of causation in failing to apply the principles enunciated by the High Court in *Amaca v Ellis* 240 CLR 111 as to causation of the Deceased's lung cancer condition in respect of the Deceased's exposure to asbestos in the employ of either *Asbestospray Fireproofing and Insulation (NSW) Pty Limited* or *Asbestospray Corporation Australasia Pty Limited*.
>
> 3. His Honour erred in point of law in his resolution of the issue of causation in finding that the Plaintiff had discharged the onus of proof as to causation of the Deceased's lung cancer condition in respect of the Deceased's exposure to asbestos in the employ of either *Asbestospray Fireproofing and Insulation (NSW) Pty Limited* or *Asbestospray Corporation Australasia Pty Limited*.
> ...
> 5. His Honour erred in point of law in awarding any damages against WorkCover Authority (NSW) in respect of the Deceased's exposure to asbestos in the employment of *Asbestospray Fireproofing and Insulation (NSW) Pty Limited*
>
> a. In finding that there was a policy of insurance within the terms of s 151AB of the Workers Compensation Act 1987 on the last day of the Deceased's employment, whether it

be that the Deceased's last day of employment by *Asbestospray Fireproofing and Insulation (NSW) Pty Limited* was on 30th June 1972 (J [14]) or May 1972 (J [158]);

b. Alternatively, given His Honour's finding (J [163]) that there was no Workers Compensation policy between June 1972 and 10 May 1974, in finding (J [146 and 158] [)], that there was a policy for Workers Compensation insurance within the terms of s 15 1AB of the Workers Compensation Act 1987 on the last day of the Deceased's employment by *Asbestospray Fireproofing and Insulation (NSW) Pty Limited*, whether it be that the Deceased's last day of employment with by [sic] *Asbestospray Fireproofing and Insulation (NSW) Pty Limited* was the 30th June 1972 (J [14]) or May 1972 (J [158]).

6. His Honour erred in point of law in ordering that the Appellant pay the Plaintiff's costs on an indemnity basis from 14th May 2010."

(b) principles enunciated in *Amaca v Ellis*

147. The written submissions in support of ground 2 merely noted that the trial judge adopted the "all exposure is causative" approach in reaching the ultimate determination of the lung cancer, and in this regard fell into error of law: written submissions, par 12. That complaint was misconceived. As explained in the joint judgment in *Amaca v Booth* , the approach adopted in *Amaca v Ellis* depended upon the evidence in that case, which differed from that called in *Amaca v Booth* : at [71] . The evidence in *Amaca v Booth* (and in the present case) also differed from that relied on in *Fairchild* : at [79] and [80] . The submissions failed to identify any more specific principle which was not applied. Ground 2 must be rejected.

(c) onus of proof

148. Neither in written submissions, nor in oral argument, did WorkCover seek to identify any issue raised by this ground which was not otherwise covered by the common ground 1. Ground 3 should also be dismissed.

(d) insurance cover

149. The liability of WorkCover arose, if at all, under s 151AB of the *Workers Compensation Act* , which relevantly provided:

"**151AB Special insurance provisions relating to occupational diseases**

(1) If an employer is liable independently of this Act for damages for an occupational disease contracted by a worker, the following provisions have effect for the purposes of any policy of insurance obtained by the employer:

(a) the liability is taken to have arisen when the worker was last employed by the employer in employment to the nature of which the disease was due ...
...
(6) In sections 151AB and 151AC :

occupational disease means a disease of such a nature as to be contracted by a gradual process, and includes:

(a) a dust disease as defined by the *Workers' Compensation (Dust Diseases) Act 1942* ...."

150. There was dispute in the Tribunal as to the insurance cover held by Asbestospray Fireproofing (the second employer). The plaintiff alleged in her statement of claim that the company was insured by Vanguard Insurance Company Ltd ("Vanguard"), seeking relief pursuant to s 6(4) of the *Law Reform (Miscellaneous Provisions) Act* . Vanguard being insolvent, WorkCover was sued as manager of the Insurers' Guarantee Fund, pursuant to s 236 of the *Workers Compensation Act* .

151. In respect of the second employer, WorkCover denied in the Tribunal that there was a contract of insurance involving Vanguard (defence to third amended statement of claim, par 5(a)) and further denied that if there were such a contract, Mr Sim was an employee of the second employer, asserting that he was an independent contractor: par 5(i).

