CITATION:	*Roussos v Amaca Pty Ltd* [2024] NTSC 20
PARTIES:	ROUSSOS, KOULLA (AS REPRESENTATIVE OF THE LATE IRAKLIS ROUSSOS) v AMACA PTY LTD (UNDER NSW ADMINISTERED WINDING UP)
TITLE OF COURT:	SUPREME COURT OF THE NORTHERN TERRITORY
JURISDICTION:	SUPREME COURT exercising Territory jurisdiction
FILE NO:	95 of 2019 (21933118)
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Negligence – Contributory negligence - Dust disease - Asbestos – Cigarette smoke - Lung cancer - Fatal injury – Compensation to relatives – Damages awarded

Compensation (Fatal Injuries) Act 1974 (NT) s 10; *Construction Safety Rules 1978* (NT); *Health and Other Services (Compensation) Act 1955* (Cth); Law *Reform (Miscellaneous Provisions*) *Act 1956* (NT) s 5, s 6, s 6(1)(c)(i), s 6(2), s 6(4); *Medical Services Act 1982* (NT); *Personal Injuries (Liabilities and Damages) Act 2003; Tobacco Act 1992 (NT)* s 6*; Tobacco Advertising Prohibition Act* (Cth)*; Tobacco Control Act 2002* (NT).

Amaca Pty Limited v Booth [2011] HCA 53; *Amaca Pty Ltd v Ellis* [2010] HCA 5; 240 CLR 111; *Bolton v Stone* (1951) AC 850; *Brierley v Ellis*

[2014] NSWCA 230; *Caterson v Commissioner for Railways* [1973] HCA 12; 128 CLR 99; *CSR Ltd v Bowditch* (1991) 7 NSWCCR 223; *GIO (NSW) v Planas* [1984] 2 NSWLR 671; *Glasgow Corporation v Muir* [1943] AC 448; *Griffith v* Kerkemeyer (1977) 139 CLR 161; *Koufos v Czarnikow Ltd* (1969) AC 350; Purkess *v Crittenden* [1956] HCA 34; 114 CLR 164; *(Re Lorizio) A B and P Constructions Pty Limited Cross Claimant v Amaca Pty Limited (formerly James Hardie & Co Pty Limited* [2006] NSWDDT 19; *Sullivan v Moody* (2001) 207 CLR 562; *Sydney Water Corporation v Turano* (2009) 239 CLR 51; *The State of South Australia v Ellis* [2008] WASCA 200; (2008) 37 WAR 1; *The Wagon Mound (No 2.)* (1967) AC 617; *Van Gervan v Fenton* [1992] HCA 54; 175 CLR 327; *Watts v Rake* [1960] HCA 58; 108 CLR 158; *Wyong Shire Council v Shirt* [1980] HCA 12; 146 CLR 40, referred to.

Amaca Pty Ltd v Ellis [2010] 240 CLR 111; *Judd v Amaca Pty Ltd* [2003] NSWDDT 12; *McDonald v State Rail Authority* (1998) 16 NSWCCR 695; distinguished.

Amaca v Werfel [2020] SASCFC 125, not followed.

REPRESENTATION:

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Defendant:	G Watson SC, H Cooper

Solicitors:

Plaintiff:	Maurice Blackburn Lawyers
Defendant:	Mills Oakley

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

Roussos v Amaca Pty Ltd [2024] NTSC 20
No. 95 of 2019 (21933118)

BETWEEN:

KOULLA ROUSSOS AS REPRESENTATIVE OF THE LATE IRAKLIS ROUSSOS

Plaintiff

AND:

AMACA PTY LTD (UNDER NSW ADMINISTERED WINDING UP)

Defendant

CORAM: SOUTHWOOD J

REASONS FOR JUDGMENT

(Delivered 13 May 2024)

Introduction

[1] The plaintiff brings this action as representative of the estate of the late Iraklis Roussos (the deceased) for an alleged negligent breach of duty of care owed to the deceased by the defendant that resulted in his death. The action is brought under s 5(1) of the *Law Reform (Miscellaneous Provisions) Act 1956* (NT) the effect of which is to confer on a deceased person's estate a cause of action that was vested in the deceased at the time of his death. The plaintiff also relies on ss 7 and 8 of the *Compensation (Fatal Injuries)*

Act 1974 (NT). The effect of the latter Act is to confer upon the personal representative of a deceased person the right to bring an action for the benefit of the deceased's family members who sustained damage due to the deceased's death because of the defendant's negligence and breach of duty.

[2] The plaintiff's case is that he was exposed to asbestos dust on a regular basis at work for much of his life, which was a cause of his lung cancer that contributed to his death at the age of 81 on 26 November 2019. A significant factor in this case is the deceased was also a cigarette smoker. He started smoking at the age of 18. By the time he was 30, he claims to have been smoking 55 to 60 cigarettes a day. He continued smoking until 2010. The defendant's case, in part, is that the cause of the deceased's cancer was his smoking and the evidence does not sustain a finding that his exposure to asbestos dust was a contributing cause.

Brief factual background of the deceased

[3] The deceased was born on the Greek island of Kalymnos on 15 June 1938. He emigrated to Australia in 1956. At that time, he could not speak English. Apart from the period of 1973 to 1976, when he returned to Kalymnos, the deceased worked in Darwin in the building industry until his retirement in 2012. By then, he was 74 years of age.

[4] According to his affidavit, the deceased arrived in Darwin in November 1956. He was employed immediately by Mr Bill Richardson as a carpenter constructing houses in and around Darwin from timber, Masonite and fibro.

All the houses had timber frames. The rooves were made of asbestos cement sheeting. The external walls, ceilings, and eaves were all flat fibro sheets. The internal walls were either flat fibro sheets or Masonite. Tilux, a patterned fibro sheet, was used in the wet areas, i.e. the kitchen, toilet, bathroom and laundry. He estimated that he cut, sawed, hammered and drilled into this material every single day using fibro cutters, handsaws, and other hand tools. The work produced a lot of dust that he breathed in and got onto his clothes and hands, not only from his own work, but also from the work of others similarly employed by Mr Richardson. During this time, the deceased estimated that he worked on fibro sheeting for an average of approximately 10 hours per day over a thirteen-month period. He mentions that his normal working hours were Monday to Friday 12 hours per day but there was a lot of overtime. It is unlikely that he worked with power tools during this period.

[5] After that, the deceased worked with Mr Richardson and about 130 other employees on building houses, a school, a clinic and a restaurant in Batchelor. Apart from the fact that the roofing was made from corrugated iron, the materials used in each building were the same as before. This project lasted for nine months. He worked long hours seven days a week. He estimated that he worked with fibro for about 12 hours per day.

[6] In late 1958, the deceased and his brother returned to Darwin and worked for Christou Brothers as carpenters for approximately one year. All of the houses they built were constructed using fibro cement, including the rooves,

in the same manner as before. He worked on average 5 or 6 days a week but there was a lot of overtime. He estimated that he worked with fibro cement for an average of 10 hours per day.

[7] In 1959, he and his brother started their own construction business called Roussos Brothers, which lasted until the early 1980s. The business started small, but developed rapidly and reached the stage where they were employing as many as 120 people. They built houses (including five to ten houses at a time on some government contracts), schools, and government buildings all using fibro cement products. They also converted the old Darwin Hospital into the Darwin University College. The deceased preferred to work on the tools and left most of the business decisions to his brother. During this period, he worked with power tools that produced much more dust than hand tools. He estimated that up until 1970 he worked a 12-hour day, 5 days per week, which was almost entirely spent on tools on site. He estimated he worked on fibro cement products for an average of 8 to 10 hours a day during this period.

[8] Between 1970 and 1980, he spent slightly less time on the tools and more time onsite inspections and running the business. He estimated that, excluding the time he was overseas between 1973 and 1976, he spent an average of six hours a day on the tools using fibro cement products.

