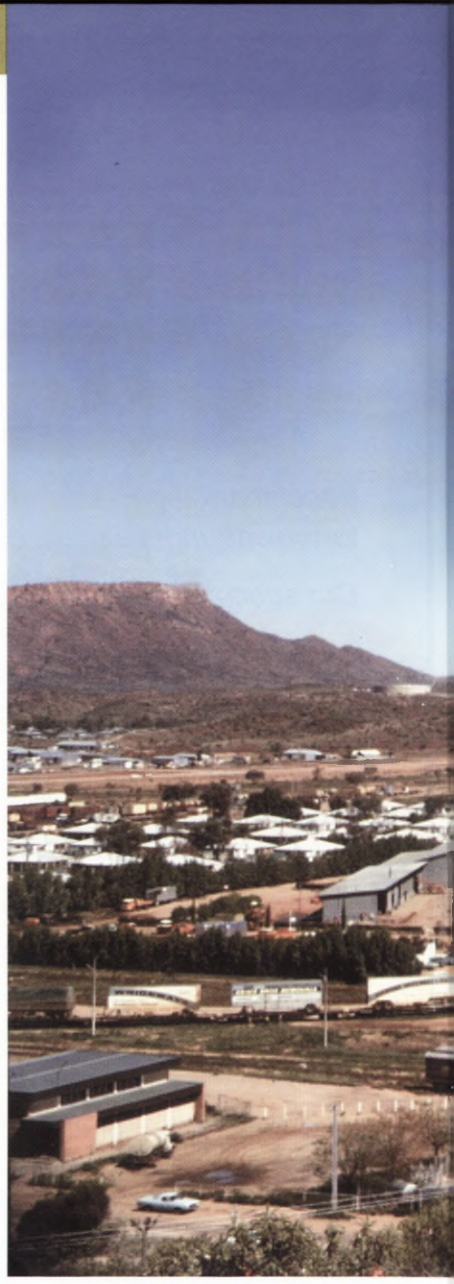
# Causation of dust diseases

By Bernard McHardy



While science is regularly an integral part of any legal enquiry into cause, a finding of causation in law is, of course, not to be equated with scientific proof.

Nobel Prize-winning physicist, Max Planck, wrote in 1932: 'Religion belongs to that realm that is inviolable before the law of causation and therefore is closed to science'. It is, nevertheless, in the nature of humanity to desire answers and certainties and, despite such wisdom from one so celebrated, scientists and creationists still went head to head in the 'monkey trials'.

In the realm of personal injury law, causation is not established by showing that a defendant's act or omission increased the risk of injury to a plaintiff unless the risk has eventuated: *Chapel v Hart.*[1]

As a general principle, scientific evidence employed to demonstrate risk and increased incidence of disease as a consequence of exposure to a noxious substance must suffer the rigour of the legal process before that scientific evidence is accepted as establishing that the substance has contributed to, or caused, the disease.

Such is the ambit of many arguments dealt with in the Dust Diseases Tribunal in the investigation of causation of dust diseases. At its best, medical science can produce emphatic evidence of the causal connection between an agent and a disease, as noted, for example, in the following quote: 'By virtue of its capacity to induce mesothelioma, asbestos ranks among the most clear-cut carcinogens to affect humans. Its role in the genesis of mesothelioma has been demonstrated repeatedly in a large body of experimental and epidemiological evidence.'[2]

For the purposes of legal inquiry, such scientific evidence establishes cause.

## ASBESTOS

When addressing the question of causation of dust diseases, it is necessary and instructive to look at both the nature of the dust and of the disease. For the purposes of this article, I will do this by reference only to asbestos and its medical consequences.

The term 'asbestos' covers a variety of naturally-occurring silicates which can be divided into serpentine (chrysotile or white asbestos) and amphibole (crocidolite or blue asbestos, and amosite or brown asbestos). The amphiboles, by the nature of their sharp, needle-shaped fibres, tend to

*Photograph by the late Mr E C Hood.*

penetrate and have far greater persistence in tissues than chrysotile.

Because of its insulating and fire-resistant properties, uses for asbestos were readily identified 4,000 years ago. The word 'asbestos' is derived from the Greek word for unquenchable.[3] The Romans mined it and the goddess Minerva was recorded as using a lamp with a wick made from the 'linen which is not consumed by fire'.[4]

Despite a plethora of historical references to the use of mineral asbestos, it was not until the surge of modern-day use of the mineral fibre that detection of, and recording of, the incidence of asbestos diseases occurred. Identification of these illnesses and their causes became more readily apparent because of the growth in industry and the extensive and diverse uses to which asbestos was put.