152. The precise point or points sought to be raised by ground 5 in WorkCover's notice of appeal was (or were) obscure. The written submissions, however, identified the relevant point as arising from two findings of fact made by the trial judge, namely that Mr Sim's employment ended on 30 June 1972, but that Vanguard's insurance policy ceased in May 1972. Accordingly, the policy did not operate as at the last day of his employment and s 151AB was not engaged: written submissions, pars 14-16. (Although paragraph 17 of the written submissions appeared to raise a separate issue as to the existence of a relevant policy, counsel indicated that no separate question was sought to be raised: Tcpt, 16/11/2011, p 41(28-31).) The ground therefore raised two issues, namely:

(a) did s 151AB require a policy extending to the last day of the worker's employment, and
(b) if it did, were the facts found by the Tribunal inconsistent with such a requirement?

153. It is convenient to consider first whether his Honour made a finding that Mr Sim's employment with the second employer continued until 30 June 1972. The third amended statement of claim made no such assertion: it alleged, consistently, that the employment ended in May 1972: pars 6 and 6A. WorkCover's defence also identified the date of termination of employment as May 1972: par 5(a), (b). In setting out the parties to the proceedings, the trial judge referred to WorkCover in the following terms at [3]:

> "WorkCover Authority (NSW) (Workcover), heir to the liabilities of Vanguard Insurance Company, the insolvent insurer of both Asbestospray Fireproofing and Insulation (NSW) Pty Ltd ... for whom Mr Sim worked between June 1967 and 30 June 1972, and Asbestospray Corporation Australasia Pty Ltd ... for whom he worked between 1 July 1972 and 17 September 1975."

154. This statement was not a finding of fact, but a summary of the parties sued, which did not accurately reflect the pleading. At [12], the trial judge noted, in summarising the history of Mr Sim's asbestos exposure, that Asbestospray Fireproofing had been registered on 15 June 1967 by Mr Bill McCudden. At [14] he stated:

> "In May 1972 Mr McCudden sold the business of Asbestospray Fireproofing to Asbestospray Australasia. Mr Sim's last day of employment with Asbestospray Fireproofing was 30 June 1972. Mr Sim became an employee of the new owner and continued work as before."

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155. There was an ambiguity raised by this description; it appears clear that Mr Sim's employment terminated when the business was sold, but whether that was May 1972 or 30 June 1972 was unclear. His Honour later said that the business was sold "in June 1972": at [27]. At that point, his Honour was seeking to resolve what he described as "a preliminary dispute", namely that Mr Sim had not worked fulltime for Asbestospray Fireproofing: at [17] . His Honour then stated at [24]:

> "In an industrial history given to the Dust Diseases Board dated 28 July 2006, Mr Sim stated that '*I was **employed by** Asbestospray from 1966 until 1975'.* Although he does not in this statement reveal the change of ownership in 1972, that lapse is understandable because it remained the same business conducted from the same premises throughout that period despite the change of ownership."

156. His Honour summarised certain other evidence and made a finding, relied on by WorkCover:

> "[26] Although in his statement of 25 January 1996, given in support of a claim for damages made by a co-worker, Mr Sim said that: *"During McAllister's time several of the staff were put on a sub contract basis, including myself"*, this statement is also to be seen in context.
>
> [27] Mr John McAllister said that he worked as manager of Asbestospray from 6 April 1972 until the business was sold in June 1972. Although Mr McCudden denied employing Mr McAllister, it may be that in this short period Mr McAllister was employed by the purchasers of the business to gain experience of its operations. In any event, Mr McCudden said that the subcontracting arrangements lasted for only about 30 days, and ceased at the request of Mr Sim.
>
> [28] I find that, with the exception of 30 days, Mr Sim worked full-time as a full-time employee of Asbestospray Fireproofing between 1967 and 30 June 1972, and as a full-time employee of Asbestospray Australasia from 1 July 1972 until 17 September 1975."