[9] After Cyclone Tracey, he returned to Darwin in about 1976 and was involved in the reconstruction of the town.

[10] The business with his brother ended in 1983 following a fall-out between the brothers. Thereafter, he worked as a sole contractor and then in partnership with Chris Mellios who owned a business called Nightcliff Builders Pty Ltd. By partnership, he explains that he bought shares in Mr Mellios' company. The use of fibro cement products ceased in the mid-1980s. He worked on the tools in this business and during the period that they were using fibro cement products, he estimates that he worked on the tools for an average of 8 hours a day, five days a week.

[11] In the pleadings, the plaintiff accepts that Mr Roussos' exposure to asbestos cement dust ceased in 1984. The first sign of lung cancer was in 2019.

[12] For nearly all the period that he worked up to the end of 1983, the deceased was breathing in the fibro dust not only from his own work, but also from the work of other workers who were working nearby.

[13] Senior counsel for the defendant submitted that I should treat the deceased's evidence with great caution as he passed away before the trial and was not cross-examined. I accept this as a general proposition, but each case must be weighed on its merits and in its context.[1] For instance, it was not seriously contended that the deceased never worked as a carpenter or that he never worked using fibro cement products or that he was never exposed to dust from fibro cement products. However, it was suggested that the hours he

1 See *Brierley v Ellis* [2014] NSWCA 230 at [24] per Meagher JA (Basten and Gleeson JJA concurring).

said he came into contact with fibro cement dust were exaggerated, and it will be necessary to consider this when I come to deal with causation.

Issues

[14] The following issues arise for determination:

1. Did the defendant owe the deceased a duty of care, which the defendant breached?

2. If yes to 1, was the breach of duty a cause of the deceased's death?

3. If yes to 1 and 2, was the deceased guilty of contributory negligence?

4. What damages, if any, should be assessed and awarded to the plaintiff in this case?

Duty of care

[15] It was not disputed that at some point the defendant became aware that exposure to asbestos products had the potential to cause serious injury to persons who worked in the building industry. Senior Counsel for the defendant submitted that a duty of care arose from the date upon which a risk of injury to the deceased utilising asbestos cement was foreseeable. It was conceded that this risk was foreseeable, at least from 1978 if not earlier.

[16] In *Sydney Water Corporation v Turano*[2] it was said that reasonable foreseeability of the class of injury sustained by the plaintiff is an essential condition to the existence of a legal obligation to take care for the benefit of

2 (2009) 239 CLR 51 at [45]-[46] per French CJ, Gummow, Hayne, Crennan and Bell JJ.

another. It is not necessary for there to be proof that the defendant was aware of the precise mechanism by which the relevant risk might arise.

[17] Counsel for the plaintiff submitted that the evidence established that the defendant was aware of the relevant risk of injury at a much earlier time. The relevant risk, in this case, is the risk of contracting lung cancer after exposure to asbestos products, especially asbestos cement. This was established by reference to documents tendered as part of Exhibit P4, which included the following:

- The defendant's in-house medical officer, Dr McCullagh, reported on 31 December 1965 that asbestos was probably the cause of two types of lung cancer, namely cancer of the bronchi and mesothelioma[3] and recommended that wherever possible new employees should be chosen from older rather than younger applicants.[4]

- At the Factory Managers' Conference on 11 July 1966 Dr McCullagh reported that recent literature had stated fairly conclusively that when inhaled asbestos dust can cause lung cancer and cancer of the chest cavity lining as well as asbestosis; that there was no safe upper limit for asbestos dust; exposure is dangerous and cumulative; the heaviest exposures cause asbestosis while lighter exposures cause cancers; the medical health scheme at the defendant's factory at

3 Ext P4, Vol 2 p320.

4 Ibid, p323.

Camellia in New South Wales included a pre-employment medical check with preference given to older men and non-smokers, with the main aim of protecting future sales which could be affected by asbestosis and cancer fears.[5]

- Dr McCullagh sent a note reporting the finding of Selikoff, Churg & Hammond that the combination of chrysotile[6] with benzpyrene[7] greatly increased the cancer rate. The note was sent to Mr F A Page at head office, Mr N. E. Gilbert at the Camellia factory, and Mr R D Palfreyman in Perth WA.

- Part 2 of Exhibit P4 is comprised of a discrete bound volume. The volume contains a document that is numbered 136 in the index to Part 2. The document is described in the index as - Unsealed Part of James Hardy & Co Pty Ltd. In the index to Part 2 of Exhibit P4 the document is dated 31 December 1967. Page 491, which is part of document numbered 136, is headed: 22-1 Bars to Engagement. The chapeau to part 22-1 states:

> Bars to Employment. In the following circumstances a prospective employee should, generally, not be employed for employment in any work involving the handling of asbestos *or its products*.

Subparagraph 22.1(a) states:

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5 P4 Vol 1 pp122-123; and Vol 3 pp770-771.

6 White asbestos.

7 A crystalline aromatic hydrocarbon produced by the incomplete combustion of organic material such as tobacco.

> Those who smoke more than 20 cigarettes or 25 grams of tobacco daily are not accepted for employment.

- Dr McCullagh and Mr Ray Palfreyman, the defendant's personnel director, attended a conference in Dresden in April-May 1968 and reported that lung cancer attributable to asbestos exposure may occur in the absence of asbestosis; no inference can be drawn as to the relationship between fibre size and carcinogenicity; the British problem of "cigarette smoking with asbestos exposure as the straw that broke the camel's back"; and that Selikoff had demonstrated the great extent to which cigarette smoking increases the likelihood of lung cancer in the asbestos exposed.[8]

[18] The defendant was also aware of the following matters:

- There was a problem with asbestos dust exposure from its AC building products on large job sites and that various union campaigns were directed towards the complete abolition of the use of asbestos in any form. It was proposed by James Hardy that the response to this was a re-education program directed at all sections of the community to minimise fears and bias against these products.[9]

- The use of certain power tools, circular saws and masonry discs, generated high dust levels up to 90 fibres per ml and in another test,

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8 P4 Vol2, pp 132, 135, 136, 137.

9 Ext P4 Vol5 pp872-876 dated 19 October 1979.

130 fibres/mil.[10] In the test carried out on 25 May 1976 cutting AC sheeting in the open air, the result indicated that "about a quarter of an hour of sawing A.C. sheet is all that could be allowed in a day in well-ventilated surroundings..."[11]

[19] The plaintiff further submitted that, as early as the mid to late 1960s, the defendant was aware of a link between the exposure to the dust from asbestos products and cancer:

- In a letter to Mr J B Reid dated 18 February 1966, the defendant's then Personnel Manager, Mr ET Pysden, said, referring to a newspaper article, "it is one of many reports of world studies that have been conducted since 1935 when the association between exposure to dust and carcinoma of the lungs, mesothelioma of the pleura, tumour of the bladder and uterus and other fatal complaints were first recognised. The nucleus is dust particles-fibre…four types of asbestos - chrysotile, amosite, crocidolite and anthophyllite - have similar effects on the body and it has not been possible to establish, that one is more pathogenic than the others… [12]

- On 14 March 1966, Mr Pysden sent Dr Bell (Director of Division of Occupational Health) a copy of the papers presented at the

10 Ext P4 Vol 5 pp1030-1059.

11 Ibid, p1044.

12 Ext P4 Vol 2 p315.

conference of the New York Academy of Science on the Biological Effects of Asbestos.[13]

- On 29 March 1966, Dr S F McCullagh wrote a commentary on the New York conference in which he said: "I think it is fair to say that asbestos fibre is generally held to be associated with and probably the cause of two forms of cancer, bronchogenic carcinoma (cancer of the bronchi) and mesothelioma…"[14]

The defendant and the industry knew that the application of powered equipment to its products would release large quantities of dust. It was known that tradesmen used power saws and abrasive discs fitted to angle grinders. It was also known most tradesmen worked without dust suppression equipment. The defendant knew that compressed sheeting, which was manufactured for use in floors, and Tilux (in bathrooms), should be cut or worked using power saws with dust suppression.[15]

[20] The evidence of Mr Kevin Bagot, who was employed by the defendant during the period from 1953 to August 1977, was to the effect that from the late 1960s he attended promotional shows at stockists on behalf of the defendant, "which would include reference to safety precautions including keeping dust down and the use of means and techniques that reduce the

13 Ext P4 Vol 2 p 316.

14 Ext P4 Vol 2 p 320.

15 Ext D6 p245.

amount of dust. From the late 1960s the promotions would include telling the audience that masks should be used when using power saws."

