

*Zabic v Alcan Gove Pty Ltd* [2015] NTSC 1

PARTIES: ZABIC, Zorko

v

ALCAN GOVE PTY LTD  
(ACN 000 453 663)

TITLE OF COURT: SUPREME COURT OF THE  
NORTHERN TERRITORY

JURISDICTION: SUPREME COURT OF THE  
NORTHERN TERRITORY  
EXERCISING TERRITORY  
JURISDICTION

FILE NO: 80 of 2014 (21439533)

DELIVERED: 2 January 2015

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JUDGMENT OF: BARR J

**CATCHWORDS:**

TORTS – Negligence – claim for damages – duty of care – asbestos – when damage sustained – ingestion of asbestos fibres over 3-4 years’ employment – symptomless physiological changes – recent onset of mesothelioma after long latency period – cause of action did not arise until actual damage sustained – damage not sustained until onset of mesothelioma – plaintiff’s claim statute-barred – claim dismissed – *Workers Rehabilitation and Compensation Act 1986* (NT), s 52, s 189.

*Limitation Act 1981* (NT) s 44, s 44(2), s 44(7)(a).

*Workers Rehabilitation and Compensation Act 1986* (NT) s 3(1), s 52, s 189, s 189(1)

*CAL No 14 Pty Ltd v Motor Accidents Insurance Board; CAL No 14 Pty Ltd v Scott* (2009) 239 CLR 390; *Cartledge and Ors v E Jopling & Sons Ltd* [1963] AC 758; *Do Carmo v Ford Excavations Pty Ltd* (1984) 154 CLR 234;

*GRE Insurance Ltd v Bristile Ltd* (1991) 5 WAR 440; *Farah Constructions Pty Ltd v Say-Dee Pty Ltd* (2007) 230 CLR 89; *Wilson v Rigg* [2002] NSWCA 246; *Williams v Milotin* (1957) 97 CLR 465; *Wyong Shire Council v Shirt* (1980) 146 CLR 40, referred to.

*Martindale v Burrows* (1996) 1 Qd R 243; *WorkCover Authority (NSW) v Chubb Australia Ltd* (2000) 20 NSWCCR 614, considered

*Orica Ltd & Anor v CGU Insurance Ltd* (2003) 59 NSWLR 14, followed

John Fleming, Carolyn Sappideen and Prune Vines, *Fleming's The Law of Torts* (Lawbook Co, 10<sup>th</sup> ed, 2011), 22

## **REPRESENTATION:**

### *Counsel:*

Plaintiff:	G Little QC
Defendant:	M Crawley

### *Solicitors:*

Plaintiff:	Ward Keller (town agents for Shine Lawyers)
Defendant:	Cridlands MB (town agents for Bartier Perry)

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IN THE SUPREME COURT  
OF THE NORTHERN TERRITORY  
OF AUSTRALIA  
AT DARWIN

*Zabic v Alcan Gove Pty Ltd* [2015] NTSC 1  
No. 80 of 2014 (21438533)

BETWEEN:

**ZORKO ZABIC**  
Plaintiff

AND:

**ALCAN GOVE PTY LTD**  
**(ACN 000 453 663)**  
Defendant

CORAM: BARR J

REASONS FOR JUDGMENT

(Delivered 2 January 2015)

**Introduction**

- [1] The plaintiff is a 74 year old man who is dying of malignant mesothelioma, an aggressive cancer affecting the membrane lining (pleura) of his lungs. He probably has only six months to live.
- [2] The plaintiff claims damages for the defendant's negligence, to include general damages for pain and suffering and loss of amenities of life, damages for loss of expectation of life, damages for past and future gratuitous care, and special damages past and future.<sup>1</sup>

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<sup>1</sup> Particulars of Claim dated 6 November 2014.

- [3] It is not necessary for me to consider and assess the quantum of damages because the parties have agreed that, if the plaintiff is otherwise successful, his damages should be \$425,000, inclusive of special damages and inclusive of interest to the date of judgment, plus costs.
- [4] I am satisfied on the balance of probabilities that the plaintiff's mesothelioma was caused by unprotected exposure to asbestos dust and fibres during the time he worked for the defendant at its Nhulunbuy alumina refinery, from about 1974 to late 1977. I am also satisfied on the balance of probabilities that the plaintiff's mesothelioma was caused by the negligence of the defendant.
- [5] Unfortunately, however, for reasons which I will later explain, the plaintiff's claim is statute-barred and cannot succeed.

### **The plaintiff's case**

- [6] The plaintiff was born in Bosnia, then a part of Yugoslavia, on 13 March 1940. After leaving primary school he worked on the family fruit and vegetable farm until he turned 18, after which he completed three years' national service in the navy of the former Yugoslavia. He then worked as a stevedore, before successfully applying to emigrate to Australia. He arrived in Sydney in June 1970 and subsequently worked for a manufacturer of wiring and cables in Newcastle for about two years. He then went prospecting for opals on the New South Wales/Queensland border for about nine months before moving to the Northern Territory to work for Nabalco

(as the defendant was then known) at its alumina refinery near Nhulunbuy, on the Gove peninsula.

- [7] The plaintiff was employed as a manual labourer at the defendant's refinery from about 1974 until the end of 1977. He worked in a number of 'zones' in the course of his employment, including the steam power station zone and the precipitation zone. He worked as a member of the 'yard gang', comprising some 12 to 18 cleaners/labourers. His work duties required him to assist with running repairs and general cleaning duties, scaffold erection, and cleaning and mucking out of the large treatment and storage tanks located in the various zones at the refinery.
- [8] The plaintiff regularly carried out running repairs and maintenance of the extensive network of pipelines at the refinery. Asbestos lagging was used extensively to insulate pipes through which hot and cold water, fuel, chemicals and slurry would pass, but in particular the pipes in the steam power station zone. The power station housed a number of boilers for generating steam power, and often steam pipes would 'blow' or leak. On such occasions (or if a general overhaul of a pipe were required), the plaintiff as a member of the yard gang would attend and remove the asbestos lagging to expose the pipe, to enable qualified tradesmen to then repair or renew the pipe.
- [9] The plaintiff provided a detailed description of his manual labour duties which was not substantially challenged by the defendant at trial. The

plaintiff's evidence was contained in a lengthy affidavit, and evidence was also taken on commission before an examiner on 29 October 2014, on which occasion the plaintiff was cross-examined. The plaintiff's affidavit and an audio-visual recording of his examination and cross examination on 29 October 2014 were tendered at trial without objection.<sup>2</sup>

[10] The plaintiff referred in his evidence to the extensive use of asbestos lagging on the piping throughout the alumina plant including in the steam power station and other zones. He described the lagging as whitish/grey in colour with a composition much like plaster of Paris. He explained that, when he was required to remove the asbestos lagging from the pipe, he used a conventional hammer. He provided the following detailed description:

By inflicting a few blows, the lagging would crumble and obliterate and fall to the ground. A fine white cloud of dust was given off with each hammer blow. The dust would hang in the air and pollute my immediate breathing space. I would inhale the dust. The fine white dust particles would settle over my face, hair and clothing.