157. The issue being addressed in the preceding passages was that of employment or independent contractor. Neither the precise date of the sale of the business, nor the date of termination of the insurance policy was in issue. When he turned to the question of insurance of Asbestospray Fireproofing at [134] his Honour addressed first the primary issue as to whether the company had been insured by Vanguard. Both Mr McCudden and a broker for the company, Mr Hicks, had given evidence that it was Vanguard. A further issue arose as to the date on which the insurance commenced, the trial judge holding at [148]:

> "Because Mr McCudden said 'In respect of Asbestos Spray Fireproofing & Insulation (NSW) Proprietary Limited I always took out workers compensation insurance ... with Vanguard [I]nsurance Company Ltd', I regard his statement that the business he conducted from 36 Chappel Street Marrickville was 'between 1969 and 1972' an error of recollection, and that as the business was conducted between 1967 and 1972, Vanguard was the insurer for the whole of that time."

158. His Honour also found at [146]:

> "The suggestion that brokers for the new owners of the business continued to place the insurance with Vanguard is confirmed by a proposal to Vanguard for insurance from 10

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> May 1974 to 10 May 1975 made by Hewitt Cummins and Associates on behalf of Asbestospray Australasia tendered in evidence. This proposal limits common law indemnity to $100,000."

159. His final conclusion was at [158], in the following terms:

> "I find that between mid-1967 and May 1972 when Mr Sim last worked for Asbestospray Fireproofing in an employment to the nature of which his asbestos diseases were due, there existed as between Asbestospray Fireproofing and Vanguard Insurance Company Ltd a contract of insurance whereby Vanguard agreed to indemnify Asbestospray Fireproofing in respect of its liability independently of the *Workers Compensation Act* , and that this indemnity was unlimited in amount."

160. Although there is a clear conflict between the last statement and the earlier statements, read as a whole, his Honour was satisfied that the insurance with Vanguard continued to the sale of the business, at which time a new policy was taken out with the same company. Similarly, Mr Sim's employment extended to the time of the sale of the business, at which stage fresh employment commenced with the third employer.

161. In written submissions in this Court, the plaintiff noted that there had been unchallenged evidence that the date of sale of the business was 6 May 1972. That evidence was not contained in the appeal books, but counsel for WorkCover at the trial, asked a question of Mr McAllister in cross-examination as to the date of sale of the business stating "We know from other records, 6 May 1972": Tcpt, 08/09/10, p 323(34). Mr McAllister commenced employment as manager of Asbestospray Fireproofing on 6 April 1972. There was no suggestion that Mr Sim's employment with the company continued after the sale of the business.

162. The only finding which might have suggested that his date of termination was other than the date of sale of the business was his Honour's conclusion that there was a period of 30 days when he worked otherwise than as a fulltime employee of Asbestospray Fireproofing: see [28], set out at [156] above. If the period of sub-contracting was indeed the last 30 days during which Mr Sim worked for Asbestospray Fireproofing, that would have taken the date of termination of his employment back to an earlier date, which would still have been within the period of the Vanguard insurance policy.

163. The mistaken reference to 30 June 1972 may well have resulted from reliance on the submissions for Workcover, which, without reference to evidence, used that date: Black Appeal Book, pp 817 and 871. Further, there does not appear to have been any issue raised before the Tribunal that the date of Mr Sim's employment extended beyond the date of expiration of the relevant Vanguard policy. Nor was it suggested that the Vanguard policy terminated before the date of sale of the business. Accordingly, the factual premise on which ground 5 is based must be rejected. The first question therefore does not arise. If it had arisen, it would have been necessary to explain how it could have been relied upon by WorkCover in the present appeal, it not having been raised below and thus not having been considered, let alone decided, by the Tribunal: see *Tribunal Act* , s 32(1) .

(e) indemnity costs

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164. The notice of appeal did not condescend to identify any error of law on the part of the Tribunal in making the indemnity costs order. The written submissions for WorkCover, somewhat cryptically, noted that the claim for indemnity costs had not been made when judgment was delivered. The relevance of that proposition was obscure. There was an issue, dealt with by his Honour, as to whether the notice of motion had been filed within the time permitted by the Uniform Civil Procedure Rules, r 36.16 . No ground was identified for setting aside his Honour's order on the basis that the application for indemnity costs had not been made within time.

165. On 14 May 2010 the solicitors for the plaintiff served on each of the defendants a notice entitled "Offer of compromise", in the following terms:

> "The plaintiff offers to compromise this action on terms that:
>
> 1. The defendants pay to the plaintiff the sum of $290,000 plus her costs as agreed or assessed.
>
> 2. This offer shall remain open for acceptance until 5pm 11 June 2010.
>
> This Offer of Compromise is made in accordance with Clause 86 of the Dust Diseases Tribunal Regulation , 2007."