[21] In oral submissions, counsel for the plaintiff referred to a letter dated 24 January 1966, written by Dr McCullagh on the defendant's head office letterhead, about "Asbestos, cigarette smoking and cancer". The letter refers to "asbestotics" who were smokers at Camellia, and certain findings that suggest "smoking predisposes [workers] not only to lung cancer but to asbestosis itself."[16] The letter goes on to record the findings of researchers to the effect that in the UK "over one half of asbestotics now die of lung cancer" and the findings of other researchers to the effect that "the combination of chrysotile (though not amosite) [and smoking] greatly increased the cancer rate." It is also noted that studies of workers in a factory in the UK "suggest that following the establishment of adequate dust control in 1933 the lung cancer rate was no different from that of the community generally."

[22] Mr Parker QC submitted that the risk of injury that needed to be shown was not a risk of injury due to lung cancer, or for that matter mesothelioma, but simply a risk that exposure to asbestos dust could give rise to respiratory illness, and that risk was well established by 1961, in fact by 1955. After the Doll Paper, it was reasonably foreseeable that asbestos exposure could cause harm.

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16 Ext P4 p368-369.

[23] The defendant's main submission was that the risk of contracting an asbestos-related disease from asbestos cement dust was not reasonably foreseeable for the whole of Mr Roussos' period of exposure. The period pleaded commenced in 1956 and continued until 1984, with the exception of a four-year period in 1973-1976 when the deceased returned to live in Kalymnos. It was submitted that the Court will need to determine at what point it did become reasonably foreseeable. I accept that submission. The determination of the point it became reasonably foreseeable is important. It is only from then on that the defendant is required to take appropriate precautions to diminish or eliminate the risk of injury.

[24] The defendant submitted that a great deal of the evidence about the development of knowledge of the risks inherent in the use of asbestos is irrelevant because the risks of exposure to asbestos are different to the risks of exposure to an end-user of asbestos cement. There is not a great deal of asbestos in asbestos-cement products. A figure of 8 to 15 percent was mentioned. The cement is added to bind the asbestos fibres in the product. However, the evidence on which the plaintiff relies referred to asbestos dust, and such dust was quite capable of entering the atmosphere from the use of power tools such as electric saws and drills on asbestos-cement products. In my opinion, the whole of the evidence is relevant. While the degree of risk of harm may be different, the risk of harm is a risk of the same or similar harm. The inhalation of asbestos dust or asbestos fibres is potentially harmful and while a greater period of exposure to asbestos dust caused by

cutting fibro may be required before harm occurs, appropriate warnings still need to be given. Consequently, knowledge of the risks of harm inherent in the use of asbestos is relevant.

[25] The defendant further submitted that the best means of understanding when the risk of harm became recognised is to look at independent sources, including government publications. In particular, the standards published by the National Health and Medical Research Council (the Council). It was submitted that over time those standards show that it was thought that persons in the deceased's class were not at any risk from asbestos fibre. In its standards published in 1961 and 1964, the Council recommended a level of 5 million particles per cubic foot of air as representing the conditions under which nearly all workers may be repeatedly exposed, day after day, without adverse effect.[17] It was argued that, according to the evidence of the plaintiff's expert Mr Kottek, this level was grossly in excess of the time-weighted averages to which the deceased was exposed, but the passage cited to support this says nothing of the kind.[18]

[26] The next standard, which was published in 1971, set a level of 4 fibres per cubic centimetre of air.[19] In 1975, the standard remained the same for both chrysotile and amosite.[20]

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17 Ext D5 p22 and 32-40.

18 Kottek, tr 221.5- 221.9.

19 Ext D5 p59.

20 Ext D5 p76.

[27] In 1978, the Council published a "Code for the handling of Asbestos by Small Users". It defined "small users" as "construction firms" which used less than "say, 5 tonnes of raw asbestos per annum."[21] For asbestos cement products, the Code said:[22]

> Asbestos cement (fibro) is a hard surfaced material in which the asbestos fibres are reasonably bonded by cement. Work with this material can be carried out safely with handsaws and other hand tools and power drills.
>
> Occasional power sawing does not present a problem to an operation of say, 15 minutes in a shift, and if carried out in the open air. For longer periods dust extraction equipment should be used, or the dust should be suppressed by the use of water, or an approved respirator should be used. Where water is used for dust suppression care should be used to ensure that tools are electrically safe.

The significance of this standard is that on the defendant's case, at no time was it foreseeable that work such as was undertaken by the deceased reached that level of exposure. However, the above statement limits the use of power tools to occasional use for 15 minutes in a shift and assumes a uniformity of dust production by various power tools.

[28] The argument of the defendant seems to be, at least in part, that even if it were reasonably foreseeable that a person working in a factory which produces asbestos products might get cancer from coming into contact with asbestos, it was not reasonably foreseeable that a tradesman using asbestos cement might get lung cancer or any other adverse effect on health at least until about 1978.

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21 Ext D5 p88.

22 Ext D5 p88-89.

[29] However, there is no evidence that the Code for the handling of Asbestos by Small Users was readily available in the Northern Territory or that the defendant relied on the Code. On the contrary, as stated at [17] to [20] above, the evidence establishes that by at least the mid-1960s the defendant was aware of the risks associated with inhaling asbestos dust and was aware that carpenters were using power tools to cut its asbestos cement products.

[30] Reasonable foreseeability of the class of injury is an essential condition of the existence of a legal obligation to take care for the benefit of another.[23] In *Wyong Shire Council v Shirt* [24] Mason J, whose judgment was concurred in by Murphy and Aickin JJ, after reviewing the authorities relating to foreseeability, notably *The Wagon Mound (No2,)*[25] *Bolton v Stone*,[26]*Caterson v Commissioner for Railways*[27] *Koufos v Czarnikow Ltd.*[28] as well as other authorities, said (at pp 47-48):

> …this Court would be well advised to accept that the law upon the point was correctly stated and applied by the Judicial Committee in *"The Wagon Mound" (No 2)…* because there are sound reasons for accepting it as a correct state of the law.
>
> In essence its correctness depends upon a recognition of the general proposition that foreseeability of the risk of injury and the likelihood of that risk occurring are two different things. I am of course referring to foreseeability in the context of breach of duty, the concept of

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23 *Sydney Water Corporation v Turano* [2009] HCA [42] at [45] per French CJ, Gummow, Hayne, Crennan and Bell JJ; *Sullivan v Moody* (2001) 207 CLR 562 at 476 [42] per Gleeson CJ, Gaudron, McHugh, Hayne and Callinan JJ.

24 [1980] HCA 12; (1980) 146 CLR 40.

25 (1967) AC 617.

26 (1951) AC 850.

27 [1973] HCA 12; 128 CLR 99 at 101-102 Barwick CJ.

28 (1969) AC 350.

