HIGH COURT OF AUSTRALIA

FRENCH CJ, KIEFEL, BELL, KEANE AND NETTLE JJ

ALCAN GOVE PTY LTD

APPELLANT

AND

ZORKO ZABIC

RESPONDENT

Alcan Gove Pty Ltd v Zabic
[2015] HCA 33
Date of Order: 12 August 2015
Date of Publication of Reasons: 7 October 2015
D5/2015

ORDER

Appeal dismissed with costs.

On appeal from the Supreme Court of the Northern Territory

Representation

G M Watson SC with J C Sheller for the appellant (instructed by Bartier Perry)

B W Walker SC with G F Little SC for the respondent (instructed by Shine Lawyers)

Notice: This copy of the Court's Reasons for Judgment is subject to formal revision prior to publication in the Commonwealth Law Reports.

CATCHWORDS

Alcan Gove Pty Ltd v Zabic

Limitation of actions – When cause of action accrues – Negligence – Damage – Statute abolished cause of action unless accrued before 1 January 1987 – Whether compensable damage suffered upon changes to mesothelial cells following exposure to asbestos – Whether mesothelioma inevitable – Relevance of hindsight – Whether "trigger" for development of disease endogenous or exogenous.

Torts – Negligence – Damage – Dust diseases – Mesothelioma – Whether changes to mesothelial cells compensable damage.

Words and phrases – "compensable damage", "endogenous", "exogenous", "hindsight", "mesothelial cell changes", "trigger".

Workers Rehabilitation and Compensation Act (NT), ss 52(1), 189(1).

FRENCH CJ, KIEFEL, BELL, KEANE AND NETTLE JJ. The issue in this appeal was whether the respondent's cause of action in negligence for damages for mesothelioma caused by the inhalation of asbestos fibres in the course of his employment with the appellant between 1974 and 1977 accrued before 1 January 1987. The issue arose because, on 1 January 1987, the substantive provisions of the *Workers Rehabilitation and Compensation Act* (NT) ("the Act") came into force¹. The Act abolished common law actions in negligence with respect to workplace injuries and provided for limited statutory rights to compensation for injured workers. If the cause of action accrued on or after 1 January 1987, it would have been statute barred.

The judge at first instance (Barr J) held that the cause of action did not accrue until the onset of malignant mesothelioma; and, according to the evidence given at trial, that was probably at a point between one and five years before the respondent first experienced symptoms of mesothelioma in 2013 or 2014. On that basis, the judge held that the cause of action did not accrue until well after 1 January 1987².

The Court of Appeal of the Northern Territory (Riley CJ, Southwood and Hiley JJ) reversed the judge's decision³. They found that, with the benefit of hindsight, it was possible to say that the respondent's mesothelial cells were so damaged shortly after inhalation of asbestos fibres between 1974 and 1977 as "inevitably and inexorably" to lead to the eventual onset of the malignant mesothelioma⁴. On that basis, the Court of Appeal concluded that the damage done to the mesothelial cells shortly after inhalation was non-negligible compensable damage sufficient to found a cause of action and that the subsequently developed malignant mesothelioma was part of the damage arising in that accrued cause of action.

The Court of Appeal was correct. On 12 August 2015, this Court made orders dismissing the appeal. These are the reasons for those orders.

- 1 The Act was enacted as the Work Health Act 1986 (NT).
- 2 Zabic v Alcan Gove Pty Ltd (2015) 292 FLR 413.
- 3 Zabic v Alcan Gove Pty Ltd (2015) 294 FLR 376.
- 4 Zabic (2015) 294 FLR 376 at 388 [61].

1

2

3

2.

The facts

5

The expert evidence given at trial was that asbestos fibres are hydrated silicates of aluminium and magnesium which are known to generate oxygen free radicals capable of setting off adverse genetic changes in susceptible cells. When asbestos fibres are inhaled they work their way to the periphery of the lung, eventually through the visceral pleura and ultimately onto the parietal pleura. Mesothelial cells form part of the parietal pleura. They contain "oncogenes" which may be so influenced by methylation and acetylation caused by free radicals as in some cases to lead to the development of abnormal "switches" in the genetic regulation of cell reproduction. Initial molecular changes occur in the mesothelial cells of the pleura soon after inhalation of asbestos fibres. To begin with, those changes are asymptomatic, and otherwise undetectable, and do not in all cases lead to mesothelioma. In cases like the respondent's, however, where they do lead to mesothelioma, the changes typically lie dormant for years, often for decades, until an unknown "trigger" sets off the development of abnormal genetic switches resulting in malignancy and the "domino effect" that culminates in the malignant mesothelial tumour which constitutes mesothelioma.