166. The judgment entered in favour of the plaintiff against WorkCover was in an amount in excess of $375,000. (The amount of the judgment against the other defendants varied, because of their proportionate shares of the separate judgments in respect of Mr Sim's asbestosis. Because the joint and several liability for lung cancer exceeded $290,000, in respect of each, the plaintiff bettered her offer.)

167. The Dust Diseases Tribunal Regulation 2007 (NSW) ("the Tribunal Regulation"), Pt 6 , dealing with offers of compromise, states that the Part "displaces any provision of rules of court with respect to the acceptance to the acceptance or rejection of an offer of compromise": cl 84 . The UCPR apply in the Dust Diseases Tribunal: UCPR, r 1.5 and Sch 1, with exceptions not presently relevant. Apart from exceptions, there may be "local rules" that prevail over the UCPR : r 1.7 . Those include the "Dust Diseases Tribunal Rules": UCPR, Sch 2. In fact, there are Dust Diseases Tribunal Rules , which are separate from the Dust Diseases Tribunal Regulation . The *Civil Procedure Act 2005* (NSW) assumes that courts and tribunals which are subject to the Act and the UCPR will continue to have their own rules which will include the uniform rules to the extent to which they are applicable: s 10 . However, the uniform rules prevail over any provision of any local rules "unless the uniform rules expressly provide that the provision of the local rules is to prevail": s 11(1) .

168. Clause 84 of the Tribunal Regulation cannot have that effect: it may be, therefore, that the provisions of Pt 6 of the Tribunal Regulation only operate to the extent that they are not inconsistent with equivalent provisions in the UCPR .

169. Although the offer claimed it was made pursuant to the Tribunal Regulation , as the trial judge noted at [9] of his costs judgment, thereby giving the plaintiff an entitlement under cl 87, the defendants' response was dealt with, at least in part, under UCPR, r 20.26(5) . (The confusion,

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which was maintained in this Court, suggests that steps should be taken by the relevant rule-making bodies to resolve potential conflicts and make manifest the rules which are to prevail in the Tribunal.)

170. WorkCover's first complaint, raised before his Honour and maintained in this Court, was that the offer was "incapable of acceptance" until the plaintiff had supplied all the material upon which she ultimately relied, which only occurred after the offer had lapsed: written submissions, par 28. Workcover relied in support of this submission on r 20.26(4) . It may be accepted for present purposes that the rule is relevant. However, that sub-rule cannot be read in isolation from sub-rules (1) and (5): together they provide as follows:

> "**20.26 Making of offer**
>
> (1) In any proceedings, any party may, by notice in writing, make an offer to any other party to compromise any claim in the proceedings, either in whole or in part, on specified terms.
> ...
> (4) Despite subrule (1), a plaintiff may not make an offer unless the defendant has been given such particulars of the plaintiff's claim, and copies or originals of such documents available to the plaintiff, as are necessary to enable the defendant to fully consider the offer.
>
> (5) If a plaintiff makes an offer, no order may be made in favour of the defendant on the ground that the plaintiff has not supplied particulars or documents, or has not supplied sufficient particulars or documents, unless:
>
> (a) the defendant has informed the plaintiff in writing of that ground within 14 days after receiving the offer, or
> (b) the court orders otherwise."

171. The trial judge addressed a complaint in substantially the same form, namely that "the plaintiff's case was not particularised, and changed fundamentally after the offer of compromise was served": at [30]. In effect, the trial judge treated such a complaint as subject to sub-rule (5). There was no argument that his Honour was in error in that respect. There was no notice in writing to the plaintiff pursuant to sub-rule (5)(a). The trial judge gave consideration to the material available to the defendants (at [32]-[40]), in support of his view that the defendants' submission was "wholly without merit": at [30] .

172. WorkCover invites this Court, in effect, to make a different evaluative judgment on the basis that the second supplementary report of Professor Henderson and further reports of Drs Bryant and Yates were served "10 days after the offer was made, and only 18 days before the offer expired": submissions, par 25. Although some particulars were sought by the defendants on 19 May 2010, to which a detailed response was given on 2 June 2010, it was submitted that this was the subject of further "clarification", sought on 2 July and provided on 8 July 2010.