At times I used both a hammer and chisel to hack into the lagging to remove it and expose the underlying pipe surface ...

As and when I engaged in the de-lagging process my face was always within close proximity of the asbestos insulation as I bashed and hacked it away. On every occasion dust was given off which polluted my immediate breathing space and entered my nasal passages.

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<sup>2</sup> Exhibit P1, affidavit of the plaintiff sworn 26 September 2014; exhibit P2, audiovisual record of the plaintiff's examination on 29 October 2014.

Often a pipeline would block ... On such occasions it was the job of the yard crew to dismantle the length of pipeline and carefully drop it to the ground. Then with the use of hand-held tools and appliances such as crowbars we would go about using brute force to hit and push at the accumulated silt within the pipeline until the blockage was clear... Upon undertaking this task the asbestos lagging encapsulating the pipeline was caused to break off, obliterate and fall to the ground. The vigorous activity of removing the blockage caused the lagging to vibrate, crack and disperse asbestos particles into my work environment and immediate breathing space.

[11] The plaintiff said that he removed asbestos lagging from pipes in this way “hundreds of times over” during the period 1974 to 1977, in several zones including the steam power station zone and the precipitation sector zone. In terms of the regularity or consistency of such work, the plaintiff explained that renewal and repair of steam lines and other piping came about “in fits and starts”. There were periods when he was doing a lot of that kind of work for weeks on end, but at other times pipeline work was limited and his time would be largely spent on other tasks and activities. Nonetheless, the removal of asbestos lagging from pipes all over the alumina plant was a consistent component of his work duties as a member of the yard gang, and he estimated that, at least two or three times a week, he was required to engage in removing asbestos from piping. He would spend the rest of his working time in general cleaning duties.

[12] The plaintiff’s exposure to asbestos occurred not only during the actual de-lagging process, but also in the subsequent clean up. Whenever the plaintiff removed asbestos lagging, he had to ensure that all the debris was swept up and removed. He used a long handled broom to sweep the dust and debris

into a mound which he then scooped up onto a shovel and placed into a wheelbarrow. This process also caused asbestos dust to disperse into the plaintiff's immediate breathing space. When the wheelbarrow was full, the plaintiff would wheel it to the rubbish disposal site for the particular zone where he was working. Each zone had its own dump site to dispose of waste and rubbish. The plaintiff said that an enormous dust cloud was given off every time he emptied the wheelbarrow into the dumping pit. The dust engulfed his immediate breathing space and settled on his hair, face and clothing.

[13] The plaintiff also described cleaning the work area around the boilers. He said that this work was "filthy and very dusty". He described that dust as whitish grey particles with larger clumps which consisted of a chalk-like composition, whitish grey in colour similar to the asbestos lagging used on many of the pipelines at the refinery. The plaintiff had to sweep up that dust and debris into mounds in the same way that he had to clean up loose asbestos debris and dust left over from the de-lagging process.

[14] The plaintiff described another task in which he engaged: cleaning and mucking out the vast treatment/storage tanks located in various zones at the plant. He said that this was "an extremely filthy job requiring physical exertion and heavyweight manual labour". The plaintiff and one or more other workers would use a jackhammer to remove the mud and residue which was stuck thick and fast to the interior circumferential wall and ceiling of the tank. It does not appear that the plaintiff was exposed to

asbestos when he carried out this work, but the level of dust generated during the mucking out process was constant and substantial. Of significance, the plaintiff was kitted out for this task in an overall (which covered his whole body), a filter mask, goggles and helmet.

### **Evidence Stephen Memis**

- [15] The plaintiff's evidence was supported by the evidence of Stephen Memis, a former fitter machinist employed at the defendant's alumina refinery. Mr Memis commenced employment in 1982, some three to four years after the plaintiff had left the defendant's employ. For the first six years of his employment, he was based in the machine shop, but his occupation as a machinist and then fitter required him to make his way through various areas of the refinery and he would observe yard gang members working on piping or engaging in general cleaning duties.
- [16] In the early days of Mr Memis's employment, there was a lot of asbestos in the refinery, particularly on pipelines which ran all around the refinery. The presence and dangers of asbestos were not made known to him. When he first started, K-Lite<sup>3</sup> was used for the lagging of some of the pipework and it was prevalent on the pipe surfaces. Asbestos was also used on the gaskets and flanges.
- [17] In the early days the yard gang used to do a lot of work cleaning the pipelines. It was the responsibility of the yard gang to attend at the site of

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<sup>3</sup> See [22] below.

a leak or blockage to remove the asbestos lagging. This was part and parcel of the duties of the yard gang and it was common knowledge that this was the type of work that they did. Mr Memis saw asbestos dislodged from pipework by men using jackhammers, crowbars and other hand-held tools to clear out blockages within pipes. He was used to seeing the yard gang crew working on and handling asbestos pipelines. However, over the years he worked at the refinery such work became less frequent because asbestos was gradually removed, and because the manual handling and disassembling of pipelines to remove internal blockages was replaced by internal hydro-blasting of the pipes.

[18] The defendant objected to the admission of the evidence of Mr Memis, on the ground that it was irrelevant. Mr Crawley, counsel for the defendant, argued that, because the observations of Mr Memis were made at least some three to four years after the plaintiff had left his employ at the refinery, they were not relevant to the situation that had prevailed during the time of the plaintiff was employed there. Mr Crawley submitted, in the alternative, that I should give the evidence of Memis little or no weight.

[19] I reject the submissions of the defendant summarised in the previous paragraph, and admit the evidence of Mr Memis. There is no suggestion that the aluminium refinery was any worse in terms of occupational health and safety vis-à-vis the risk of asbestos at the time Mr Memis started work; indeed it is logical to infer that the situation was either the same as at the time the plaintiff was employed by the defendant, or better. Assessed in

that light, the evidence of Mr Memis provides considerable support for the plaintiff's evidence as to the nature of the work performed by the yard gang and as to the significant presence of asbestos at the refinery.