6

In this case, the respondent inhaled asbestos fibres in the course of his employment between 1974 and 1977 and it was probable that he did not develop a malignant mesothelial tumour until shortly before first experiencing the symptoms of mesothelioma in 2013 or 2014. Nonetheless, based upon the expert evidence concerning the pathology of the disease, it could be inferred that the asbestos fibres inhaled between 1974 and 1977 had then or shortly afterwards (and therefore before 1 January 1987) resulted in initial molecular changes to mesothelial cells which ultimately culminated in the malignant mesothelial tumour. Initially asymptomatic and otherwise undetectable, the changes are likely to have lain dormant until between one to five years before the first manifestation of symptoms. At that point an unknown trigger set off the development of abnormal genetic switches that resulted in malignancy. As will be seen, the evidence of the precise nature of that trigger is decisive in this case. The expert evidence regarding the trigger and the conclusions to be drawn from the evidence are discussed in detail later in these reasons.

The Workers Rehabilitation and Compensation Act

At the relevant times⁵, s 52(1) of the Act provided:

"Subject to section 189, no action for damages in favour of a worker or a dependant of a worker shall lie against:

(a) the employer of the worker;

. . .

7

in respect of:

(d) an injury to the worker".

Section 189(1) provided:

"Where a cause of action in respect of an injury to or death of a person arising out of or in the course of his or her employment arose before the commencement of this section, a claim or action (including a claim or action at common law) in respect of that injury or death may be made, commenced or continued after the commencement of this section as if this Act had never commenced and for that purpose the repealed Act shall be deemed to continue in force."

Section 3(1) relevantly defined "injury" as including a disease, and "disease" as including a physical "ailment, disorder, defect or morbid condition, whether of sudden or gradual development". "Cause of action" was not defined in the Act, but it plainly refers to the "fact or combination of facts which gives rise to a right to sue"⁶.

⁵ These proceedings were commenced on 28 August 2014. The Act was subsequently renamed the *Return to Work Act* (NT): see *Workers Rehabilitation and Compensation Legislation Amendment Act* 2015 (NT).

⁶ Do Carmo v Ford Excavations Pty Ltd (1984) 154 CLR 234 at 245 per Wilson J; [1984] HCA 17.

8

4.

Damage is the gist of the action

The law is clear that actual damage or injury is an essential element of a cause of action in negligence for personal injury. (It is not disputed that the respondent established the other elements of his cause of action, namely, the existence and breach of a duty of care, and that the mesothelioma was caused by the appellant's breach of duty.) What may qualify as actionable damage is, however, a question of fact and degree and ultimately of policy. Kiefel J observed in *Tabet v Gett* that the "damage necessary to found an action in negligence ... is the injury itself and its foreseeable consequences". As Hayne and Bell JJ said in the same case, damage refers to "some difference to the plaintiff [which] must be detrimental" In similar vein, in *Harriton v Stephens*, Crennan J, with whom Gleeson CJ, Gummow and Heydon JJ agreed, said II:

"Because damage constitutes the gist of an action in negligence, a plaintiff needs to prove actual damage or loss and a court must be able to apprehend and evaluate the damage, that is the loss, deprivation or detriment caused by the alleged breach of duty. Inherent in that principle is the requirement that a plaintiff is left worse off as a result of the negligence complained about, which can be established by the comparison of a plaintiff's damage or loss caused by the negligent conduct, with the plaintiff's circumstances absent the negligent conduct."

- Williams v Milotin (1957) 97 CLR 465 at 474; [1957] HCA 83; Wardley Australia Ltd v Western Australia (1992) 175 CLR 514 at 526 per Mason CJ, Dawson, Gaudron and McHugh JJ; [1992] HCA 55; Harriton v Stephens (2006) 226 CLR 52 at 102 [161] per Hayne J, 115 [218] per Crennan J (Gleeson CJ, Gummow and Heydon JJ agreeing at 58 [1]-[4], 113 [208]); [2006] HCA 15.
- 8 See and compare Stapleton, "The Gist of Negligence Part I: Minimum Actionable Damage", (1988) 104 Law Quarterly Review 213; Fleming's The Law of Torts, 10th ed (2011) at 225-226.
- 9 (2010) 240 CLR 537 at 585 [135] (emphasis removed); [2010] HCA 12.
- **10** (2010) 240 CLR 537 at 564 [66]. See also *Rothwell v Chemical & Insulating Co Ltd* [2008] AC 281 at 289 [7] per Lord Hoffmann.
- 11 (2006) 226 CLR 52 at 126 [251].

The effect of previous decisions

9

10

11

12

Over the last thirty years, there have been a number of decisions concerning actionable damage in relation to mesothelioma. They provide a degree of guidance as to relevant considerations. It is necessary, however, to keep in mind that, because the question is essentially one of fact, each case turns on its own facts and circumstances and, therefore, what has been decided in the past may not necessarily be of great assistance in deciding new and different cases.