The first diagnosis of asbestosis[5] was reportedly made by Dr H Montague-Murray in 1899 at Charing Cross Hospital of a man who worked in the carding room of an asbestos factory. The patient was the sole survivor of ten who worked in the factory. All the others died at around age 30.[6]

The progression of medical knowledge from that time, and the identification of the problems now universally associated with asbestos, are such that arguments about foreseeability in the Dust Diseases Tribunal are rare. The milestones that mark the progress of knowledge through medical, industrial and scientific publications are regularly raised through the procedural advantage of s25(3) of the *Dust Diseases Tribunal Act* which removes the need to repeat evidence in relation to matters already accepted by the Tribunal in past cases.

Apart from the fatal cancer mesothelioma, asbestos can elicit a variety of pleural lesions known as pleural plaque, benign asbestos pleuritis and diffuse pleural fibrosis. The clinical features of each pleural disorder differs greatly.

Uncomplicated pleural plaques causes no measurable impairment of the pulmonary function, and without impairment the issue of compensation does not arise. Identification of the disease from which an individual suffers, and any nexus to asbestos, are >>

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therefore often matters of contention in proceedings before the Tribunal. It is particularly in the connection between asbestos exposure and cancer that the more vexed problems arise for legal determination.

## GENERAL PRINCIPLES OF CAUSATION

Justice Mason in *Sutherland Shire Council v Heyman*[7] said, 'when there is a duty to take a precaution against damage occurring to others ... breach of the duty may be regarded as materially causing or materially contributing to that damage, should it occur, subject of course to the question whether performance of the duty would have averted the harm'; and 'generally speaking, if an injury occurs within an area of foreseeable risk, then, in the absence of evidence that the breach [of duty] had no effect ... or that the injury would have occurred even if the duty had been performed, it will be taken that the breach ... caused or materially contributed to the injury.'

### Statutory precautions

The House of Lords in *McGhee v National Coal Board*[8] established that an employer's breach of statutory duty to provide showering facilities to enable brickworkers to wash off brick dust was a cause of a worker's industrial dermatitis, because having to cycle home without a shower added materially to his risk that this disease might develop. *Bonnington Castings v Wardlaw*,[9] *Nicholson v Atlas Steel Foundry & Engineering Co Ltd*[10] and *Quinn v Cameron & Robertson Ltd*[11] all dealt with an employer's liability for breach of a statutory duty to remove silica dust generated by industrial processes.

The principles established by these cases are particularly relevant where an employer has taken no steps to minimise a foreseeable risk to his employees and no steps to measure the extent of that hazard.

However, as Justice Mason said in *Bendix v Barnes*,[12] 'the law does not equate the situation where the defendant has materially increased the risk of injury with one where he had materially contributed to the injury'.

## SELTSAM PTY LTD V MCGUINESS

Epidemiological evidence was put under the microscope in the case of *Seltsam Pty Ltd v McGuiness & Anor*.[13] This case was concerned with whether exposure to asbestos caused renal cell carcinoma. The plaintiff succeeded at trial and the principal issue raised in the appeal was the use of epidemiological evidence to prove causation. It was found that causation in an individual case can be established by a process of inference from circumstantial evidence which combines primary facts like 'strands in a cable' rather than 'links in a chain'.[14] In that regard, epidemiological evidence of the effects on populations of exposure to an agent was regarded as circumstantial. Ultimately, it was found that the trial judge had not given appropriate consideration to the quality of the epidemiological evidence, which by a 2:1 majority the Court of Appeal ruled did not support a finding that asbestos exposure caused or materially contributed to the respondent's renal cell carcinoma.

The question as approached by the trial judge involved two questions: whether inhalation of asbestos more probably than not was capable of causing or contributing to the cancer; and, secondly, whether Mr McGuiness's renal cell carcinoma was more probably than not caused, or contributed to, by his inhalation of asbestos. These issues were referred to as 'general causation' and 'specific causation'.

Chief Justice Spigelman, commenting on the trial judge's treatment of the epidemiological evidence, said: 'His Honour made no reference to the epidemiological studies in the context of answering the second question he posed for himself. His discussion of epidemiology occurred in the context of the first question; namely, whether or not asbestos was capable of causing renal cell carcinoma. I am of the view that his Honour did not take into account the strength or quality of the epidemiological evidence in answering the second question – causation in the specific case of the respondent.'[15]

The quality of the epidemiological research on the association between asbestos exposure and renal cell carcinoma was a relevant consideration for the trial judge – one which the Court of Appeal found he failed to take into account.

His Honour was careful to say that a long line of authorities would reject the proposition that a court should not infer causation where scientists, including epidemiologists, would not do so, but it still weighed heavily on the court in the *McGuiness* case that the quality of the epidemiology relied upon was deficient.

Justice Stein, in his dissenting judgment, agreed with the Chief Justice in rejecting the submission that commonsense and intuition have no part to play in considering epidemiological evidence.[16] His assessment of the whole of the evidence led to his view that the trial judge's conclusion was 'well open to him' noting that, apart from the epidemiological evidence, there was evidence of biological plausibility supporting a commonsense approach to causation.