### **Defendant's company documents**

[20] At a time after 25 September 1987, the General Manager of the defendant reported to the seventy-fifth meeting of directors of the defendant on the topic of "Asbestos at Nabalco facilities – Gove".<sup>4</sup> It appears from the report that the issue of asbestos had arisen at the seventy-fourth meeting. Information provided by the General Manager can be summarised, relevantly, as follows:

- During construction of the refinery at Gove, asbestos-containing materials were used for Plant and Steam Power Station insulation.
- Extensive use was made of K-Lite insulation during plant construction. This is asbestos-reinforced calcium silicate with 10 to 15% of amosite.
- The turbines in the Steam Power Station were originally asbestos lagged. This was removed and replaced with non-asbestos containing product in mid-1970s. All high-temperature pipework, steam drums, exposed headers, downcomers and links were K-Lite insulated.
- Removal/replacement of this product is undertaken on a condition based program. Where either the cladding requires work or the insulation must be removed, K-Lite is replaced with asbestos-free product.
- In the Alumina Plant, K-Lite was used extensively for pipework, vessels and tank roof insulation. The extent of use is documented in

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<sup>4</sup>Attachment "C" to the report of the General Manager, undated, but referred to in 'Matters Arising' from the 74th meeting held on 25 September 1987.

the above-mentioned Contracts. As a generalisation it is fair to say that the vast majority of pipework was K-Lite insulated.

[21] The above extracts are clear evidence of the extensive use of asbestos at the Nhulunbuy refinery at the time of its construction and subsequently, and in particular for insulation (lagging) of pipework.

[22] K-Lite is a lightweight insulation material made from amosite asbestos and hydrous calcium-silicate material, manufactured by James Hardie and Coy Pty Ltd until the closure of the manufacturing plant in 1974.<sup>5</sup> The fact that K-Lite insulation at the defendant's refinery was progressively replaced with an asbestos-free product from the mid-1970s is not of itself evidence that the defendant actually knew of the dangerous properties of that product, because the replacement of K-Lite was part of a "condition-based program", not an occupational health and safety program, and K-Lite was probably not utilized because it was not manufactured after 1974.

### **What the defendant should have known**

[23] Evidence that the defendant should have known of the dangerous properties of the asbestos used so extensively at its Nhulunbuy refinery is provided by the evidence of Ann Cameron Batt, librarian. Ms Batt provided a report in response to a request from the plaintiff's solicitors as to the sources available to an Australian librarian in the period up to 1974 had that person been asked in that period for information on the effects of exposure to dust

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<sup>5</sup> Letter Warwick Gazzard to Shine Lawyers dated 5 November 2014, exhibit P5. Amosite asbestos is also known as "brown asbestos".

containing asbestos.<sup>6</sup> Appendix 10 to Ms Batt's report is a list of relevant literature in the period up to 1968. Library locations throughout Australia in which each book, journal or article was held are listed in Appendix 11.

[24] I set out below a brief (and not comprehensive) review of relevant materials.

[25] In May 1930, Merewether wrote an article entitled "The Occurrence of Pulmonary Fibrosis and Other Pulmonary Affections in Asbestos Workers". It was published in the *Journal of Industrial Hygiene*. It included the following at page 250:

With regard to the effect on the lungs of different varieties of asbestos, no evidence was found to indicate that any one of its varieties is more potent in producing fibrosis than the others, other factors such as concentration of dust, being equal. There is now no doubt, however, that both chrysotile and crocidolite will produce fibrosis; while the third, the comparatively newly discovered amosite, resembles crocidolite so closely in its chemical constitution, and in the characteristics of the dust, that there can be no reasonable doubt, also, with respect to it.

[26] In a 1930 publication, published by His Majesty's Stationery Office, Merewether and Price wrote a report entitled "Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry". It included a description of asbestosis at page 9,

It is helpful to visualise fibrosis of the lungs as it occurs in asbestos workers as the slow growth of fibrous tissue (scar tissue) between the air cells of the lung wherever the inhaled dust comes to rest. While new fibrous tissue is being laid down like a spider's web, that

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<sup>6</sup> Report exhibit P4.

deposited earlier gradually contracts. This fibrous tissue is not only useless as a substitute for the air cells, but with continued inhalation of the causative dust, by its invasion of new territory and consolidation of that already occupied, it gradually, and literally, strangles the essential tissues of the lungs.

[27] In the same report, under the heading “Summary and Recommendations” at page 31, was included the following:

The appropriate methods for suppression of dust may only be fully determined when the harmful effects of comparatively low concentrations of asbestos dust are duly appreciated.

[28] In November 1931, an article by Frederick Wilson was published in *Safety Engineering*. It was entitled “The Very Least an Employer Should Know About Dust and Fume Disease”. It included the following,

Under best conditions it cannot help but be harmful to a degree, while under adverse conditions the shortening of life is almost inevitable. We do know, however, the breathing of dust under the following conditions is seriously harmful:

...

Asbestos and every operation in which it is used.

[29] In 1942, W C Hueper published a text “Occupational Tumours and Allied Diseases”. At page 404, the author wrote, “There is an incidence of lung cancer and asbestosis of the lung which is definitely excessive”.

[30] On 10 July 1956, in a Melbourne journal, *Manufacturing and Management*, an editorial staff article included the following:

People prone to the disease are those handling asbestos in its raw state or processing it to make lagging materials; also operatives sawing and cutting any finished product containing asbestos such as brake linings, asbestos sheeting and various insulation materials. Dismantling old lagging is a hazardous occupation.

Once established, asbestosis constitutes a grave threat to life and health. Symptoms are shortness of breath, tightness and pain in the chest, together with loss of energy and tiredness.

[31] In 1957, Dr Thomas wrote an article in the *Medical Journal of Australia*.

He stated:

The following occupations are involved: handling the substance in its raw state; grinding the substance prior to its use in some process; mixing with diatomaceous earth or kaolin to form lagging materials; sawing, cutting and finishing any product containing asbestos – for example, brake linings, asbestos sheeting and various insulating materials; tearing down old lagging – this is a very dangerous process, even in the open air; spraying asbestos on walls and ceilings as an insulator.

[32] In 1960, the paper of Wagner & Ors was published in the *British Journal of Industrial Medicine*. It included:

This hypothesis could not be supported at once from the original histories obtained from the patients, for they included housewives, domestic servants, cattle herders, farmers, a water bailiff, an insurance agent, and an accountant, none of whom were working on the asbestos mines at the time. We therefore undertook a detailed investigation of their past occupation and place of residence, and the association with asbestos exposure was discovered ... In only one case do the relatives deny that the patient ever visited the asbestos mines or was exposed to asbestos.