> foreseeability in connexion with the existence of the duty of care involving a more generalized enquiry.
>
> A risk of injury which is quite unlikely to occur, such as that which happened in *Bolton v Stone*, may nevertheless be plainly foreseeable. Consequently, when we speak of a risk of injury as being "foreseeable" we are not making any statement as to the probability or improbability of its occurrence, save that we are implicitly asserting that the risk is not one that is far-fetched or fanciful. Although it is true to say that in many cases the greater the degree of probability of the occurrence of the risk the more readily it will be perceived to be a risk, it certainly does not follow that a risk which is unlikely to occur is not foreseeable.
>
> In deciding whether there had been a breach of the duty of care the tribunal of fact must first ask itself whether a reasonable man in the defendant's position would have foreseen that his conduct involved a risk of injury to the plaintiff or to a class of person including the plaintiff. If the answer be in the affirmative, it is then for the tribunal of fact to determine what a reasonable man would do by way of response to the risk. The perception of the reasonable man's response calls for a consideration of the magnitude of the risk and the degree of probability of its occurrence, along with the expense, difficulty and inconvenience of taking alleviating action and any other conflicting responsibilities which the defendant may have. It is only when these matters are balanced out that the tribunal of fact can confidently assert what is the standard of response to be ascribed to the reasonable man placed in the defendant's position.
>
> The considerations to which I have referred indicate that a risk which is remote in the sense that it is extremely unlikely to occur may nevertheless constitute a foreseeable risk. A risk that is not far-fetched or fanciful is real and therefore foreseeable. But, as we have seen, the existence of the foreseeable risk of injury does not in itself dispose of the question of breach of duty. The magnitude of the risk and its degree of probability remain to be considered with other relevant factors.

[31] In relation to foreseeability of the risk, the question is not only what the defendant actually knew about the risk, but what he ought to have known. In *The Wagon Mound (No 2*) [29] Lord Reid said:

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29 *The Wagon Mound (No 2)* (1967) AC 617.

> If a real risk is one which would occur to the mind of a reasonable man in the position of the defendant's servant and which he would not brush aside as far-fetched, and if the criterion is to be what a reasonable man would have done in the circumstances, then surely he would not neglect such a risk if action to eliminate it presented no difficulty, involved no disadvantage and involved no expense.
>
> In the present case the evidence shows that the discharge of so much oil on to the water must have taken a considerable time, and a vigilant ship's engineer would have noticed the discharge at an early stage. The findings show that he ought to have known that it is possible to ignite this kind of oil on water, and that the ship's engineer probably ought to have known that this had happened before. The most that can be said to justify inaction is that he would have known that this could only happen in very exceptional circumstances; but that does not mean that a reasonable man would dismiss such risk from his mind and do nothing when it was so easy to prevent it.

[32] Thus, the standard of care of the reasonable man is theoretically objective, but as Lord MacMillan observed in *Glasgow Corporation v Muir*:[30]

> Legal liability is limited to those consequences of our acts which a reasonable man of ordinary intelligence and experience so acting would have in contemplation…The standard of foresight of the reasonable man is in one sense an impersonal test. It eliminates the personal equation and is independent of the idiosyncrasies of the particular person whose conduct is in question. Some persons are by nature timorous and imagine every path beset by lions; others, of more robust temperament fail to foresee or nonchalantly disregard even the most obvious dangers. The reasonable man is presumed to be free both from over-apprehension and from overconfidence. But there is a sense in which the standard of care of the reasonable man involves the application of a subjective element. It is still left to the judge to decide what in the circumstances of the particular case the reasonable man would have had in contemplation and what accordingly the party sought to be made liable ought to have foreseen. Here there is room for diversity of view, as indeed, is well illustrated in the present case. What to one judge might seem far-fetched may seem to another both natural and probable.

30 [1943] AC 448 at 457.

[33] In my opinion, a reasonable man in the position of the defendant ought to have foreseen the risk to persons such as the deceased contracting lung disease, be it mesothelioma or cancer from asbestos dust fibres by at least 1966. That such a risk was foreseeable by then is established by the matters referred to in paragraphs [16] to [21] inclusive above. On the evidence before the Court, it cannot be said that such a risk was far-fetched or fanciful.

[34] The deceased was using multiple products manufactured by the defendant to construct houses day in and day out over a period of 25 to 30 years. For the majority of that time he was using power saws and abrasive disc equipment. These were not fitted with wetting devices or dust suppressing equipment.

[35] The defendant and the industry knew that the application of power equipment to its products would release significant amounts of dust. The defendant knew that tradesmen used power saws and abrasive discs fitted to angle grinders and most tradesmen worked without dust suppression equipment. James Hardie knew that carpenters should use power saws with dust suppressors to cut and work Tilux and the compressed sheeting it manufactured for use in floors.

[36] I accept the plaintiff's submission that the defendant knew or ought to have known that dust including asbestos dust to which builders such as the deceased were exposed presented a foreseeable risk of injury to a person using the materials in the manner intended.

The response to the risk was inadequate

[37] The plaintiff submitted that, at least by 1961, to the defendant's knowledge carpenters were using power tools such as power saws to cut fibro sheets and other fibro products. Reference was made to Mr Bagot's affidavit. However, while his affidavit refers to power tools it is not clear when the defendant became aware as alleged, save that, according to Mr Bagot, the defendant recommended the use of masks when using power tools from the late 1960s. In addition, the evidence of Mr Pickford was that from 1959-1960 there was an increasing number of people using power tools.[31] Hence, the plaintiff submitted that the defendant should have provided appropriate warnings by at least 1961. However, for the reasons stated at [16] to [21] above, I have determined that the defendant should have done so by 1966.

[38] The plaintiff contends that by 1961 the defendant had a duty to warn users of its products of the risks of harm and the appropriate steps to be taken to reduce those risks. The defendant accepts that by 1978 it had such a duty of care. It is common ground that after 1978 the defendant took steps to warn its consumers of those risks by attaching warning labels to its products. The defendant contends that the labels were adequate to warn of the risks involved when using its products. The submission of the plaintiff is that the warnings were inadequate and given far too late. Reference was made by Senior Counsel for the plaintiff to the decision of the New South Wales Dust Diseases Tribunal in *(Re Lorizio) A B and P Constructions Pty Limited*

31 Tr p 242.

Cross Claimant v Amaca Pty Limited (formerly James Hardie & Co Pty Limited[32] that criticized the steps taken by the defendant and found them to be grossly inadequate. The criticisms made by Duck J included the fact that the warnings made no mention of catching mesothelioma, cancer or any other lung disease; the note suggests that it was affixed as a matter of form and that there was really no problem.

[39] The warning is contained in Exhibit D2 at page 114. It states:

> Caution
>
> This product contains asbestos.
>
> Breathing asbestos dust can damage health.
>
> Keep dust down.
>
> This product contains a small percentage of asbestos bound firmly into it by cement and other materials. Asbestos dust can damage health. Keep dust down by following these simple safety rules:
>
> 1. When sawing, drilling, etc. work in a well ventilated space, preferably outdoors.
> 2. Use hand tools designed for cutting asbestos-cement sheets such as fibro cutters or a score and snap knife – or use an old hand saw. If cutting by power saw use one which has a dust suppression attachment.
> 3. Avoid drilling overhead where possible by pre-drilling before fixing.
> 4. Damp down waste dust and dispose of in a sealed bag.
>
> NOTE: When fixed in position, asbestos cement sheets present no known risk to health.

[40] Duck J also stated at [32]:

> These cautions contain no reference to contracting mesothelioma, cancer or other lung diseases. They are anodyne in the extreme. The

32 [2006] NSWDDT 19 at [33-34].

note with which the caution is finished tends to convey the idea that that there really is no problem and that this cautionary label is being affixed as a matter of form.

[41] In my opinion, for the same reasons given by Duck J, the above warning is inadequate to bring the risks involved in using the product to the attention of a tradesman such as the deceased working in the building of houses where asbestos cement sheeting was going to be used extensively in fabricating external walls, ceilings, internal walls, and in kitchens, toilets and showers.

[42] There is also another warning sticker in evidence. It has not been clearly established that this warning sticker was used in the Northern Territory. The warning on the sticker states:

Caution

Contains asbestos fibre.

Avoid creating dust.

Breathing asbestos dust may cause serious damage to health including cancer. Smoking greatly increases the risk.

In accordance with N.H. & M.R.C. recommendation.