In *Battaglia v James Hardie & Co Pty Ltd*¹², Vincent J was required to decide whether "pleural thickening" which occurred relatively shortly after the inhalation of asbestos fibres was sufficient to found a cause of action. Pleural thickening is different from the kind of initial mesothelial cell changes which may occur shortly after inhalation of asbestos fibres. It is not malignant and, so far as is known, is not part of any process which leads to malignancy. At most it is an indicium of the possible presence of otherwise undetectable mesothelial cell changes which may lead to mesothelioma. Vincent J ruled that because there was no evidence that the pleural thickening would or even could lead to malignant mesothelioma, it was not compensable damage¹³. Soon after, in *Papadopoulos v James Hardie & Co Pty Ltd*, Kaye J held that pleural plaques, which can lead to pleural thickening, were not sufficient damage to found a cause of action because, at the time of trial, the pleural plaques had not developed into mesothelioma or resulted in other symptoms¹⁴.

There is also a decision of the New South Wales Court of Appeal concerned with the question of whether the mere risk of contracting mesothelioma following inhalation of asbestos fibres is sufficient to amount to compensable damage.

In *Orica Ltd v CGU Insurance Ltd*¹⁵, each of the members of the Court of Appeal (Spigelman CJ, Mason P and Santow JA) concluded that the kind of injury which is suffered by reason of no more than the inhalation of asbestos

- 12 Unreported, Supreme Court of Victoria, 12 March 1987.
- 13 Battaglia unreported, Supreme Court of Victoria, 12 March 1987 at 17-18.
- 14 Unreported, Supreme Court of Victoria, 12 February 1988 at 10-11.
- 15 (2003) 59 NSWLR 14.

13

6.

fibres is not compensable damage and therefore that a cause of action does not accrue unless and until mesothelioma develops. Spigelman CJ expressed his conclusion thus¹⁶:

"Damage is the gist of the action in negligence. The 'injury' occasioned at the time of penetration of the lung by a fibre, if it be injury within the meaning of the policy at all, which I doubt, is so negligible in and of itself, as distinct from its potential, that it does not constitute damage that is compensable at common law."

Mason P, to the same ultimate effect, specifically rejected the notion that, because it was possible to look back with the benefit of hindsight and say that the injury suffered upon or shortly after the inhalation of asbestos fibres had resulted in mesothelioma, the claimant's cause of action had accrued at or shortly after inhalation¹⁷:

"With the benefit of hindsight we know as a fact (because it is shown as more probable than not) that the worker, Mr Dunstan, suffered the disease-inducing injury between 1959 and 1961 when he was employed at the ICI plant. I agree with Santow JA's analysis as to the process whereby it was established that the risk of injury to which he was negligently exposed came home during this period, with the consequence that the negligent employer fell under a potential liability to compensate the worker, his estate and dependants according to tort law. But that liability remained inchoate, in the eyes of tort law, because damage is the gist of the relevant cause or causes of action.

The worker did not sue or recover damages on the debatable basis that the chance or fear of contracting mesothelioma was the damage suffered.

Mr Dunstan might have suffered devastating injury or been killed, perhaps due to a third party's negligence, before the ingested asbestos fibre triggered the manifestation of his disease that is recognised as the accrual of his tortious cause of action stemming from the negligent exposure at the ICI plant. This did not occur."

¹⁶ Orica (2003) 59 NSWLR 14 at 23 [32].

¹⁷ *Orica* (2003) 59 NSWLR 14 at 28 [72]-[74] (citations omitted).

14

Santow JA referred to the possibility of a claimant bringing an action for increased risk of contracting mesothelioma but rejected the idea as being against the weight of English and Australian authority. After referring to Professor Stapleton's observations as to the hostility of English judges to the idea of allowing "pure-loss-of-a-physical-chance" as actionable damage¹⁸, his Honour continued¹⁹:

"The weight of authority in Australia is to similar effect, though there may come a time and case as in the United States where it may be possible to demonstrate actual damage, in the form of some psychological condition induced by anxiety, or even increased life insurance premiums or other discernible disadvantage by reason of a greater risk of contracting a disease like mesothelioma from past exposure.

So far it has been held in Australia that where there has been an inhalation of asbestos that has led to pleural thickening of the lung at the time of trial, but which has caused no physical discomfort or disability, with only the potential for more serious developments, those physiological changes wrought to that stage could not be said to have amounted to an actionable injury because of the lack of any established harm. The potential for more harmful developments could not alter that situation."

15

The position in the United Kingdom appears to be similar²⁰. In *Barker v Corus UK Ltd*²¹, Lord Hoffmann proposed the view that, given the exception to the ordinary rules of causation in relation to cases of mesothelioma which was recognised in *Fairchild v Glenhaven Funeral Services Ltd*²², it would be appropriate henceforth to treat the risk of contracting mesothelioma as forming the gist of the action²³. But his Lordship's view has since been repudiated by a

Stapleton, "Cause-in-Fact and the Scope of Liability for Consequences", (2003) 119 *Law Quarterly Review* 388 at 424, quoted in *Orica* (2003) 59 NSWLR 14 at 54 [149].

¹⁹ *Orica* (2003) 59 NSWLR 14 at 54 [149]-[150] (citations omitted).

²⁰ *Rothwell* [2008] AC 281.