[33] In 1965 the *British Journal of Industrial Medicine* published an article by Newhouse and Thompson. Eighty-three patients with confirmed

mesothelioma were studied for possible exposure to asbestos. The article included the following,

In 76 of the series full occupational and residential histories were obtained. Forty (52.6%) gave a history of occupational or domestic (living in the same house as an asbestos worker) exposure to asbestos ... There was also evidence that neighbourhood exposures may be important. Among those with no evidence of occupational or domestic exposures, 30.6% of the mesothelioma patients and 7.6% of the in-patients with other diseases lived within half a mile of an asbestos factory ...

*Domestic Exposures* – The group of nine, seven women and two men, whose relatives worked with asbestos, are of particular interest. The most usual history was that of the wife who washed her husband's dungarees or work clothes. In one instance a relative said that the husband, a docker, came home 'white with asbestos' every evening for three or four years and his wife brushed him down. The two men in this group, when boys of 8 or 9 years old, had sisters who were working at an asbestos factory. One of these girls worked as a spinner from 1925 to 1936. In 1946 she died of asbestosis. The press report of the inquest states: 'She used to return home from work with dust on her clothes'. Her brother had apparently no other exposure to asbestos ... He died in 1956 of a pleural mesothelioma.

...

There seems little doubt that the risk of mesothelioma may arise from both occupational and domestic exposures to asbestos.

[34] In 1964, an article by Dr Selikoff was published in the *Journal of the American Medical Association*. It included:

*Environmental Asbestos Exposure* – The recent demonstration, by South African and British investigators of pleural and peritoneal neoplasms among individuals who had chance environmental exposure to asbestos many years before raises the very important question of possible widespread carcinogenic air pollution. The possibility of environmental exposure has long been known. Soon after the initial clarification of asbestosis as a clinic entity, Hadow

demonstrated asbestos bodies in a man not employed in the industry but living next door to an asbestos factory. This finding was later mirrored in the finding of chronic beryllium disease among residents of a community near a beryllium factory. What is new, however, is an appreciation of the potential extent of the problem. Thomson and associates have reported the frequent findings of asbestos bodies in the lungs of urban dwellers. Among 6,312 individuals x-rayed in an area about an asbestos mine in Finland, Kiviluoto found 499 cases of pleural calcification of the type characteristically seen among asbestos workers, without obvious cause. In a comparable area without any asbestos mine, no cases were found among 7,101 persons x-rayed. It should be noted that these were not people who worked in the mine – none did – but, rather, were farmers, housewives, and others who lived in the general location.

[35] In 1965, an article by Dr Selikoff was published in the *New England Journal of Medicine*. The article included the following:

Ten deaths from mesothelioma among 307 consecutive deaths is an extraordinarily high rate and permits the conclusion that this disease is an important complication of asbestos exposure. This conclusion, moreover, refers to such exposure in the United States, under working conditions of the recent past, with relatively light and intermittent asbestos exposure. It indicates too, that mesothelioma may not necessarily be entirely a problem of only one kind of asbestos (crocidolite) and that it is surely not limited to South Africa.

In another case we found asbestos fibers in the lung of a man who died of pleural mesothelioma and who had worked in a dry-cleaning plant as a “spotter”. This observation calls to mind the finding of asbestos bodies and fibers in the sputum of people employed in a dry-cleaning works in South Africa in an area 100 miles away from the nearest mine, ... as well as cases of berylliosis caused by handling contaminated work clothes ... These examples of indirect occupational exposure and community exposure raise the important question whether mesothelioma will be found to be a community problem in addition to that existing with industrial exposure to asbestos.

[36] In 1968, an article by R H Mortimor and C B Campbell was published in the *Medical Journal of Australia*. The opening sentence was: “The

association of pleural mesothelioma with asbestos exposure is now widely recognized” (p 720). Just before the summary, it added,

These facts support the suggestions of Jacob and Anspach (1965) that the incidence of pleural neoplasms may be increased in people with relatively little asbestos exposure and insignificant respiratory insufficiency. They considered that people with heavy asbestos exposure develop severe pulmonary fibrosis and cor pulmonale, and do not live long enough to develop neoplasms.

Development of mesotheliomas after minor asbestos exposure has recently caused widespread concern (Leading Article, 1967*a, b*). The industrial applications of asbestos are increasing, and exposure is becoming more widespread. The incidence of this neoplasm is already rising, and prevention of further cases is a public health problem of some magnitude (Elmes, 1966).

[37] Standard handbooks or texts could have been utilised by the hypothetical librarian as a guide to appropriate reference works. Subject fields for search included medicine, hygiene, industrial chemistry, occupation and health, industrial hygiene and toxicology. There were a significant number of medical indexes available to a librarian in which relevant articles could have been identified and then found.

[38] There was a medical encyclopaedia available in Australia during the relevant period: the British Encyclopaedia of Medical Practice. An extract from the first edition (1937) was appendix 7 to Ms Batt’s report. From that I have noted the following passage in relation to knowledge of the risks of asbestos:

The manifold uses of asbestos in industry during recent years have enormously widened the risk of asbestosis and the disease may crop up in unexpected places. In addition to the mining, milling, carding,

spinning and weaving risks, there are risks from the use of asbestos as heat insulating for boilers and steam pipes and as electric insulating material and in the construction of electrical heating devices.

[39] I conclude from my consideration of the content of the many appendixes to Ms Batt's statement that there was a substantial amount of literature available in relation to the dangers of asbestos and as to the connection between asbestos exposure and serious pulmonary diseases, including mesothelioma, in the period up to 1974 and continuing to late 1977.

[40] I find that the defendant had access to, and the means to acquire the relevant knowledge and understanding of the risks of asbestos exposure in the period up to 1968.

### **The plaintiff's cause of action**

[41] I refer to the brief summary in [1], [2] and [4] above. The particulars of the defendant's negligence pleaded by the plaintiff are as follows:<sup>7</sup>

- (a) Exposing the plaintiff to asbestos dust and fibres when it knew or ought to have known that it was or was likely to be dangerous to the health of the plaintiff to do so;
- (b) Requiring or permitting the plaintiff to work with, on or near and be exposed to asbestos dust and fibres;
- (c) Failing to take any or any adequate precautions for the safety of the plaintiff whilst he was engaged upon his work;
- (d) Failing to provide and/or to maintain safe and proper places at which the plaintiff could perform his work;

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<sup>7</sup> Statement of Claim, par 8.