## JUDD V AMACA PTY LTD

In the judgment of Justice Curtis in *Judd v Amaca Pty Ltd*,[17] his Honour was called to decide whether there was a causal nexus between the former carpenter's lung cancer and his past exposure to asbestos, where the plaintiff did not suffer from clinically detectable asbestosis and his lung-fibre burden was unknown. The plaintiff was 63 years old and had been a heavy smoker for over 42 years.

The plaintiff's contention in the case was that his lung cancer was more likely to have been induced by asbestos, either alone or in combination with smoking, than either risk alone; or that all lung cancers which develop after exposure to both cigarette smoke and asbestos fibre are caused by the synergistic effect contributing to the pathological process that resulted in his cancer.

In dealing with the epidemiological evidence, his Honour commented that with regard to the contention of causation by increase in risk: 'Unless a plaintiff can rely upon *other circumstances* that operate to weigh the probabilities in his favour, proof of causation which relies upon epidemiological evidence generally requires proof that his relative risk was greater than two'.[18]

'Relative risk' is a measure of the incidence of a disease in a population exposed to an agent compared to background incidence of the disease in a non-exposed population. A relative risk of two then indicates that the background risk is doubled and that one of every two cases of the disease may be attributable to the agent. On the balance of probability, logic would say that cause is established for the purposes of our civil law only when the relative risk is more than two.

Attention was given by his Honour in his judgment to the level of exposure to asbestos fibre postulated as necessary to double the risk of contracting lung cancer. This involved an examination of a meeting of 'like-minded pathologists, radiologists,

occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists and clinical and laboratory scientists'.[19] This was a group chaired by Professor Henderson, who was an expert called in the *Judd* case. The group formulated criteria in its consensus report, familiarly known as the Helsinki Criteria, for the guidance of occupational health authorities and tribunals in compensating persons suffering from asbestos diseases. >>

The report concluded that accumulative exposure of 25 fibre years (fibres per cubic centimetre per year) led to a rough doubling of relative risk, although at that level asbestosis may not be present or detectable. It also concluded that heavy exposure in the absence of radiologically diagnosed asbestosis is sufficient to increase the risk of lung cancer. The fact that in evidence in *Judd*, Professor Henderson conceded that he did not know why the figure of 25 fibre/ml years was adopted, led Justice Curtis to observe that: 'These observations do not justify the requirement that an expert's "assumed" or "accepted" facts be identified', and he went on to refer to the dicta of Justice Heydon in *Makita (Australia) Pty Ltd v Sprowles.*[20]

His Honour concluded that: 'Unfortunately, the generalities offered by Professor Henderson in possible support of such an inference [that other evidence besides a relative risk of two invites a commonsense conclusion that a 25 fibre/ml year lung cancer should be attributed to asbestos] ... do not satisfy the test of admissibility mandated by *Makita Pty Ltd v Sprowles.*'[20]

By reference to other evidence given, his Honour expressed the opinion that the relative risk of contracting lung cancer in consequence of work such as Mr Judd's, doubled at 50 fibre/ml years of cumulative exposure.[21] In his Honour's view: 'The plaintiff has not persuaded me that upon the probabilities he would have contracted his cancer in the absence of his asbestos exposure.'[22]

## APPORTIONMENT OF CAUSATION

Exposure of a worker to asbestos in the products of competing manufacturers or by a succession of employers is dealt with by the apportionment of liability. At issue in these circumstances is a comparison of the parties' relative culpability, and consideration of relative blameworthiness and of the relevant causal potency of each party's action.

Section 5(2) of the *Law Reform (Miscellaneous Provisions) Act* 1947 relevantly provides that: 'In any proceedings for contribution under this section, the amount of the contribution recoverable from any person shall be such as may be found by the court to be just and equitable having regard to the extent of that person's responsibility for the damage.'

> On the balance of probability, logic would say that cause is established for the purposes of our civil law only when the relative risk is more than two.

In *E M Baldwin & Sons Pty Ltd v Plane*,[23] Judge Curtis concluded: 'The medical evidence indicates that it is not possible to determine which period of employment was the "causative" exposure, and in these circumstances, each of the three defendants must be held liable.'

The plaintiff was awarded damages for having contracted mesothelioma. Liability was apportioned 95 per cent against his employer (Baldwin) and 5 per cent against Jsekarb Pty Ltd, the company which supplied brake blocks containing asbestos to the employer during the latter period of the plaintiff's employment. The asbestos contained chrysotile, which was contaminated by a form of asbestos known as tremolite. Expert evidence established that this was many times less potent than crocidolite asbestos, which was a significant aspect of the plaintiff's work environment in the early phase of his employment by Baldwin.