²¹ [2006] 2 AC 572.

²² [2003] 1 AC 32.

²³ Barker [2006] 2 AC 572 at 589-591 [35]-[39].

16

17

8.

majority of the Supreme Court of the United Kingdom in *Durham v BAI (Run off) Ltd*²⁴. In this country, where the *Fairchild* exception has not to date been recognised²⁵, there is still more reason to reject it.

It remains to mention *Martindale v Burrows*²⁶, in which Derrington J held that initial mesothelial cell changes which led to mesothelioma amounted to compensable damage. His Honour reasoned that, because it was possible to look back in hindsight on the basis of the evidence and infer that the initial mesothelial cell changes occurred shortly after the inhalation of asbestos fibres, and that they were the initial step in a natural progression which led inexorably to the mesothelioma which the plaintiff had developed by the time of trial, the initial cell changes were compensable damage²⁷.

In summary, therefore, the effect of the previous decisions mentioned to this point appears to be that:

- (1) The mere risk of contracting mesothelioma which arises upon the inhalation of asbestos fibres is not compensable damage, because the risk may not eventuate²⁸.
- (2) Pleural plaques or pleural thickening, which may occur shortly after inhalation of asbestos fibres, although a form of physical injury, are not compensable damage because they are asymptomatic and there is not, or at least there was not at the time of the relevant decisions, evidence that

- **26** [1997] 1 Qd R 243.
- **27** *Martindale* [1997] 1 Od R 243 at 245.
- **28** *Durham* [2012] 1 WLR 867; [2012] 3 All ER 1161.

^{24 [2012] 1} WLR 867 at 895-896 [64]-[65] per Lord Mance JSC (with whom Lord Kerr of Tonaghmore JSC agreed), 904 [77] per Lord Clarke of Stone-cum-Ebony JSC, 907 [90] per Lord Dyson JSC, cf at 915-917 [123]-[130] per Lord Phillips of Worth Matravers PSC; [2012] 3 All ER 1161 at 1193-1194, 1202-1203, 1205, 1213-1215.

²⁵ See *Amaca Pty Ltd v Booth* (2011) 246 CLR 36 at 58 [52]-[53] per French CJ, 66-67 [80]-[82] per Gummow, Hayne and Crennan JJ; [2011] HCA 53.

the pleural plaques or thickening had any potentiality for harm, whether considered on its own or in conjunction with some other process²⁹.

- (3) The risk of contracting mesothelioma to which a claimant is exposed upon the inhalation of asbestos fibres does not become compensable damage by reason only that, with the benefit of hindsight, it is possible to say that the risk has eventuated and therefore that the inhalation of asbestos fibres caused the claimant's mesothelioma³⁰.
- (4) Nevertheless, the kind of mesothelial cell changes which sometimes occur shortly after the inhalation of asbestos fibres may be regarded as compensable damage if, in the case of a claimant who is suffering from mesothelioma, and so with the benefit of hindsight, it can be seen that those mesothelial cell changes were the beginning of a continuum that led inexorably to the onset of mesothelioma³¹.

The judgment at first instance

18

The judge at first instance characterised the issue as being "the same" as the issue in *Orica*. He also took the view that the medical evidence and other facts were "essentially the same" as in *Orica*. Consequently, he treated *Orica* as "persuasive" and on that basis held that the initial mesothelial cell changes were not compensable damage³²:

"All three members of the Court [in *Orica*] concluded that the employee had not suffered damage compensable at common law until such time as he suffered the onset of mesothelioma. Until that time, 'the tort was not complete'.

•••

²⁹ Battaglia unreported, Supreme Court of Victoria, 12 March 1987; Papadopoulos unreported, Supreme Court of Victoria, 12 February 1988.

³⁰ *Orica* (2003) 59 NSWLR 14.

³¹ *Martindale* [1997] 1 Qd R 243.

³² Zabic (2015) 292 FLR 413 at 431-432 [68], [71].

19

20

10.

Because the plaintiff had not suffered damage prior to 1 January 1987, his cause of action in negligence for contracting malignant mesothelioma had not arisen before that date."

The difficulty with that approach, however, is that neither the issue nor the evidence in *Orica* was precisely the same as in this case. The issue in *Orica* was framed as being whether, under an insurance contract, the insured was liable to an employee upon that employee inhaling asbestos fibres. The focus of the judgments, and the issue upon which Santow JA differed from Spigelman CJ and Mason P, was whether the term "liability" encompassed an inchoate or potential liability or, alternatively, a liability that would necessarily arise in the future³³. The Court appeared to assume, rather than decide, that "damage" at common law did not occur until the onset of mesothelioma³⁴. So far as appears, there was no evidence in *Orica* nor any consideration of whether the kind of initial molecular

mesothelial cell changes which occurred in this case amount to compensable damage if they may be seen in hindsight to have led inexorably to mesothelioma.