- (e) Failing to provide and/or require and/or to maintain a safe and proper system of work;
- (f) Failing to provide any or any proper or sufficient face masks or other protective clothing, appliances and/or equipment;
- (g) Failing to supply the plaintiff with any or any proper or sufficient breathing apparatus;
- (h) Failing to warn or instruct the plaintiff as to the need to use and wear protective clothing, equipment and appliances whilst working in the presence of or exposed to asbestos dust and fibres;
- (i) Failing to warn or instruct the plaintiff as to the need to use breathing apparatus whilst working with or in the presence of or whilst exposed to asbestos dust and fibres;
- (j) Failing to provide or maintain any or any proper or efficient ventilation or means of ventilation in premises at which the plaintiff was required to work in the presence of or when exposed to asbestos dust and fibres;
- (k) Failing to take any or any adequate steps to inform itself of the dangers to health consequent upon exposure to or inhalation of asbestos dust and fibres;
- (l) Ignoring and/or failing to accept and act upon advice, warnings and learning as to the dangers of exposure to asbestos dust and fibres;
- (m) Failing to take any or any proper, reasonable or adequate precautions to alleviate and minimise the exposure to asbestos dust and fibres of employees including the plaintiff;
- (n) Failing to warn the plaintiff;
  - i) Of the presence of asbestos materials on equipment and premises where the plaintiff was assigned to work;

- ii) Of dangers to health consequent upon exposure to and inhalation of asbestos dust and fibres
- iii) Of the need to take precautions to avoid and minimise exposure to and inhalation of asbestos dust and fibres;
- (o) Failing to have any or any proper or efficient method of suppression of asbestos fibres or dust or removal of asbestos from environments, sites and premises where the plaintiff was placed to work and/or was required to work;
- (p) Failing to install and/or maintain any or any proper dust extraction or removal plant or system at the sites and premises where the plaintiff was required to work;
- (q) Failing to measure or alternatively arrange for the measurement of levels of airborne asbestos dust and fibres in the environment in which the plaintiff was required to work;
- (r) Failing to take or require any adequate precautions for the safety of the plaintiff whilst he was engaged on sites where he was required to remove and disturb asbestos lagging resulting in the liberation of asbestos dust and fibres into the immediate breathing space of the plaintiff;
- (s) Failing to take any or any proper, reasonable or adequate precautions to alleviate or minimise the exposure to asbestos dust and fibres of persons, including the plaintiff within its employ;
- (t) Failing to specify or require wetting down prior to or during removal of asbestos lagging;
- (u) Failing to heed warnings and reports of the Factories Inspectorate and the Chief Inspector of Factories relating to the dangers to health of workers exposed to dust generally and to asbestos dust and fibres in particular;
- (v) Causing requiring or permitting the plaintiff to hammer, chisel, sweep and dispose of asbestos without warning of the dangers

and the need or requirement for respiratory protection to be worn by the plaintiff.

[42] There is significant repetition and overlap in the particulars. In brief, the negligence alleged against the defendant can be summarised as:

(1) exposing the plaintiff to dangerous asbestos dust and fibres without taking adequate precautions for his safety, specifically in not providing a face mask or breathing apparatus, and protective clothing; (2) generally failing to provide a safe system of work in terms of appropriate instructions, warnings and suppression measures, for example: providing adequate ventilation and wetting down of asbestos lagging prior to or during removal. I note also particular (l), following (k): “ignoring and/or failing to accept and act upon advice, warnings and learning as to the dangers of exposure to asbestos dust and fibres”.

### **Findings**

[43] I am satisfied on the balance of probabilities that the plaintiff has established that the defendant’s work operations involved a risk of injury which was reasonably foreseeable. The risk was real and not fanciful, far-fetched or trivial.<sup>8</sup> If the degree of risk of injury is seen a product of two factors, namely the likelihood that injury will be caused and the nature and degree of such injury, it must be borne in mind in relation to mesothelioma that, although it is comparatively rare in relation to other asbestos-related lung diseases, it is aggressive and always fatal. Moreover, the causal link

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<sup>8</sup> *Wyong Shire Council v Shirt* (1980) 146 CLR 40 at 48 per Mason J.

between mesothelioma and unprotected exposure to asbestos dust and fibres was well-known prior to 1974, when the plaintiff commenced work at the refinery.

[44] I am also satisfied on the balance of probabilities that, if the defendant was not aware of the real risk of injury, it should have been. There was a substantial amount of published material available to the defendant prior to 1974, and subsequently, as to the risk posed to the health and safety of persons working with asbestos, including asbestos lagging. The injurious properties of asbestos were well known at the relevant time.

[45] I am satisfied on the balance of probabilities that asbestos in the workplace posed a significant risk to the health and safety of the plaintiff, in particular as he went about the regular task of removing asbestos lagging from pipes and then sweeping up the debris.

[46] I am satisfied on the balance of probabilities that there were reasonably practical means of obviating or eliminating the risk to the plaintiff. As mentioned above, when the plaintiff was engaged in cleaning the large treatment/storage tanks in various sites at the defendant's refinery, he was provided with a protective garment or 'overall', which covered his whole body, as well as a filter mask, goggles and helmet. For all the tasks carried out by the plaintiff which exposed him to asbestos dust and fibres, the defendant could have provided him with a filter mask or breathing apparatus, and protective clothing. The defendant could have given the

plaintiff appropriate instructions and warnings. The defendant could have put in place suppression measures, for example, wetting down of asbestos lagging prior to or during removal; removing and disposing of contaminated clothing; showering of the plaintiff after he had carried out the de-lagging and other tasks which exposed him to asbestos dust and fibres. Clearly, there were reasonably practicable alternatives which the defendant could have put in place at the refinery in order to protect the plaintiff and thereby obviate or eliminate the risk of injury.

[47] I am satisfied on the balance of probabilities that the defendant's failure to obviate or eliminate the risk showed a lack of reasonable care for the plaintiff's safety. The defendant breached its duty of care to the plaintiff.

[48] In relation to causation, I am satisfied on the balance of probabilities that the plaintiff's mesothelioma was caused by the defendant's breach of duty of care. The medical evidence is clear,<sup>9</sup> and no competing cause has been identified.

### **The defence case**

[49] The defendant did not mount a positive case in defence of the plaintiff's claim in negligence. I refer to my observation in [9] above. However, the defendant pleaded and contended at trial that the plaintiff's common law action is barred by s 52 *Workers Rehabilitation and Compensation Act*, which provides, relevantly, as follows:

52 Abolition of certain rights to bring action

(1) 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;

(b) *not relevant*; or

(c) *not relevant*,

in respect of:

(d) an injury to the worker; or

(e) the death of the worker:

(i) as a result of; or

(ii) materially contributed to by,

an injury.

(1A) *not relevant*

(2) The purpose of subsection (1) is to ensure that, so far as the legislative power of the Legislative Assembly permits, no action for damages at common law shall lie in the Territory or otherwise in the circumstances described in that subsection and nothing in this Act shall be construed as derogating from that purpose.