This is an AC building product manufactured by James Hardy and Coy. Pty. Limited, which contains a small percentage of asbestos bound firmly in by cement and other materials.

When cutting or drilling follow these simple safety rules.

1. When sawing, drilling, etc. work in a well-ventilated area, preferably outdoors.
2. Use hand tools designed for cutting asbestos-cement sheets such as fibro cutters or a score and snap knife—or use an old hand saw. If cutting by power saw use one which has a dust suppression attachment.
3. Avoid drilling overhead where possible by pre-drilling before fixing.
4. Dispose of any waste and dust in a safe manner.

> NOTE: When fixed in position, asbestos cement sheets present no known risk to health.

[43] It is unclear on the evidence before the Court whether this sticker came into use in 1978 or 1979 at a later date. In *(Re Lorizio) A B and P Constructions Pty Limited Cross Claimant v Amaca Pty Limited (formerly James Hardie & Co Pty Limited* Duck J suggested that it came into existence in 1982.

[44] In my opinion, the above warning sticker is also inadequate. The warning it provides does not accurately state the degree of risk of harm involved in using fibro products in the construction of houses. The effectiveness of the warning is significantly reduced by the inclusion of the words: "which contains a small percentage of asbestos bound firmly in by cement and other materials." Nor does the warning adequately state what dust suppression techniques and safety masks must be used when power tools are being used to cut and apply the materials. I also agree with Duck J's criticism of the warning sticker as follows at [33]:

> The only difference relevantly between this caution and the one before mentioned is that two words are slipped in, that is to say "including cancer". The same comments apply in respect of it. Further its form is an imitation of the form which had previously prevailed, and how anyone is likely to pick reference to the words "including cancer" is not clear. It is to be remembered that these materials are to be used by people doing building work. I do not think that a caution in that form is sufficient to attract the attention, firstly, of those to whom it is directed. Secondly the content of the caution is insufficient to bring home the real risks associated with the inhalation of asbestos dust.

[45] The evidence of the deceased was that he had no recollection of ever seeing any such warnings. He was not aware asbestos dust was dangerous and could

cause injury, including asbestosis and cancer. Given the date that the offender stopped work, the second warning would be of little value if it was given in 1982. However, the question is not whether the deceased read the warning labels but whether, if he had read them, they were suitable to draw attention to the relevant risks. In my opinion, for the reasons given above, they were not and I find that the defendant breached its duty of care to the deceased.

Causation

[46] There are a number of factual matters to be considered before dealing with the expert evidence. First, the deceased was a smoker. According to his statement, he began smoking when he was aged 18 in 1956, and continued to smoke until 2010 when he was aged 72. His consumption of cigarettes increased gradually. By his mid-twenties he was smoking one packet a day. The evidence is unclear whether this means 10, 20, 30 or possibly more cigarettes, as there is no evidence of how many cigarettes there were in the packets he smoked. By the time he was 30, he was smoking up to 55 - 60 cigarettes a day, although the numbers varied to half a packet. Overall, the deceased estimated that his average consumption was one packet of cigarettes per day. This evidence must be taken with some caution, as it is far from clear and was not able to be clarified by cross-examination. It will be necessary to consider the effect of his smoking history on causation of his lung cancer.

[47] Secondly, counsel for the defendant has raised the possibility that the defendant did not always manufacture the fibro cement products the deceased was using. It was put that Wunderlich Ltd also supplied fibro cement products in Darwin. However, the evidence that the deceased might have been exposed to asbestos dust from products made by Wunderlich is minimal. There is a single advertisement published in the NT News in 1961 for Wunderlich products and another in 1975. In the former case, the advertisement states that Burns Philp had a complete range of their products. The evidence is that the defendant acquired Wunderlich in 1974. *Yet no documentation is available to show that Wunderlich had an extensive sales history in the Darwin region*. There is evidence of a letter from the defendant to Wunderlich dated 1961 about the distributors of certain products which shows that there was an agreement that Wunderlich would use Darwin Milling and Trading and the defendant would use Millar and Sandover[33]. The deceased said that the products he used when working for Roussos Brothers were bought from Millar and Sandover usually and were all bought from Bunnings when Millar and Sandover became Bunnings. There is no evidence as to who Darwin Milling and Trading might have been. There is evidence that Wunderlich supplied its products "in the Northern Territory" in 1954 from Adelaide. Presumably, the products were sent - probably by rail - to Alice Springs, but that does not mean they ended up in Darwin as well. The evidence of Mr Hoens was that James Hardie's

33 Tr p48 Ext D5 pp10-11.

products had different names for the same products sold by Wunderlich. For example - corrugated sheets were called “Super Six” (Hardie) and “Deep Seats” (Wunderlich), flat sheets were Fibrolite and later HardieFlex (Hardie) and “Durasbestos” and “Wunderflex” (Wunderlich), “Tilex” (Hardie) and “Duradek” (Wunderlich)[34]. In the deceased’s statement, he refers to using Hardieflex, Hardieplank, Tilux and Versilux. None of these products was shown to be manufactured or distributed by Wunderlich.

[48] According to Mr Hoens, his information from the Perth sales manager, John Ford, was that Darwin was a “Hardie’s town” which he accepted because Wunderlich did not have a factory in Perth, whereas the defendant did, and consequently it would not have been economical for Wunderlich to ship their products to Darwin.[35] However, this line of reasoning is of little or no weight as the evidence is hearsay and speculation.

[49] I find the suggestion that Wunderlich supplied some of the products used by the deceased quite unsatisfactory and unconvincing. That is not to say that no product was purchased from Wunderlich. However, the onus was on the defendant to prove that a significant quantity of product used by the deceased was purchased from Wunderlich. It failed to do so. The evidence before the Court establishes that the defendant was the main supplier of the fibro products used by the deceased.

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34 Tr p43.

35 Tr p45-46.

Deceased's medical history and causes of his death- Dr Burdon's reports

[50] The deceased's medical history is set out comprehensively in the report of Dr Burdon of 10 February 2020[36]. The deceased passed away on 26 November 2019. He was aged 81. The causes of his death, as noted on the death certificate were:

- Liver kidney and heart failure
- Pneumonitis (2 weeks)
- Pneumonia (2 weeks)
- *Stage IV non-small cell lung cancer*
- Atrial fibrillation; ischaemic heart disease
- Gastritis
- Abdominal aortic aneurysm

[51] Dr Burdon refers to the deceased having heart problems going back to a myocardial infarction in the early 1980s and a second one in 2010 which was followed up with a bypass in November 2010 and a pacemaker in July 2017. There was also a transient cerebral ischaemic episode in 2013. The deceased had continuous problems due to his heart condition from 2015 onwards. As far as cancer is concerned, the report refers to a CT scan that was carried out in March 2019 and showed, inter alia, "the presence of asbestos related pleural disease …the left upper lobe mass was biopsied on 25 March 2019 and pathological examination showed histology consistent with moderately differentiated squamous cell carcinoma of the lungs."[37] He

36 ExP1 p1, pps5-8.

37 Ibid, p6

subsequently underwent radiotherapy and immunotherapy. He was too ill for chemotherapy and too old for surgery.

[52] In summary, Dr Burdon concluded the deceased did not suffer from asbestosis but that he suffered from lung cancer that either was caused by, or significantly contributed to, by his exposure to asbestos.

[53] Dr Burdon is a specialist consultant respiratory physician with broad experience in all aspects of respiratory disease and a significant interest in occupational lung disease including managing numerous patients with asbestos related lung disease. He cites three published articles in medical journals that refer to the interaction between asbestos and smoking as causes of lung cancer.