The reasoning of the Court of Appeal

In contrast, the Court of Appeal characterised the issue, correctly, as being whether the initial mesothelial cell changes which in hindsight could be seen to have led inexorably to mesothelioma amounted to compensable damage. After noting the decision of the House of Lords in *Cartledge v E Jopling & Sons Ltd*³⁵, which held that a plaintiff's cause of action for damages for pneumoconiosis had accrued before the pneumoconiosis could be detected, their Honours continued³⁶:

"Determining whether a cause of action has arisen involves an assessment of objective fact, rather than an assessment of the subjective capacity of a plaintiff to muster proof.

It follows that hindsight is permitted in determining when a cause of action accrues. The aim is to ascertain when relevant facts, namely the

³³ See *Orica* (2003) 59 NSWLR 14 at 55 [154] per Santow JA.

³⁴ *Orica* (2003) 59 NSWLR 14 at 23 [32] per Spigelman CJ, 28 [71]-[74] per Mason P, cf at 55 [153] per Santow JA.

³⁵ [1963] AC 758.

³⁶ Zabic (2015) 294 FLR 376 at 385-386 [46]-[49] (footnote omitted).

presence of compensable damage, objectively came into existence. A Court should not be limited to ascertaining whether relevant facts were provable or discoverable at a particular time. For example, the outcome in *Cartledge* would not have been possible without the Court using contemporary medical evidence to infer when undetectable damage first occurred in the past. Hindsight is frequently employed when one is endeavouring to ascertain the cause or causes of an injury or damage which does not become manifest until some later time.

In the present case we consider that hindsight can be used to establish that there was compensable damage, namely changes to the mesothelial cells, prior to 1987. The toxic carcinogen amphibole asbestos had lodged in the [respondent's] lungs and caused genetic change leading to aberrant and abnormal cell growth which culminated many years later into malignant mesothelioma. The cause of action arose when the nonnegligible damage was first suffered. The subsequent mesothelioma is part of the damage arising in the accrued cause of action.

That damage was no less real, significant and compensable than it would have been had there been medical investigative technologies available at the time that could have identified the damage."

The Court of Appeal also rejected the idea that damage consisting of the initial mesothelial cell changes was no more than contingent or prospective. In contrast to a case like *Wardley Australia Ltd v Western Australia*³⁷, in which liability under a guarantee remained contingent until and unless a future event occurred, and so was not compensable, the Court of Appeal held³⁸:

"This was not the case here. Although the medical evidence was to the effect that a person with abnormalities in the mesothelial cells may or may not acquire malignant mesothelioma, the [respondent's] condition was such that the cells would so develop. That conclusion is now established, albeit with the benefit of hindsight.

• • •

21

³⁷ (1992) 175 CLR 514.

³⁸ Zabic (2015) 294 FLR 376 at 387-388 [58]-[61].

12.

[W]e consider that the [respondent] sustained compensable damage at the time when such inhalation caused changes in his mesothelial cells. According to the medical evidence these changes began to occur very soon after the [respondent's] exposure to asbestos, and prior to 1987.

Even though such changes were not symptomatic, and even if such changes may not have been discoverable by medical investigation methods available then, or even now, the subsequent development of the [respondent's] malignant mesothelioma establishes that the damage to the [respondent's] mesothelial cells, prior to 1987, was material damage, and thus compensable. That damage inevitably and inexorably led to the onset of malignant mesothelioma."

The nature of the trigger

22

An inference, based on the fact that mesothelioma has occurred, that there must have been earlier initial mesothelial cell changes which inevitably and inexorably led to the mesothelioma logically implies an anterior inference that, once the initial mesothelial cell changes occurred, they were bound to lead to the mesothelioma (assuming only that the respondent did not die of other natural or extraneous causes before the mesothelioma developed).

23

As was previously noticed, initial mesothelial cell changes do not develop into mesothelioma in the absence of a "trigger". It follows that, in order for the Court of Appeal to infer that the initial mesothelial cell changes which occurred in this case were bound inevitably and inexorably to lead to mesothelioma, it was necessary for the Court of Appeal to be able to infer that, once the initial mesothelial cell changes had occurred, the trigger already existed or was otherwise bound to occur.

24

Logically, whether it can be posited that a trigger exists or is bound to occur depends on the nature of the trigger. If the trigger is exogenous, which is to say outside the cell, the point at which the initial mesothelial cell changes become bound to lead to mesothelioma will depend on the nature and timing of the trigger.

25

Suppose, for the sake of illustration, that the trigger is in the nature of another carcinogenic substance or effect that affects mesothelial cells. To take the case of a claimant who is suffering from mesothelioma where it appears that the trigger was the consequence of cigarette smoking which the claimant did not take up until after the occurrence of initial mesothelial cell changes, the earliest point at which the mesothelial cell changes could have become bound to lead to

13.

the mesothelioma would have been when the claimant took up cigarette smoking. Similarly, in the case of a claimant who is suffering from mesothelioma where it appears that the trigger was the consequence of radiation to which the claimant was exposed after the initial mesothelial cell changes, the earliest point at which the mesothelial cell changes could have become bound to lead to the mesothelioma would have been when the claimant was so exposed. Until and unless the trigger occurred it would be no more than a possibility and, therefore, consistently with *Wardley*, the initial mesothelial cell changes would not be compensable damage.