173. The timing, the contents of the letters and the failure of the defendant to call in aid r 20.26(5) i n seeking particulars casts no doubt on the evaluation undertaken by the trial judge.

174. Finally, WorkCover relied upon the fact that Professor Henderson's third supplementary report was served after the offer had lapsed. It is true that Professor Henderson's third

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supplementary report provided significant evidentiary support to the plaintiff's claims, but it was, in substance, a response to a report prepared for Allianz by Professor Berry dated on 24 August 2010.

175. The trial judge did not refer to the service of Professor Henderson's third supplementary report, possibly because he had formed the view on the basis of material available on 14 May 2010 that WorkCover was in a position to consider fully the offer and perhaps because, at the hearing, no great weight was placed by WorkCover upon the later material replying to a report which it apparently had no part in supplying. This Court was not taken to any submissions in that respect made at the costs hearing. Neither in its written submissions in reply in this Court, nor in oral argument, did it raise any response to the plaintiff's submissions in this respect. It cannot be said that his Honour erred in the evaluative judgment he formed in respect of this issue, let alone that he erred in law.

176. The second objection raised by WorkCover, and repeated in this Court, was that the offer was not open for "such time as is reasonable in the circumstances", pursuant to r 20.26(7)(b) .

177. The trial judge held that the offer was open for a reasonable time, given the circumstances: at [41]-[43]. The argument in this Court asserted that his Honour failed to take into account the need for WorkCover to be informed in full of the case it was to meet. That complaint, in substance, echoed the first objection and should be rejected for similar reasons. There was no attempt to identify any relevant error of law in the reasoning of the trial judge.

178. Thirdly, WorkCover submitted that "no defendant individually could have accepted the plaintiff's offer of compromise given the terms of the offer of compromise": written submissions, par 32. The reason for this proposition was that an acceptance on the part of one defendant would result in the plaintiff recovering the whole of her costs from that defendant.

179. **Following paragraph cited by:**

Tan Republic Pty Ltd (ACN 147 290 926) v Isabella Shop Fitout and Design Pty Ltd (ACN 147 193 815) (No. 2) (31 July 2013) (Gibson DCJ)

> 23. In *Allianz Australia Ltd v Sim; WorkCover Authority (NSW) v Sim; Wallaby Grip (BAE) Pty Ltd (In liq) v Sim* [2012] NSWCA 68 at [179] the court noted that an offer of compromise, to be valid, must be put to all defendants, in order to "trigger the operation of the rule". Those defendants may protect their respective interests by offers between themselves (*Stevedoring Industry Finance Committee v Gibson* [2000] NSWCA 179). However, the fact that a plaintiff fails against one defendant out of three must be of significance.

In response to what must have been a similar argument put at trial, the Tribunal stated at [44]:

> "An offer by a plaintiff sufficient to trigger the operation of the rule may be put to multiple defendants. Their several interests may be protected in terms of offers between themselves (*Henderson v Amadeo* (unreported, Federal Court, Vic, 22 March 1990, *Stev edoring Industry Finance Committee v Gibson* [2000] NSWCA 179)."

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180. No legal error was identified as attending that reasoning. Accordingly, the third basis of challenge to the order must also be rejected.

(10) Conclusion

181. Each of the appeals has failed on all grounds and must therefore be dismissed. The appellants must pay the costs of Mrs Sim in respect of each appeal.

182. **MEAGHER JA**: I agree, for the reasons given by Basten JA and for the additional reasons given by Allsop P that these three appeals should be dismissed and the appellants ordered to pay the costs of Mrs Sim.

****

Cited by:

George v State of New South Wales [2025] NSWDC 292 (06 August 2025) (Catsanos SC DCJ)

> 5. As expounded in, for example, Allianz Australia Ltd v Sim [2012] NSWCA 68 .

Ioannidis v Carretero [2025] NSWDC 258 (15 July 2025) (Catsanos SC DCJ)

> 3. See generally Allianz Australia Ltd v Sim; WorkCover Authority (NSW) v Sim; Wallaby Grip (BAE) Pty Ltd (In Liq) v Sim [2012] NSWCA 68 and the authorities discussed, especially at [37] – [52] .