[54] In his second report, dated 20 January 2021,[38] Dr Burdon addresses additional reports provided to him and maintains his previous opinion. Of significance, he referred to reports from various other experts that he states he either accepts in whole (Professor Klebe) or in part (Professor McKenzie) or disagrees with (Professor Fox). As far as Professor McKenzie is concerned, Dr Burdon agrees with his opinion that - “there is an interaction between the risks from smoking and asbestos which is more than additive and probably less than multiplicative and is increased at higher exposure levels”. However, he disagrees with Prof McKenzie’s statement that the exposure to asbestos was “*de minimus*”. Dr Burdon notes that the report of

38 P1 p14

the occupational hygienist Mr Pickford seems to support that view but the report of the other expert occupational hygienist, Mr Kottek, opined higher degrees of exposure and if that is correct, the level of exposure was not *de minimus*. He also maintains that lung cancer was the major cause of the deceased's death.

[55] In his third report dated 30 May 2021[39], Dr Burdon was asked to comment on the reports of three occupational hygienists, Mr Pickford, Mr Kottek and Mr Shepherd, who have each given differing opinions of the total fibre burden caused by exposure to asbestos cement. In response, Dr Burdon states that, having considered their opinions, he remains of the opinion that the deceased's exposure to asbestos made a material contribution to his cancer. He refers specifically to the opinion of Professor Klebe and to an article she and others wrote and was published in 2020[40] that states, "any asbestos exposure even in a heavy smoker, contributes to causation."[41]

[56] Dr Burdon's reports were tendered without objection as part of Ext P1.

[57] In cross-examination, Dr Burdon conceded that he did not calculate the dose of the deceased's exposure to asbestos particles, but relied on the opinions of an occupational hygienist to get an accurate assessment.[42] He said that he took Mr Kottek's assessments at 180 fibre mil years or 90 if the work was

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39 Ex P1 p23.

40 S. Klebe et al *Asbestos, smoking and lung cancer: an update*, published in Int J Environ Res Public Health 2020;17;248 which became Ext C.

41 Ibid, referred to in the third report of Dr Burdon dated 30 May 2021, Exhibit P1 p 25.

42 Tr p168.

done outside, as being reasonable. He was aware of Mr Pickford's assessment of 19.3 fibre mil years.

[58] In his second report, Dr Burdon was asked to comment on the following statement of Associate Professor McKenzie.

> ... the squamous cell carcinoma was most likely caused by smoking with a possible contribution from asbestos. Using an asbestos exposure of 10f/mL years and smoking of 50 pack years, the asbestos component is less than 1% and therefore, in my opinion, de minimus.

[59] Dr Burdon reported:

> I would agree with Professor McKenzie's first comment and with his second if it were correct. It would appear that Professor McKenzie has assumed that "Mr Roussos may have been exposed to an estimated total airborne asbestos cumulative exposure (dose) of approximately 19 fibres/mLyears" see the report of Mr Geoffrey Pickford dated 11 October 2020.

He then went on to state in his report that he preferred the evidence of Mr Kottek.

[60] During his cross-examination, Dr Burdon was asked the following questions and gave the following answers about the above matters.

Counsel:	And you will see that you put two bullet points in relation to an opinion stated by Professor McKenzie where he said that the asbestos component could be dismissed as de minimus. And you go on to say, "I would agree with Professor McKenzie if it is correct". And by correct you were referring to the dose of asbestos?
Dr Burdon:	Correct, yes.
Counsel:	And you go on to note that Professor McKenzie was concluding that, on the basis of different assessments, one by himself and another by Geoffrey Pickford?

Dr Burdon: Correct.

Counsel: So, you would agree that the role of asbestos can be dismissed in some cases as de minimus?

Dr Burdon: Based on – yes, in some cases. I would have to accept that if the dose was what Professor McKenzie believed it to be, then the – the risk – or I could say the contribution of the asbestos to the development of this man's lung cancer, would be extremely small.

Counsel: But you know that Professor McKenzie made his assessments assuming a wide range of doses of asbestos, don't you?

Dr Burdon: I am aware of that.

Counsel: What you were fixing on was the fact that he said, for example, with the asbestos as less than 1 percent, that it could be dismissed as de minimus?

Dr Burdon: That may be what Professor McKenzie was saying, I'm saying any exposure is significant

Counsel: Well, hold on. Professor McKenzie was assuming an exposure. And you agree with him, that it went up to 90 fibres per millitre years, that that could be dismissed as de minimus?

Dr Burden: What I said was, it could be dismissed – as being extremely small. I did not use the term de minimus.

[61] Much of the remainder of the cross-examination was directed towards his knowledge of the relative experience and value of these differing opinions, which in the end result, may depend upon what I find as a reliable assessment (if indeed any assessment is reliable). In relation to the cross-examination generally, Dr Burdon tenaciously stuck to the opinion, supported by Professor Klebe, that any exposure to asbestos is a contributor to cancer. He said that there was no lag period where the risk of cancer from exposure to asbestos reduces to a lower figure over time. In cross-examination, reference was made to a study by Jarvholm and Anor that

suggests that 20 years after significant exposure to asbestos, the risk of cancer drops down to the same as a person with very low exposure.[43] The witness replied that there was still a significant risk.

The report of Associate Professor Klebe of 9 October 2020

[62] Associate Professor Klebe is a consultant pathologist who was called to give evidence by the plaintiff. The witness is highly qualified in asbestos-related disorders, having written numerous published papers and other works. She is a member of various international and Australian panels relating to the study of lung cancer. [44] She has personally examined and reviewed more than a thousand cases of mesothelioma as well as lung cancer and lung cancer precursor lesions as well as numerous other cases of benign asbestos related diseases of various types (plural plaques, diffuse pleural fibrosis, benign asbestos pleuritis with effusion, rounded atelectasis, and asbestosis). Undoubtedly, her expert credentials are impeccable. Her report is very detailed and references some 109 articles and papers from medical journals and other sources.

[63] Associate Professor Klebe stated the following in her report dated 9 October 2020:

- No safe threshold level of exposure to any type of asbestos has been identified for lung cancer.[45]

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43 Tr p174-175.

44 Ext P1 p28 at 30-31.

45 Ext P1 p 28 at p3 of the report dated 9 October 2020.

- Lung cancer is the result of a series of mutations in the lung parenchymal or bronchial cells. When asbestos fibres are inhaled, a proportion is deposited in the lung tissue, causing multiple mutations that together eventually lead to a clinically detectable carcinoma. The retained fibres have ongoing effects on the surrounding tissues, causing multiple mutations, but each additional inhalation episode adds further fibres that also have these effects.[46]
- Each episode of being exposed to asbestos as a result of working with asbestos building products made a material contribution to the development of squamous cell lung carcinoma in Mr Roussos.[47]
- The latency interval between Mr Roussos' exposure during his employment from 1958 to 2000 (sic - should be 1983) and the diagnosis of his lung carcinoma in 2019 (approximately 61 years later) falls into the range of latencies acceptable for a causal contribution from asbestos to the development of lung cancer.
- The prevailing evidence indicates that the relationship between cumulative asbestos exposure and lung cancer is governed by a "no-threshold linear dose-response model". Inherent in the model is that each episode of exposure contributes to the cumulative dose, and hence to the development of the tumor.[48]
- Where there is exposure to both smoking and asbestos, they both materially contribute to the development of lung cancer. The effects cannot be separated out at a biological level.[49]
- The prevalent current consensus view is that asbestos exposure and tobacco smoking interact synergistically for the causation of lung cancer. Asbestos participates in both the initiation and proliferation phases of tumor development together with cigarette smoke.[50]
- All cumulative exposure to asbestos plays a contributory role in causation of the tumours, that cigarette smoking increases the binding of asbestos fibres to lung epithelial cells which become damaged, some of which become malignant and proliferate at different times. Components of cigarette smoke impair clearance of particulates, including asbestos fibres, from the upper airways. The more there are fibres, the more there are free radicals and the greater the probability of genetically damaged and proliferating cells at any given point of time. The cigarette smoke would have enhanced the

46 Ext P1 p 28 at p3 of the report dated 9 October 2020.

47 Ibid

48 Ext P1 p 28 at p6 of the report dated 9 October 2020.

49 Ext P1 p 28 at p7 of the report dated 9 October 2020.

50 Ext P1 p 28 at p7 of the report dated 9 October 2020.

retention of the fibres. Ultimately "the lung cancer in Mr Roussos is the result of cumulative mutations and epigenetic alterations, caused by asbestos and smoking', and "cannot be partitioned off into the individual effects of smoking and asbestos."[51] From a pathobiological perspective and as a matter of definition, the interactive effect cannot be partitioned into the individual effects of smoking and asbestos.