[50] The definition of “injury” is contained in s 3(1) of the Act. Subject to the requirement that it must arise out of or in the course of a worker’s employment, “injury” includes a disease and the aggravation etc. of a pre-existing disease. The word “disease” is defined to include a physical

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<sup>9</sup> See [54] to [58] below.

ailment, element, disorder, defect or morbid condition whether of sudden or gradual development.

[51] Section 189(1), to which s 52 is expressly subject, is set out below:

189 Claim etc. before or after commencement of Act

- (1) 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.

[52] The defendant contends that the plaintiff was a “worker” and the defendant the “employer of the worker” for the purposes of s 52 *Workers Rehabilitation and Compensation Act*, and that the malignant mesothelioma from which the plaintiff suffers is an “injury” within the definition contained in s 3 of the Act: relevantly a disease arising out of the plaintiff’s employment with the defendant. The plaintiff does not contend otherwise. Therefore, on the defendant’s contention, the plaintiff’s common law damages action is barred by s 52 of the *Workers Rehabilitation and Compensation Act*; moreover, the plaintiff’s cause of action is not preserved by s 189(1) of the Act, because the cause of action did not arise before 1 January 1987, the commencement date of s 189.<sup>10</sup>

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<sup>10</sup> The *Workers Rehabilitation and Compensation Act* was enacted as the *Work Health Act*, no. 49 of 1986. Section 189 commenced on 1 January 1987.

### **When did the plaintiff's cause of action arise?**

[53] The defendant's contentions make it necessary to determine when, in relation to 1 January 1987, the plaintiff's cause of action arose. The crucial issue is when the plaintiff first suffered damage, since damage is an essential element of the cause of action in tort for negligence, and the cause of action is not complete until damage is sustained.<sup>11</sup> The relevant cause of action "is only complete when appreciable or other than purely minimal damage occurs."<sup>12</sup> Since damage is the gist of the cause of action,<sup>13</sup> it is necessary, for the cause of action to accrue, that a plaintiff suffer "actual damage as distinct from the risk or prospect of damage or contingent damage"<sup>14</sup> as a result of a defendant's breach of duty.

[54] The evidence of the plaintiff is that he began to experience chest pains in January 2014.<sup>15</sup> That accords with the history noted by Professor Roger Allen, that the plaintiff developed right-sided chest pain in January 2014.<sup>16</sup> It is possible that the plaintiff first became unwell in November 2013, when he developed pain on the right side of his chest and became breathless, as noted by Dr Robert Edwards.<sup>17</sup> However, the difference between the reported dates of onset of symptoms is not presently relevant.

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<sup>11</sup> See, for example, John Fleming, Carolyn Sappideen and Prune Vines, Fleming's The Law of Torts (Lawbook Co, 10<sup>th</sup> ed, 2011), 22 ; *Wilson v Rigg* [2002] NSWCA 246 at [21] per Giles JA; *Orica Ltd and Anor v CGU Insurance Ltd* (2003) 59 NSWLR 14 per Spigelman CJ at [32], per Mason P at [72], [78].

<sup>12</sup> *GRE Insurance Ltd v Bristile Ltd* (1991) 5 WAR 440 at 451.40; *Cartledge and Ors v E Jopling & Sons Ltd* [1963] AC 758 at 774, per Lord Evershed, M.R.

<sup>13</sup> See, for example, *Williams v Milotin* (1957) 97 CLR 465 at 474; *Do Carmo v Ford Excavations Pty Ltd* (1983-84) 154 CLR 234 at 245.7, per Wilson J.

<sup>14</sup> *Wilson v Rigg* [2002] NSWCA 246 at [23] per Giles JA.

<sup>15</sup> Exhibit P1, par 138.

<sup>16</sup> Exhibit P7, report of Prof Roger Allen dated 11 September 2014.

The parties agree that malignant mesothelioma probably commences within one to two years before, and almost certainly within five years before the onset of symptoms.<sup>18</sup>

[55] Professor Roger Allen, thoracic physician, provided expert evidence in the plaintiff's case.<sup>19</sup> He explained that, based on epidemiological studies, there is a well-accepted minimum latency period of approximately 10 years from the time of exposure to asbestos to the subsequent development of malignant mesothelioma. Professor Allen also wrote that, although the shortest latency is around 10 years, many patients develop mesothelioma many decades later, evidenced by the higher incidence of this condition in elderly patients. I set out below Professor Allen's evidence in relation to the process whereby exposure to asbestos ultimately leads to the development of malignant mesothelioma:

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 malignant tumour, i.e. a domino effect. ...

It is an accepted fact that there are oncogenes in cells, with the genetic material influenced by processes such as methylation and acetylation, leading to the development of abnormal "switches" in the tissue which regulates cell replication and if aberrant and abnormal will predispose them to unrestrained cell growth with no internal checks and balances, i.e. a malignant tumour. It is thought that asbestos fibres which are hydrated silicates of aluminium and

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<sup>17</sup> Exhibit D1, report of Dr Robert Edwards dated 19 September 2014.

<sup>18</sup> Exhibit C1, Further Agreed Facts.

<sup>19</sup> Exhibit P8, report of Prof Roger Allen dated 3 November 2014.

magnesium generate oxygen free radicals which... are known 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. ....

On the assumption that his exposure to asbestos commenced in 1974 and continued to 1977, during that period the asbestos fibres in his lungs set in train genetic abnormalities [in the mesothelial cells] which lay dormant well prior to 1987, and which led to the subsequent development of mesothelioma. ....

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.<sup>20</sup>

[56] Dr Robert Edwards, thoracic specialist, provided expert evidence in the defendant's case, including as to the process whereby exposure to asbestos ultimately leads to the development of malignant mesothelioma. I set out below part of Dr Edwards' evidence:

Mr Zabic had a history of exposure to asbestos whilst working at Alcan between 1974 and 1977. He has now developed a malignant mesothelioma.

It is not possible to state the exact time that the mesothelioma would have developed. .... it is known that the changes in the mesothelial cells commence very soon after the exposure to asbestos. However, it takes at least 10 years and probably 20 years before the cells are likely to become malignant. There is no upper [outer] limit as to time when mesothelioma may develop.

Mr Zabic has a latency period of between 37 and 40 years for the development of the malignant mesothelioma.<sup>21</sup> This is entirely in keeping with the known mechanism for malignant mesothelioma to develop.

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<sup>20</sup> Exhibit P8, report of Prof Roger Allen dated 3 November 2014, p 2.