Uniform Evidence Manual [2023] JCV Uniform_Evidence_Manual (06 May 2025)

> It is often necessary for a court to make a ruling under s 79 before all the relevant evidence of the factual basis is adduced. In such cases, s 57 (Provisional relevance) may be used to enable opinion evidence to be provisionally admitted, subject to further evidence of the factual basis being admitted (see, e.g. Allianz Australia Ltd v Sim; WorkCover Authority (NSW) v Sim; Wallaby Grip (BAE) Pty Ltd (In liq) v Sim [2012] NSWCA 68).

Berhero Pty Ltd v Hinds [2023] NSWSC 1022 (30 August 2023) (Rees J)

> 80. As Allsop P also noted in *Allianz Australia Ltd v Sim* [2012] NSWCA 68, the requirements of section 79 "should not be elevated into something more than they are: procedural rules to limit evidence to that which is rational and coherent and properly arising from expertise and directed to areas in respect of which the court needs assistance": at [9] .

Return to Work Corporation of South Australia v BI (Contracting) Pty Ltd [2022] SASCA 49 (02 June 2022)

> 48. The initial response of BIC on the appeal was to say that while *Amaba v Booth* was a mesothelioma case, it could have easily relied on a series of lung cancer cases where the aetiology is the same. To this end, it pointed to cases where Professor Henderson's 'cumulative effect' theory of causation as described in *Amaba v Booth* , set out in the passages

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reproduced above, have been endorsed, including with respect to lung cancer. [21] Professor Henderson gave evidence in those cases as well, applying the same process of analysis to the aetiology of lung cancer as an indivisible dust disease injury.

via

[21] *Lorraine Fay Sim v Allianz Australia Ltd* [2010] NSWDDT 19 at [185]-[193] ; *Allianz Australia Ltd v Sim; WorkCover Authority (NSW) v Sim; Wallaby Grip (BAE) Pty Ltd (In liq) v Sim* [2012] NSWCA 68 at [116]-[122] .

Dickson v Northern Lakes Rugby League Sport and Recreation Club Inc [2019] NSWDC 426 (15 August 2019) (Abadee DCJ)

35. Counsel for both parties also referred to s 80 of the *Evidence Act* ; and especially s 80(a) . It should, I think, be obvious that s 80 should not be construed in a way that maintains the old common law prohibition. Further, in the context of the opinion in 20(c), although that opinion is directed to an issue of causation, it has been accepted that evidence from an expert is admissible on that subject (*Allianz Australia Ltd v Sim* [2012] NSWCA 68 per Allsop P (Meagher JA agreeing) at [34]-[35] and Basten JA at [118]-[121]).

Lymbery v Shoalhaven City Council [2016] NSWWCCPD 38 (29 July 2016) (Acting Deputy Larry King SC)

37. Then in relation to Dr Bodel, as outlined above in his report of 18 September 2015 he squarely expresses the view that the requisite causal connection exists, part of that expression of opinion being extracted by the Arbitrator (at [29]). It seems to me that she discounts the view expressed by the doctor for want of sufficient reasoning of explanation on a *Makita* basis, but in my opinion this too exacting a reading of the report, which should be seen as giving support to the appellant's claim. Authority subsequent to *Makita* has pointed out, modifying the potential impact of that decision, that it need only appear that the opinion being expressed must, on the probabilities, be based wholly or substantially on specialised knowledge, a proposition at its most forceful in relation to expert medical evidence: see *Hancock v East Coast Timber Products Pty Ltd* [2011] NSWCA 11; 80 NSWLR 43; *Allianz Australia Limited v Sim* [2012] NSWCA 68 and also *Sydneywide Distributors Pty Limited v Red Bull Australia Pty Limited* [2002] FCAFC 157. Again with due respect to the learned Arbitrator I think it would be unrealistic to imagine that Dr Bodel, in saying what is set out in [29] of her Reasons, was either on the face of things stepping outside the field of medical training and expertise or needed to explain how he was staying within it. I think he was clearly within it.