- The interaction of multiple carcinogenic factors together happened at a biological/cellular level and is not just a statistical interaction. Asbestos causes chronic inflammatory changes, which release cytokines (including growth factors), providing a selective growth advantage for cells, which have undergone cancerous mutation due to carcinogens in the tobacco smoke or due to asbestos itself.
- Referring to Mr Kottek's assessment of the deceased's exposure, she notes that a significant part of the exposure occurred before 1975, Mr Kottek limits his calculation to the active working time which may underestimate the exposure having regard to the fact that the deceased was working in the close proximity of others who would have continued to use electric saws etc. even when he was not; the fibres can be suspended in the air for a considerable period of time; the fibres would have accumulated in the deceased's hair and clothing and be resuspended into the air by activities such as brushing the hair and removing and shaking clothing in preparation for washing. In her opinion the exposure was well above the 25 fibre/mil years based on her own experience.[52]
- There is radiological evidence of significant exposure to asbestos in the form of pleural plaques.

The nub of the defendant's case

[64] It will be necessary to refer to the cross-examination of Associate Professor Klebe in due course, but at this stage, it is convenient to set out the nub of the defendant's case as presented by its expert witnesses. In essence, Senior Counsel for the defendant submitted that:

- It is possible to calculate the deceased's cumulative level of exposure using certain data to arrive at a time-weighted average, which reflects the average exposure over the working shift. Once

51 Ext P1 p28 at p 8 of the report dated 9 October 2020.

52 Ex P1 p28, at p10-12 of the report dated 9 October 2020.

this is done, it is possible to arrive at a calculation made by reference to the number of years the exposure occurred. The result is a figure expressed as fibres per millitre years, commonly expressed as f/ml/y.

- The evidence of Mr Pickford was that the exposure was 19 f/ml/y. The evidence of Professor McKenzie was that it was likely that the dose was less than 10 f/m/yr. The evidence of Mr Kottek was between 90 and 180 f/ml/y. The evidence of Mr Pickford should be preferred.
- The risk of contracting lung cancer from smoking is best determined by calculating the cumulative dose expressed in "pack years". This is a simple calculation, which in this case is the average number of cigarettes smoked per day multiplied by the number of years of smoking. In this case, the pack years based on the evidence that he smoked an average of three packets or 60 cigarettes per day over 54 years resulting in 192 pack years.
- The deceased developed squamous cell carcinoma, which was diagnosed in March 2019.
- The medical science of epidemiology uses the concept of "relative risk" ('RR") to calculate the extent to which a particular agent has increased the risk of a particular outcome, in this case, exposure to asbestos causing cancer.
- The calculation is more complex when there is more than one agent involved. This was an issue dealt with by the High Court in *Amaca Pty Ltd v Ellis*.[53]
- The relative risk due to smoking alone was calculated by Dr Leigh as 63 based on an assumption of 54 pack years. The plaintiff called this witness. It was submitted that this opinion was grossly understated as the court should find that the number of pack years was 192.
- Professor McKenzie, who was called by the defendant, gave evidence that the relative risk was between 49 and 120 depending on the number of pack years. The RR of 120 was based on 150 pack years, and would have been even greater if the type of cancer considered was squamous cell type.
- All the experts except Dr Burdon accepted that the fact that the deceased developed COPD substantially increased the risk of contracting lung cancer through smoking, which is the cause of this condition in the deceased. Dr Leigh accepted that COPD can increase the RR by as much as 3 to 6 times.

53 (2010) 240 CLR 111.

- Dr Leigh estimated that the RR from asbestos alone was 3.3 but that estimate was based on assumptions that were wrong, viz, that the asbestos emanating from fibro asbestos cement was 50% amphiboles and 50% chrysotile.
- The evidence of Mr Pickford and Professor McKenzie to the effect that the RR from exposure to fibro asbestos cement was minimal should be accepted.
- Further, the passage of time from the last exposure to the development of cancer reduced the RR even further, and it may be that the exposure to asbestos had nothing to do with the contraction of cancer.
- The argument of the plaintiff that the interaction between tobacco and asbestos is either additive or multiplicative is unsettled as a matter of medical science. Professor McKenzie calculated that, even on the basis that it was multiplicative, the gross effect was overwhelmed by the increased risk due to smoking alone, a conclusion that Dr Leigh said was "not unreasonable."
- The evidence of the defendant's expert that was based on epidemiology was to be preferred to that of the plaintiff's experts, Associate Professor Klebe, Dr Leigh, Dr Burton, and Mr Kottek.

[65] One problem with the epidemiological evidence, which stands out, is the huge discrepancies between the opinions of the various experts. These discrepancies appear to be related to the assumptions made about the number of cigarettes per day the deceased smoked, the number of hours the deceased worked when he would have come into contact with asbestos, whether the asbestos products contained equal quantities of amphiboles and chrysotile, and whether this would have made a significant difference. For example, the plaintiff's expert Dr Leigh calculated the deceased's RR from smoking based on an average of 20 cigarettes per day[54] (the average according to the deceased's statements)[55] whereas the calculation relied on by counsel for the

54 Dr Leigh's report, extP1, 6; p5 of the report.

55 Ext P2 pp11-12.

defendant was based on an average of 60 cigarettes a day, which was in turn based upon contemporaneous medical records, according to which the deceased gave a history of smoking 60 a day. This was used to calculate the number of pack years, calculated to be 192 pack years, not RR.[56] The difference between the calculations for RR is dealt with in the defendant's submissions[57] where it is put that the RR of 63 arrived at by Dr Leigh "was based on an assumption that the cumulative dose was 54 pack years... In other words, it is a gross underestimate of the true risk." There is little doubt that the medical records referred to by counsel for the defendant support prima facie a finding that the deceased's actual smoking rate was likely to be higher than 20 a day. Dr Burdon calculated the pack years as being something in the order of 50 pack years, but he said that getting the history from the patient is difficult and you would likely get different results on two different occasions. It is always possible that the true number could be as high as 150.[58] An obvious problem with the defendant's calculations is that it assumes a constant average of 60 cigarettes a day from the age of 18 until he gave up smoking in 2010. The contemporaneous medical records only go back to the early 1980s whereas the deceased began smoking in 1956, and it is inherently unlikely that the deceased started consuming 3 packets of 20 cigarettes when he first started. The deceased said in his statement that it was not until he reached the age of 30 that he began to

56 See defence submissions p 9 based on records contained in ext D4.

57 P11.

58 Tr p167-168.

smoke up to 58-60 cigarettes daily.[59] Apart from the brand names (Rothmans Blue and Peter Taylor tailored cigarettes) we know nothing about whether the cigarettes were filter tipped or not, or anything else likely to be relevant to the amount of the carcinogens to which the deceased was likely to have been exposed from cigarette smoking. The evidence of the deceased's RR from smoking cannot be accurately calculated which explains why the defendant's expert, Professor McKenzie, came up with three assumed cumulative doses of RRs of 49, 73 and 120 depending on the number of pack years used for the calculation[60]. It was put that these calculations did not take into account in calculating the risk that the cell type was squamous, and if that fact had been added to the mix, the risk would be even higher. I find that it has not been proven that the RR for smoking was as low as between 50 to70. It is likely to be more than that - possibly as high as an RR of 120 to 150. Beyond that, no accurate calculation is possible.