26

By contrast, where evidence establishes that the trigger is endogenous, which is to say a state of affairs inside the cells which creates an inherent predisposition or susceptibility to mesothelioma, it may logically be inferred that once the initial mesothelial cell changes occurred they were thenceforth bound to lead to mesothelioma.

27

Here it appears that the Court of Appeal proceeded on the basis that the trigger was endogenous. Their Honours inferred from the fact that the respondent is now suffering from mesothelioma that he must have experienced initial mesothelial cell changes shortly after the inhalation of asbestos and that, because he had an inherent predisposition or susceptibility to mesothelioma, those initial mesothelial cell changes were from the moment of their occurrence bound to lead inevitably and inexorably to mesothelioma.

28

Based on the evidence adduced at trial, that conclusion was correct. As counsel for the respondent submitted, although neither of the experts whose reports were in evidence specifically stated that the trigger was endogenous, it is implicit in each report that the trigger was an endogenous cytogenic process within the cells. That appears most clearly in Professor Allen's report, with which Dr Edwards stated his agreement, in the following passage:

"As with many cancers, the carcinogen (here asbestos) has an adverse impact on the cellular makeup of the tissues exposed to the carcinogen, and this sets off cellular and nuclear changes in the genes of the tissue, which lie dormant for some years until a trigger (often unknown), which leads to the subsequent development of [the] tumour ...

It is thought that asbestos fibres which are hydrated silicates of aluminium and magnesium generate oxygen free radicals ... which are know[n] to have an adverse impact on the genetic makeup of susceptible cells, namely mesothelial cells, and hence lead to the subsequent development of malignant mesothelioma.

14.

I therefore see no intellectual or epidemiologic problem in accepting the fact that his exposure to asbestos in the 1970s led to the development of mesothelioma decades later. ...

In individual cases, it is not always known what triggers off the abnormality, ie what flicks the switch, which in turn leads to the tumour.

Our knowledge of the cytogenetics of carcinogenesis, including of oncogenes, is not sophisticated or precise enough to point to a particular event which occurs in one particular cell, on one particular day, to give rise to a malignancy."

29

The natural and ordinary meaning of the references to "cytogenetics" (scil, the study of the genetics of cells) and to "oncogenes" (scil, genes with the potential to transform a cell in which they are contained into a tumour cell), and the explication of what occurs in "one particular cell, on one particular day", is that the trigger was considered to be within the cell. And although that conclusion was prefaced on what "it is *thought* that asbestos fibres" do, the subsequent statement that there was no hesitation in accepting "intellectually" (scil, logically) the connection between the initial cell changes and the subsequent malignancy bespeaks what was logically and epidemiologically considered to be probable. The stated uncertainty as to what flicks the "switch" is beside the point. It is plain that what is described is a cytogenic process which occurs within an oncogene within a person's cells and ultimately perhaps in "one particular cell" on "one particular day" without external stimulus.

30

As counsel for the respondent submitted, that conclusion is fortified by the appellant's decision not to cross-examine either expert at trial, not to call other expert evidence to contradict them, not otherwise to suggest at trial or on appeal that the trigger was not endogenous, and not to contend that the trigger was in any way in the nature of a *novus actus interveniens*.

31

That is not to say that the appellant thereby waived the need for the respondent to prove that the initial mesothelial cell changes were from the time of their occurrence, or at least from before 1 January 1987, bound to result in mesothelioma. Unlike a statute of limitation properly so called (which prevents the enforcement of an existing cause of action and so can be waived), the effect of s 52(1) of the Act read in conjunction with s 189(1) was to prevent a cause of action arising unless it arose before 1 January 1987. This means that the burden of proof was on the respondent to prove that his cause of action accrued before

1 January 1987 and nothing in the appellant's conduct of its case could have relieved him of that requirement³⁹.

Nevertheless, although the appellant was incapable of waiving proof that the cause of action accrued before 1 January 1987, the appellant was able by the conduct of its case at trial and on appeal impliedly to admit or not dispute facts pertinent to proof. And the fact that the trial and appeal proceeded as they did shows that the appellant did not dispute that the trigger was an endogenous cytogenic process within oncogenes within the cell without any suggestion of exogenous stimuli. In effect, it seems not to have been in issue that the trigger was endogenous.

Granted, the trial judge did state at one point in his reasons that, as at 1 January 1987, the respondent did not suffer from any reduction in life expectancy. Taken alone, that observation might be thought to suggest the judge was not persuaded that, as at 1 January 1987, the trigger was bound to occur; and perhaps for the reason that the judge was not persuaded that the trigger was endogenous. But, as counsel for the respondent submitted, read in context, the judge's observation concerning life expectancy is to be seen as directed only to the dramatic reduction in life expectancy which occurred once the respondent began to experience the symptoms of mesothelioma.