<sup>21</sup> The reference to the "latency period of between 37 and 40 years" is to the time from exposure in the period 1974 to 1977, to 2014, when symptoms of malignant mesothelioma became apparent.

The asbestos fibres are inhaled and worked their way to the periphery of the lung and eventually work their way through the visceral pleura and eventually onto the parietal pleura. They then start to cause changes within the mitochondria and other elements of the mesothelioma [mesothelial] cells. This mechanism takes many years to develop into a malignant cell.

There is no upper limit beyond which mesothelioma may not develop after exposure to asbestos fibres. It is generally accepted that at least a latency period of 10 years is required before mesothelioma is likely to develop. The majority of mesotheliomas that have been diagnosed have between a 20- and 30-year latency period. Therefore the initial commencement of mesothelioma is probably somewhere between [19]74 and [19]77 when the first changes of the asbestos fibres interacting with the mesothelial cells would have occurred.<sup>22</sup>

[57] Although Dr Edwards refers in the final paragraph of the above extract to “the initial commencement of mesothelioma” having probably occurred between 1974 and 1977, I understand him to be describing the stage when changes in the mesothelial cells commenced, and not the malignant transformation stage, because, on his evidence, there is a substantial latency period, 10 to 20 years, before the mesothelial cells become malignant.

[58] My reading of the medical evidence of Dr Edwards and Professor Allen has led me to conclude that their opinions are largely consistent with one another. Dr Edwards’ opinion that the changes in the plaintiff’s mesothelial cells would have commenced very soon after his exposure to asbestos is consistent with the opinion of Professor Allen that the plaintiff’s exposure

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<sup>22</sup> Exhibit D2, report of Dr Robert Edwards dated 12 November 2014.

to asbestos caused genetic abnormalities well prior to 1987, albeit abnormalities which lay dormant.

[59] The defendant contends that the plaintiff did not suffer *damage* until he first experienced symptoms of malignant mesothelioma, alternatively not until one or two, or possibly five years before he first experienced such symptoms. Accordingly, it was not until then that the plaintiff's cause of action in tort arose. The defendant thus argued that the plaintiff's cause of action did not arise until November 2013, and certainly not before November 2009.

[60] The plaintiff concedes that it would not have been possible, immediately prior to 1 January 1987, to state that the changes in the plaintiff's mesothelial cells (or any genetic abnormalities) would probably lead to the development of malignant mesothelioma. Nonetheless, the plaintiff contends that it is clear from what has happened subsequently that the changes or abnormalities did in fact lead to the development of malignant mesothelioma. The ultimate outcome proves that, prior to January 1987, the plaintiff had suffered damage, albeit not then apparent but still sufficient to enable his cause of action to arise.

[61] The plaintiff relies on the decision of Derrington J in *Martindale v Burrows*:<sup>23</sup>

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<sup>23</sup> (1996) 1 Qd R 243 at 246.

While vulnerability to injury or the potential for harm does not itself amount to an injury (*Wardley Australia Ltd v Western Australia* (1992) 175 CLR 514, 527) that is different from the position where a morbid condition is initiated, leading naturally to more serious developments at a later stage. It is not a matter of potentiality in such a case simply because in other cases such a consequence might not follow. In *Wardley*, no harm whatever was done at the earliest stage and it was only when another event in the form of a trigger occurred that any harm followed. ...

It does not follow that if it is established that the condition has developed into mesothelioma, there will have been no relevant injury until the commencement of that development. The appearance of that condition establishes that the earlier morbid changes were indeed so serious as to be productive of mesothelioma at the later stage and were not merely potentially so. This means that the early changes did cause harm substantial enough to amount to injury at law.

.....

Although it is true that the initial “pathological change at the molecular level” without further pathological changes during the long latent period before the development of the mesothelioma was the only effect of such exposure at that stage, however that molecular change can be regarded as significant damage in the eyes of the law when it is established of the evidence that it led to the consequential development of mesothelioma. The substantial nature of such a change is not reduced by the postponement of grave harm flowing from it nor by its imperceptibility at the time, when in fact that state is demonstrable in the light of subsequent events.

[62] In *Martindale v Burrows* the plaintiff alleged that he had contracted mesothelioma as a result of his inhalation of asbestos during successive periods of employment commencing in 1956 and ending in the 1980s. Symptoms became apparent only in 1995 when the plaintiff suffered symptoms of cough and shortness of breath. The malignant transformation occurred some 12 – 18 months prior to November 1995, that is, in the period May to November 1994. The plaintiff applied before trial for a

declaration that his injury was suffered during that period, and hence that his cause of action arose at some time during that period, in order to avoid having to apply for an extension of the limitation period. Derrington J dismissed the application because he found that the injury constituting the plaintiff's cause of action occurred when the asbestos particles he ingested began to cause changes in his lungs, which occurred before the long latency period and well before the ultimate development of mesothelioma. The plaintiff's application for a declaration was dismissed and the plaintiff was left to pursue his application for an extension of the limitation period.

[63] In *Orica Ltd & Anor v CGU Insurance Limited*<sup>24</sup> the New South Wales Court of Appeal considered a case of a worker who had suffered injury through exposure to asbestos dust in unprotected conditions between 1959 and 1961 but had not experienced the first symptoms of mesothelioma until 2001. The worker obtained a damages settlement against his former employer which then sought indemnity from its insurer under workers compensation insurance policies in force in the period 1959 to 1961. Those policies provided the employer with indemnity in respect of liability independently of workers compensation legislation (including liability at common law).

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<sup>24</sup> [2003] NSWCA 331; (2003) 59 NSWLR 14.

[64] The Court held that the policies did not respond during their currency in respect of the employer's liability, independently of the Act, in damages for negligence. The reasoning of Spigelman CJ was as follows:<sup>25</sup>

If the words of the policy – “liable to pay” – mean the same with respect to the two circumstances to which they apply – that is, “compensation under the Act” and “in respect of his liability independently of the Act” – then the policy responds in the latter case only if the employer's liability has, within the relevant period, “vested” or “accrued”. In my opinion that does not occur earlier than the time of onset of mesothelioma. Damage is the gist of the action in negligence. The “injury” occasioned at the time of penetration of the lungs by 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.