May v Military Rehabilitation and Compensation Commission [2015] FCAFC 93 (30 June 2015) (Allsop CJ, Kenny, Besanko, Robertson and Mortimer JJ)

Allianz Australia Ltd v Sim [2012] NSWCA 68

Amaca Pty Ltd v Booth

May v Military Rehabilitation and Compensation Commission [2015] FCAFC 93 (30 June 2015) (Allsop CJ, Kenny, Besanko, Robertson and Mortimer JJ)

219. These statements have been often applied: in such cases as *Tubemakers of Australia v Fernandez* (1976) 50 ALJR 720; 10 ALR 303 at 307, 310-311, *EMI (Australia) Ltd v Bes* [1970] 2 NSWR 238 at 241 ; *Amaca Pty Ltd v Booth* [2011] HCA 53; 246 CLR 36 at 61-62 [69] ; *Tabet v Gett* [2010] HCA 12; 240 CLR 537 at 588 [149] ; *Australian Iron and Steel Ltd v Connell* [1959] HCA 54; 102 CLR 522 at 535-536 [6] ; *Allianz Australia Ltd v Sim* [2012] NSWCA 68 at [48] ; *Evans v Queanbeyan City Council* [2011] NSWCA 230; *Webb v Repatriation Commission* [1988] FCA 127 at [18] ; *Australian Telecommunications Commission v Barker* [1990] FCA 489; 12 AAR 490 at 493-494 ;

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Major Engineering Pty Ltd v Timelink Pacific Pty Ltd (No 2) [2009] VSCA 83 at [19] . It is also the point made by Latham CJ in *Hume Steel* : 75 CLR 242 at 252.

Tan Republic Pty Ltd (ACN 147 290 926) v Isabella Shop Fitout and Design Pty Ltd (ACN 147 193 815) (No. 2) [2013] NSWDC 321 (31 July 2013) (Gibson DCJ)

23. In *Allianz Australia Ltd v Sim; WorkCover Authority (NSW) v Sim; Wallaby Grip (BAE) Pty Ltd (In liq) v Sim* [2012] NSWCA 68 at [179] the court noted that an offer of compromise, to be valid, must be put to all defendants, in order to "trigger the operation of the rule". Those defendants may protect their respective interests by offers between themselves (*Stevedoring Industry Finance Committee v Gibson* [2000] NSWCA 179). However, the fact that a plaintiff fails against one defendant out of three must be of significance.

Van Soest v BHP Billiton Limited [2013] SADC 81 (17 June 2013) (Parsons J)

Parker v BHP Billiton Limited [2011] SADC 104; *Cadoo v BHP Billiton Limited* [2012] SADC 31; *Roads and Traffic Authority of New South Wales v Dederer* (2007) 234 CLR 330; *BHP Billiton Limited v Parker* (2012) 113 SASR 206; *Bankstown Foundry Pty Ltd v Braistina* [1986] HCA 20; *Czatyrko v Edith Cowan University* (2005) 79 ALJR 839; *Dovuro Pty Ltd v Wilkins* (2003) 215 CLR 317; *McPherson's Limited v Eaton* (2005) 65 NSWLR 187; *Seltsam Pty Limited v McNeil* (2006) 4 DDCR 1; *The Bell Group Ltd (In Liq) v Westpac Banking Corporation (No.9)* [2008] WASC 239; *Bendix Mintex Pty Ltd v Barnes* (1997) 42 NSWLR 307; *AMACA Pty Limited (Under NSW Administered Winding Up) v Booth & Another and AMABA Pty Limited (Under NSW Administered Winding Up) v Booth & Another* [2011] 246 CLR 36; *AMA BA Pty Limited (Under NSW Administered Winding Up) v Booth and AMACA Pty Ltd (Under NSW Administered Winding Up) v Booth* [2010] NSWCA 344; *Hamilton v BHP Billiton Ltd* [2012] SADC 25; *Coc katoo Dockyard Pty Limited v Browne* [2001] NSWCA 58; *The Council of Shire of Wyong Council v Shirt* (1980) 146 CLR 40; *Bonnington Castings Limited v Wardlaw* [1996] AC 613; *Strong v Woolworths Limited* (2012) 285 ALR 420; *Allianz Australia Ltd v Sim; Worksover Authority (NSW) v Sim; Wallaby Grip (BAE) Pty Ltd (in liq) v Sim* [2012] NSWCA 68 ; *Dasreef Pty Limited v Hawchar* [2010] NSWCZ 154; *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111; *March v Stramare (E & M H) Pty Limited and Another* (1991) 171 CLR 506, considered.