So much is confirmed by the fact, noted by the Court of Appeal, that the judge approached the case as if the issues were as in *Orica* and so eschewed any consideration of whether the changes in the respondent's mesothelial cells following his inhalation of the asbestos fibres were bound to lead inevitably and inexorably to the mesothelioma which later resulted.

Is the risk of mesothelioma sufficient to constitute compensable damage?

Much of the appellant's argument before this Court was directed to whether an initial mesothelial cell change is productive of such a risk of resulting mesothelioma that it should for that reason be considered to be compensable damage. Counsel for the appellant placed heavy emphasis on the decisions earlier referred to, particularly *Orica*, which establish that a risk of mesothelioma

33

32

34

35

³⁹ See *R v McNeil* (1922) 31 CLR 76 at 96 per Knox CJ and Starke J, 100-101 per Isaacs J; [1922] HCA 33; *The Commonwealth v Verwayen* (1990) 170 CLR 394 at 425 per Brennan J; [1990] HCA 39; *Western Australia v Wardley Australia Ltd* (1991) 30 FCR 245 at 259.

36

37

38

39

40

16.

is not compensable damage. He urged the Court to adhere to that view and submitted that any departure from it would likely be productive of widespread and unpredictable ramifications in the field of personal injury cases.

Those submissions, however, were essentially misdirected. Although the trial judge may have conceived of the case as one of risk, it is clear that the Court of Appeal approached the matter on the basis that it was not the risk of mesothelioma but rather the physical injury constituted of the initial mesothelial cell changes that amounted to compensable damage sufficient for the respondent's cause of action to accrue.

For present purposes, it may be accepted that, without more, a risk of developing a compensable personal injury cannot sustain a cause of action in negligence for damages for personal injury. It is only when and if the risk eventuates that compensable damage is suffered and, therefore, it is only then that the cause of action in negligence accrues. To borrow Mason P's illustration in $Orica^{40}$, until and unless that occurs, it is possible that a claimant might suffer some other devastating injury causing death or be killed due to a third party's negligence.

It may also be accepted, as the respondent conceded at trial, that at the time when the initial mesothelial cell changes occurred, and therefore as at 1 January 1987, the respondent had no way of knowing whether any such changes had occurred and therefore could not then have established the existence of a cause of action.

More generally, it may be said that, given the current state of medical knowledge disclosed by the evidence, it would be impossible to say that the inhalation of asbestos fibres is bound to lead to mesothelioma until and unless it does in fact lead to mesothelioma, and, therefore, as the law now stands, it would be impossible to sustain a proceeding on a cause of action for negligence for damages for mesothelioma until and unless there are symptoms of mesothelioma.

For present purposes, however, all of that is essentially irrelevant. The question here is not the time at which sufficient evidence first became available to establish the existence of a cause of action or, therefore, when the respondent could first have initiated a proceeding for damages based on that cause of action.

As Latham CJ observed in *Willis v The Commonwealth*⁴¹, in a statement which was adopted by Wilson, Toohey and Gaudron JJ in *Johnson v Perez*⁴²:

"[W]here the extent and character of what would at one time be described as prospective injury depends upon the happening or non-happening of a particular event and that event has in fact happened, it is unnecessary to speculate as to whether or not this event might happen and, if so, when. In such a case prospective damage ... has become actual."

The question is whether it could be inferred in hindsight that a cause of action had accrued before it could have been detected.

In point of principle, there is no reason why that could not be inferred. As was established in *Cartledge*⁴³, there is nothing illogical or otherwise exceptionable about drawing an inference after symptoms of a disease first appear that, because of what is known of the aetiology and pathology of the disease, the disease is likely to have begun at an earlier point of time when there were no symptoms or other means of detecting its presence.

On the evidence which was available in this case, there is also no reason in fact why it could not be inferred that there were initial molecular changes in the mesothelial cells which preceded the appearance of symptoms of mesothelioma, and that those initial cell changes led inevitably and inexorably to mesothelioma.

Counsel for the appellant called in aid the decision of the Court of Session in *Brown v North British Steel Foundry Ltd* that a cause of action for damages for pneumoconiosis was not shown to have accrued before the date on which an applicable limitation period was first introduced in Scotland⁴⁴. He submitted that it established a principle that a cause of action in negligence for damages for personal injury the result of respiratory disease does not accrue until and unless

- **41** (1946) 73 CLR 105 at 109; [1946] HCA 22.
- **42** (1988) 166 CLR 351 at 368-369; [1988] HCA 64.
- 43 [1963] AC 758; cf *Brown v North British Steel Foundry Ltd* 1968 SC 51 at 64-65 per Lord President Clyde; *Rothwell* [2008] AC 281 at 311 [86] per Lord Rodger of Earlsferry.
- **44** 1968 SC 51.