The findings of liability against each defendant were undisturbed on appeal. However, on apportionment, Acting Justice Fitzgerald (with whose reasons Justice Beazley agreed) concluded that the evidence provided no rational basis for the division of causation between the earlier and later periods of the plaintiff's employment by Baldwin. He said: 'Axiomatically, causation therefore cannot be proportionally allocated between his different work activities or the different amphibole asbestos fibres to which he was exposed. In these circumstances, I consider it impossible to establish that any apportionment is "just and equitable" other than an equal apportionment of responsibility for Mr Plane's damage between Baldwin and Jsekarb.'[24]

Justice Meagher dissented on the apportionment issue. He noted the limited grounds upon which an apportionment order may be disturbed and said: 'His Honour the trial judge gave reasons for his apportionment, and these reasons seem to me far from unjustified. For example, his principal reason: firstly [sic], it is established by the consensus of expert evidence on both sides of the record that chrysotile asbestos is many times less potent than crocidolite asbestos.' Indeed it is. Baldwin for many decades had exposed Mr Plane to the latter, more deadly, asbestos, while Jsekarb had, at worst, exposed Mr Plane to the second, milder non-amphibolic form of asbestos, chrysotile, for a period of no more than 10-15 years. This reason alone should justify his Honour's order.'[25]

In contrast, in the case of *Bitupave Ltd v McMahon & Ors*,[26] the Court of Appeal observed that *Plane* should be read as turning on its own facts which were distinguishable from *Bitupave*.

In *Bitupave* the court determined that the three defendants were found to have contributed materially to the disease and circumstances involving tortious responsibility, but that there were differences as to the length of exposure, the nature of the employment and the state of knowledge about the dangers of asbestos.

Justice Mason said: 'The primary judge was entitled to have regard to these factors in the "rough and ready" apportionment exercise. The manner in

which he exercised the task has not been shown to have involved error.'[27]

Justice Meagher and Acting Justice Cole agreed with the essence of Justice Mason's determination that *Plane* was a factual determination of apportionment limited to the facts of the particular case and did not express any general principle of law.

## CONCLUSIONS

The general principles of causation for the purposes of the law remain as stated by Justice Mason in *Sutherland Shire Council v Heyman*. The particular difficulties that arise in the assessment of evidence from epidemiologists, occupational hygienists and occupational and pulmonary physicians must all be weighed before any judgment can be made that a breach of duty may be regarded as materially causing or materially contributing to any damage suffered.

While an intuitive or commonsense approach may often be necessary in weighing such evidence, a thorough analysis of the merits of any expert evidence or the qualitative value of any epidemiological reports or other statistical data must be made in considering the question of whether a plaintiff's claim overcomes mere possibility and enters the realm of probability.

As for the apportionment of cause, a 'rough and ready' approach in the absence of persuasive subjective factors will still be a necessary and satisfactory legal approach where medical science does not provide more conclusive answers. ■

**Notes:** **1** [1998] 195CLR 232. **2** D W Henderson et al, *Malignant Mesothelioma*, Hemisphere Publishing Corporation, New York, 1992, p9. **3** W K C Morgan & A Seaton eds, *Occupational Lung Diseases*, 2nd ed, W B Saunders, Philadelphia, 1984, pp323-76. **4** W E Cooke; 'Pulmonary Asbestosis', B R Med J 2; 1927, 1024-25. **5** Blakiston's *Gould Medical Dictionary* definition 'diffuse interstitial pulmonary fibrosis due to the prolonged inhalation of asbestos dust'. **6** D W Henderson et al, *Malignant Mesothelioma*, p2. **7** [1985] 157 CLR 424, 464. **8** [1973] 1WLR1. **9** [1956] AC 613. **10** [1957] 1All ER 776. **11** [1957] 1All ER 760. **12** [1997] 42 NSWLR 307 at 316. **13** [2000] 49 NSWLR 262. **14** [2000] NSWCA 29, para 91. **15** [2000] NSWCA 29, para 28 & 29. **16** [2000] NSWCA 29, para 206. **17** [2003] NSW DDT 12 (7 April 2003). **18** [2003] NSW DDT 12, para 9. **19** [2003] NSW DDT 12, para 18. **20** [2001] 52 NSWLR 705, 744. **21** [2003] NSW DDT 12, para 44. **22** [2003] NSW DDT 12, para 118. **23** [1999] Australian Torts Reports 91-499. **24** [1999] Australian Torts Reports 81-499. **25** (at 65,622). **26** [1999] NSWCA 330. **27** [1999] NSWCA 330, para 52.

**Bernard McHardy** *is a partner at McLaughlin & Riordan, Sydney.*
**PHONE** *(02) 9223 2411* **EMAIL** *info@mclaughlin.com.au*