[65] Santow JA applied an earlier New South Wales Court of Appeal decision of *WorkCover Authority (NSW) v Chubb Australia Ltd*,<sup>26</sup> and held that the tort was not complete until the occupational disease of mesothelioma was diagnosed. His Honour's analysis was as follows:<sup>27</sup>

149 In the present case, had the employee in 1961 sought to bring proceedings for his increased risk of contracting mesothelioma, it is clear he could not have succeeded then. But that is only for want of proof, available only in hindsight, that his ingestion of asbestos fibres had caused him damage. This is so even if we assume that the hypothetical court hearing that action in 1961 was armed with the evidence courts now accept regarding the causation and aetiology of mesothelioma. The most that could be said was that this exposure *increased the risk* of the employee contracting mesothelioma, as compared to the non-exposed population. That is to say, the employee “lost the chance” of not being at material risk of contracting mesothelioma, by his three year exposure at the hands of his careless employer. On the present state of the law that is not

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<sup>25</sup> *Orica Ltd & Anor v CGU Insurance Ltd* (2003) 59 NSWLR 14 at 23[32].

<sup>26</sup> (2000) 20 NSWCCR 614

<sup>27</sup> *Orica Ltd & Anor v CGU Insurance Ltd* (2003) 59 NSWLR 14 at 45[149].

enough. As Professor Stapleton in her recent article “Cause-in-Fact and the Scope of Liability for Consequences” (2003) 119 LQR 388 at 424 explains:

“English judges seem uniformly hostile to allowing the pure-loss-of-a-physical-chance to form actionable damage when there are, as yet absolutely no physical changes. [*Gregg v Scott* [2002] EWCA Civ 1471 (awaiting judgment on appeal to the House of Lords)]. It is thought that mere exposure to risk, say to asbestos, without any physical changes would allow in too many speculative claims ...”

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.

150 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: *Battaglia v James Hardy and Co Pty Ltd* (unreported, Vincent J, Supreme Court of Victoria, 12 March 1987); *Papadopoulos v James Hardy and Co Pty Ltd* (unreported, Kaye J, Supreme Court of Victoria, 12 February 1988).

...

...

153 In *WorkCover Authority (NSW) v Chubb Australia Limited* (2000) 20 NSWCCR 614, Stein JA, with Powell JA and Foster A-JA agreeing, concluded:

“...it is important to note that there is a significant difference between a closed claim for industrial deafness and a claim in

respect of mesothelioma. The former is a succession of completed tortious insults, whereas the latter, because of its aetiology is not complete until the disease of mesothelioma strikes, usually many years after the asbestos exposure. Also one will normally not know which asbestos fibre caused the mesothelioma. It could be an asbestos fibre inhaled during any time over a lengthy period of exposure.” (at 620 [26])

Stein JA concluded (at 621 [33]) that “the tort was not complete” until the occupational disease of mesothelioma was diagnosed. That must be taken to be the law.

[66] Mason P agreed with the analysis of Santow JA, deciding that the employer’s liability, such as it was, remained inchoate, in the eyes of tort law, because damage is the gist of the relevant cause of causes of action. The extracted paragraphs from his Honour’s decision set out below illustrate his reasoning:

71 The law has concepts known as causes of action. These define the criteria capable of giving rise to a liability recognised and enforced at law. A cause of action tells a plaintiff what must be proved as a bare minimum, when proceedings may first be brought, and when the time commences to run for limitation purposes. In relation to a right of indemnity against liability, it prescribes the matters capable of triggering a claim. The concept also forms the basis of *res judicata*. See generally *Letang v Cooper* [1965] 1 QB 232 at 242-3, *Port of Melbourne Authority v Anshun Pty Ltd* (1981) 147 CLR 589 at 597, 611; *Wardley Australia Ltd v Western Australia* (1992) 175 CLR 514 at 527, 558; *Effem Foods Pty Ltd v Trawl Industries of Australia Pty Ltd (Receivers and Managers appointed – in Liquidation)* (1993) 43 FCR 510 at 521; *Clarke v Bailey* (1993) 30 NSWLR 556 at 564; *Judamia v State of Western Australia*, Supreme Court of Western Australia, unreported, 1 March 1996.

72 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.

73 The worker did not sue or recover damages on the debatable bases that the chance or fear of contracting mesothelioma was the damage suffered (cf *GRE Insurance Ltd v Bristle Ltd* (1991) 5 WAR 440 at 442, per Pidgeon J and contrast *Metro-North Commuter Railroad Co v Buckley* 521 US 424 (1997); *Scarcella v Lettice* (2000) 51 NSWLR 302 at 306, *Segal v Fleming* [2002] NSWCA 262).

[67] As a single judge of this Court, and in the absence of binding High Court authority, I have a duty to follow the ratio decidendi of the New South Wales Court of Appeal in *Orica* unless I were to conclude that it is plainly wrong.<sup>28</sup>

[68] Although I may not necessarily be bound by the manner in which the New South Wales Court of Appeal applied the law in a case where the issue decided was in part an issue of fact, namely whether the ‘plaintiff’ had, at or by a particular time, suffered damage as a result of the negligence of another, I consider that the analysis of each of the members of the Court in *Orica* is persuasive. The issue decided was as to when damage was sustained sufficient to ground an action in tort for an employer’s negligence in respect of an employee who had suffered the onset of mesothelioma after a long latency period. That is the same issue as in the present case. The medical evidence and other facts were essentially the

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<sup>28</sup>*Farah Constructions Pty Ltd v Say-Dee Pty Ltd* (2007) 230 CLR 89 at 151-152 [135]; *CAL No 14 Pty Ltd v Motor Accidents Insurance Board*; *CAL No 14 Pty Ltd v Scott* (2009) 239 CLR 390 at 412 [49].

same as in the present case. All three members of the Court 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”.

[69] It assists to resolve the crucial issue in the present case to refer to the plaintiff’s pleading of damage, particularized in paragraph 21 of the Statement of Claim as follows:

- Malignant mesothelioma
- Breathlessness
- Chest pain
- Discomfort, lethargy, nausea
- Necessity to undergo numerous painful and invasive diagnostic medical procedures
- Restriction on social, domestic and recreational activities
- Dependency on others for assistance with daily personal and domestic tasks
- Reduced life expectancy and imminent death.

[70] The plaintiff had not suffered and was not suffering any of the above injuries, disabilities, restrictions, impairments or symptoms prior to or as at 1 January 1987.

[71] 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. It follows that the plaintiff’s claim for damages

against the defendant after his malignant mesothelioma developed was statute-barred by s 52 *Workers Rehabilitation and Compensation Act*, and not preserved by s 189 of that Act.

[72] Because of my conclusions in [71], it is not necessary for me to determine the parties' arguments in relation to the *Limitation Act*, but if things were not as they are, I would extend time to enable the plaintiff's action to be commenced and proceed on the merits. His case would clearly come within s 44 of the *Limitation Act*.<sup>29</sup>

[73] The plaintiff's claim should be dismissed.

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<sup>29</sup> *Limitation Act* (NT), s 44(2) and s 44(7)(a).