41

42

43

44

45

46

18.

the symptoms of the disease first appear. But, properly understood, that is not the effect of that decision. On the evidence adduced in that case, it was not possible to say that pneumoconiosis inevitably resulted from the inhalation of silica or, although it had in fact resulted, to say when it had begun. As Lord Migdale observed⁴⁵:

"It was clear on the evidence that pneumoconiosis does not inevitably follow on the inhalation of noxious dust. Accordingly the deceased could not have raised an action founded on the bare averment that he had been exposed to noxious dust. He would also have to aver that as a result of that exposure he had contracted pneumoconiosis. *There is nothing to show that he had it at that time.*"

Time of accrual of cause of action

It is trite law that a tortfeasor must take the victim of the tort as the victim is found to be⁴⁶. Hence, if as a result of an employer's negligence an employee is caused to inhale asbestos fibres and, due to the employee's predisposition to mesothelioma, the inhalation of fibres results in mesothelioma, the employer will be held liable for the damage thereby inflicted.

The question then is whether, as a matter of law, there is any reason why the initial changes in the mesothelial cells which it could be inferred were bound from the time of their onset to lead inevitably and inexorably to the mesothelioma from which the respondent now suffers should not be seen as compensable damage sufficient for the respondent's cause of action in negligence to have accrued at that point.

It assists to answer that question to consider what the position would have been if, at the time the initial mesothelial cell changes occurred, there had been evidence available to establish that they had occurred and that, because of the

45 1968 SC 51 at 70 (emphasis added).

46 Bourhill v Young [1943] AC 92 at 109-110 per Lord Wright; Smith v Leech Brain & Co Ltd [1962] 2 QB 405 at 414 per Lord Parker CJ, approved in Mount Isa Mines Ltd v Pusey (1970) 125 CLR 383 at 406 per Windeyer J; [1970] HCA 60; March v E & M H Stramare Pty Ltd (1991) 171 CLR 506 at 534 per McHugh J; [1991] HCA 12; Tame v New South Wales (2002) 211 CLR 317 at 386 [203] per Gummow and Kirby JJ (approving Windeyer J in Pusey), 413 [279] per Hayne J; [2002] HCA 35.

respondent's predisposition to mesothelioma, they were bound inevitably and inexorably to lead to mesothelioma.

47

In those circumstances, the respondent would have had a cause of action in negligence for damages for personal injury caused by the inhalation of asbestos fibres, which was bound to lead to mesothelioma. The malignant tumour would not have begun at that point and therefore there would remain a chance that the respondent would die from other causes before the tumour began. But, even so, the fact that the respondent would otherwise be bound to die from mesothelioma would be sufficient to found a cause of action in negligence for damages for loss of expectation of life⁴⁷; and clearly, if the malignant tumour began before the matter came to trial, the respondent would be entitled to add to his claim for damages the fact that the tumour had begun⁴⁸.

48

Parity of reasoning dictates the same result here. Given that with the benefit of hindsight it can be seen that initial mesothelial cell changes occurred shortly after the respondent's inhalation of asbestos fibres, and that they were bound to and did lead inevitably and inexorably to the malignant mesothelioma from which he now suffers, the respondent's cause of action in negligence accrued when those initial mesothelial cell changes occurred and, as the Court of Appeal held, damages for the mesothelial tumour from which he now suffers are recoverable in that cause of action.

49

As a final observation, it does not detract from that conclusion that time may run under statutes of limitation against persons who have been exposed to asbestos fibres but who have not yet contracted mesothelioma or another disease as a result. Their position will be protected by statutes of limitation which, in all States and Territories, either set the limitation periods for personal injury by reference to the time at which a cause of action becomes discoverable or provide for postponement of limitation periods until after the time when the material facts can reasonably be ascertained by the plaintiff⁴⁹.

⁴⁷ See, eg, Flint v Lovell [1935] 1 KB 354; Rose v Ford [1937] AC 826; Benham v Gambling [1941] AC 157; Skelton v Collins (1966) 115 CLR 94; [1966] HCA 14.

⁴⁸ See, eg, *O'Brien v McKean* (1968) 118 CLR 540 at 545 per Barwick CJ; [1968] HCA 58; *Baker v Willoughby* [1970] AC 467 at 490-491 per Lord Reid.

⁴⁹ Limitation Act 1985 (ACT), s 16B; Limitation Act 1969 (NSW), Pt 2, Div 6; Pt 3, Divs 3-4; Limitation Act (NT), s 44; Limitation of Actions Act 1974 (Q), ss 30-31; Limitation of Actions Act 1936 (SA), s 36(1a); Limitation Act 1974 (Tas), ss 5-5A; (Footnote continues on next page)

20.

In light of the result reached, it is not necessary to consider the respondent's notice of contention.

Conclusion

For these reasons, the appeal was dismissed.

Limitation of Actions Act 1958 (Vic), Pt IIA; Limitation Act 2005 (WA), s 55 (general personal injury), s 56 (injury attributable to the inhalation of asbestos).