Van Soest v BHP Billiton Limited [2013] SADC 81 (17 June 2013) (Parsons J)

714. The recent case in the New South Wales Court of Appeal of *Allianz Australia Ltd v Sim; Workcover Authority (NSW) v Sim; Wallaby Grip(BAE) Pty Ltd (in liq) v Sim* [497] also concerned the role of the “but for” test. Similarly in that case the expert evidence relied upon by the plaintiff was such that it could not be said in respect of each of the appellants that, but for its negligent exposure of the plaintiff to asbestos, he would not have contracted his lung cancer. The Court applied *AMACA v Booth* .[498]

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714. The recent case in the New South Wales Court of Appeal of *Allianz Australia Ltd v Sim; Workcover Authority (NSW) v Sim; Wallaby Grip(BAE) Pty Ltd (in liq) v Sim* [497] also concerned the role of the “but for” test. Similarly in that case the expert evidence relied upon by the plaintiff was such that it could not be said in respect of each of the appellants that, but for its negligent exposure of the plaintiff to asbestos, he would not have contracted his lung cancer. The Court applied *AMACA v Booth* .[498]

via

[497] [2012] NSWCA 68

CSR Timber Products Pty Ltd v Weathertex Pty Ltd [2013] NSWCA 49 (11 March 2013) (Bathurst CJ, Meagher and Hoeben JJA)

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28. I do not understand Weathertex to allege that it is entitled to a more limited indemnity under s 151Z(1)(d) on the basis that the worker was entitled independently of the Act to take proceedings against it and CSR to recover damages so that the provisions of s 151Z(2) might apply. Weathertex and CSR would each be liable to the worker if both negligently exposed him to conditions which materially contributed to the carcinoma. In those circumstances each would be liable for the worker's loss subject to the application to one or both of them of the modified damages regime in Division 3 in Part 5 of the 1987 Act: *Grant v Sun Shipping Co Ltd* [1948] AC 549 at 563 ; *Dingle v Associated Newspapers Ltd* [1961] 2 QB 162 at 188-189 ; *Thompson v Australian Capital Television Pty Ltd* [1996] HCA 38; 186 CLR 574 at 600 ; *Elayoubi bhnf Kolled v Zipser* [2008] NSWCA 335 at [57] ; *Gett v Tabet* [2009] NSWCA 76; 254 ALR 504 at [367] ; *Sienkiewicz v Greif (UK) Ltd* [2011] 2 WLR 523 at [90] ; *Amaca Pty Ltd v Booth* [2011] HCA 53; 86 ALJR 172 at [70] ; *Strong v Woolworths* [2012] HCA 5; 86 ALJR 267 at [26] ; *Allianz Australia Ltd v Sim* [2012] NSWCA 68 at [41]-[43] , [49] . The carcinoma contracted by the worker is an "indivisible" disease as that expression is used in this context, because once contracted its severity is not affected by the quantity of wood dust that has been or continues to be inhaled or ingested.

Fraser (nee Butcher) v Burswood Resort (Management) Ltd [2012] WADC 175 (18 December 2012) (Stevenson DCJ)

270. At [98] Basten JA in ***Allianz Australia Ltd v Sim*** [2012] NSWCA 68 said:

> The first step in the argument is undoubtedly correct: increase in risk is not to be equated with factual causation. As noted by French CJ in ***Amaca Pty Ltd (under NSW administered winding up) v Booth*** [2011] HCA 53; 86 ALJR 172 at [41] :
>
> > Causation in tort is not established merely because the allegedly tortious act or omission increased a risk of injury. The risk of an occurrence and the cause of the occurrence are quite different things.

Fire & Rescue NSW v Hayman [2012] NSWWCCPD 66 (14 November 2012) (Deputy Bill P Roche)

74. Mr Perry further submitted that Dr Klug used the expression “balance of probabilities” and it was for the Arbitrator to make a determination. Again, this was a surprising submission. It is common practice for medical experts in the Commission to express their opinions in terms of the balance of probabilities and there is nothing wrong with that practice. Expert evidence may be given on factual questions of causation, even where the opinion is expressed in language that is employed in legal analysis in causation (*Allianz Australia Ltd v Sim* [2012] NSWCA 68 Allsop P (Meagher JA agreeing) at [34] [35] , Basten JA at [118] – [121] (Meagher JA agreeing)). It goes without saying that the ultimate decision on liability is for the Commission.

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