[66] The next area of contention is the differing opinions about the RR from asbestos. Once again, the opinions of the experts differed markedly, depending upon the factors taken into account in the calculations. Three occupational hygienists, Mr Kottek, Mr Pickford and Mr Shepherd gave evidence on this topic. Mr Kottek estimated the cumulative exposure was 180 fibres per ml. This estimate was based on the deceased cutting asbestos products for 30 minutes a week using power tools. If the work was done

59 Ext P2 p11.

60 Ext D1 pp10-11.

outdoors, the cumulative exposure is around 90 fibres/ml.[61] Mr Pickford estimated a total airborne asbestos cumulative exposure (dose) of approximately 19 fibres/mil year,[62] and Mr Shepherd's estimate was "somewhere between those levels reported by Mr Pickford and Mr Kottek."[63] In some detail in his report, Mr Shepherd dealt with why it is not possible to get an accurate calculation of the amount of exposure to asbestos using this methodology. After setting out a long list of variables and assumptions that are often made,[64] Mr Shepherd observes:

> Mr Roussos' cumulative exposure to asbestos dust is very difficult to determine for the reasons outlined above. Any determination of a value is speculative at best and dependent on numerous factors which can be challenged by industry experts from opposing sides. By slightly adjusting the many variables used to estimate cumulative exposures we can obtain significantly different results.[65]

[67] Notwithstanding this criticism, the report of Mr Pickford is very detailed and appears prima facie to give reasonable weight to the many variables, whereas the report of Mr Kottek relies on experiments carried out by the author that have not been peer reviewed. However, counsel for the plaintiff submitted that the estimate of 19 f.ml/yr. by Mr Pickford is unreasonably low in view of the evidence of the deceased about the extent to which he used power tools to cut asbestos sheets including compressed asbestos sheets. It was submitted that no allowance was made for the fact that the

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61 Ext P1 p187.

62 Ext D1 p103.

63 Ext P1 p6 of the report.

64 Ext P1 p3 of the report.

65 Ext P1 p4 of the report.

deceased used compressed thick sheeting for floors, decks and patios; that he was likely to have used a power saw or angle grinder equipped with a masonry disc to cut this material, which was likely to have generated dust up to 90 ml/yr. I accept the plaintiff's submission that Mr Pickford failed to give appropriate weight to the deceased's evidence about the extent to which he used power tools. Nor did Mr Pickford give appropriate weight to the deceased's evidence about the amount of time he spent working with asbestos in the particular circumstances of the Northern Territory. The plaintiff submitted that, if the estimate of 20 minutes per day use of power tools was increased, the resulting exposure increased in a roughly linear manner.[66]

[68] Mr Pickford dismissed the plaintiff's suggestion that his estimate that the deceased only cut asbestos cement for 20 minutes a day was too low. His estimate was based on an assumption that on average the deceased used a power saw for 4% of a standard 8 hour day[67], and assumed an average working week of 40 hours[68], despite the deceased's evidence that he averaged 6 hours per day working on fibro[69]. Some of the factors relied upon by Mr Pickford were based on his own experience in observing housing construction. What is not clear is whether the data and factors considered would be affected by the fact that the deceased was working in hot tropical

66 Mr Pickford, tr p249.6.

67 Ext D1 p 121; Tr 248.5.

68 Ext D1 para 30 of the report.

69 Ext P2 p 22.

conditions where day-time maximum temperatures almost always exceeded 30 degrees centigrade every day of the year; where one would have expected that in those circumstances the work would have been performed under cover whenever possible to escape the heat, and in the wet season, the rain; and where it is likely that the work would have been performed under the floorboards of houses being built on piers, which was typical of such houses at the time, with limited access to breezes; and the deceased's evidence that even when he was not cutting fibro, he was often in the presence of other workers who were and he was exposed to the dust they created.[70] As Mr Shepherd observed in his report, very small adjustments using power saws or grinders produce significantly different outcomes.[71] Accordingly, an increase in the estimate of the time the deceased used power tools from 4% to 10%, together with a 5% decrease in the estimate of him carrying and handling asbestos concrete, and a 1% decrease of the time he spent measuring, yields a doubling of the time weighted average of his total exposure to 2.0 fibres/ml. Further, there is evidence that the standard exposure of 25 m/L year is only one of three accepted criteria at which exposure to asbestos is sufficient to infer causation. The other two criteria are heavy exposure for at least a year, or a moderate exposure of 5 to 10

70 Ext P2 par (46), p10.

71 Ext P1 p232

years in construction or shipbuilding. The defendant's expert, Dr McKenzie, conceded that the deceased fitted the third of these criteria.[72]

[69] Having considered the evidence of Mr Pickford, Mr Kottek and Mr Shepheard, I find that the deceased's exposure to asbestos was more than 25 fibre/mil-year which is generally regarded as the minimum level of exposure required to cause lung cancer by exposure to asbestos. In so doing I have adopted the approach taken by Mr Pickford, but consistent with the evidence of the deceased, I have allowed for a six day working week, a 10 hour working day, the use of power tools for 40 minutes a day to cut asbestos cement (an even greater time could have been involved if there was a division of labour and the deceased mainly cut the asbestos fibro in order to manage his work crew from a central position), and he cut the fibro underneath the houses that he was constructing to minimise his exposure to the sun. It is also important to note that the deceased and his brother had teams of men working for them and from time to time, they were building more than one house at a time. If more than one house is being constructed by a team of men logic and the division of labour would dictate that one man may be cutting the fibro cement for more than one house.

[70] The defendant submitted that the correct approach to the issue of causation requires as a first step that the state of medical science presently cannot explain the biological mechanism through which lung cancer is caused.

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72 See par (90) infra.

Therefore, it was submitted that the Court and the experts must rely on epidemiology and statistics. In support of this argument, reference was made to four cases, the first of which is *McDonald v State Rail Authority*.[73]. In that case, there was no radiographic, clinical or histologic evidence that the deceased had asbestosis. The trial Judge dealt with two conflicting theories about whether or not exposure to asbestos, which did not result in asbestosis, could cause cancer. A number of experts were of the opinion that, unless there was asbestosis, there was no connection between cancer and asbestosis: the "necessary precursor hypothesis". The plaintiff's experts were of the opinion that this was not necessary. They supported the "fibre burden hypothesis". After reviewing the literature and the evidence in some detail the trial Judge concluded that carcinoma of the lung may be attributed to asbestos exposure in the absence of asbestosis where the exposure was sufficient to cause asbestosis. The minimum level of exposure required was accepted as being 25 fibre/mL-year. The trial Judge said that, in the absence of "real" evidence, it was still open to him to determine the level of exposure by inference or by estimation, but on the evidence he was unable to find what the cumulative exposure was. Accordingly, the case was dismissed. That case is not authority, let alone binding authority, for the proposition for which it is cited, but turned on its own facts. It should be noted that in the case before me, for example, there is radiological evidence

73 (1998) 16 NSWCCR 695.

of "calcified pleural plaques indicating previous asbestos exposure"[74], the importance of which I will come to later. However, that is not to say that the principles applied by the trial Judge to decide if legal causation was proven were incorrect.

[71] The next case relied upon by the defendant is *Judd v Amaca Pty Ltd*.[75] In that case the claimant was also a smoker of many years who had been exposed to asbestos for a substantially shorter period of time than in the present case. The evidence of the plaintiff's experts depended to some degree upon what was called "the Helsinki report", which adopted a statement that the relative risk after exposure to asbestos is doubled if the exposure reaches the threshold of a cumulative exposure of 25 fibre years; cumulative exposures of less than 25 fibre years are also associated with an increase in relative risk but to a lesser extent. The trial Judge held that the Helsinki Report was "a position statement from an expert group" and not a document which is able to be seen as drawing together expert identified sources upon which it relies; accordingly, on the point at which the relative risk doubles, it was not based on reliable epidemiological evidence and was therefore valueless. He held that the figure was more likely to be 50 fibre years, but that as the figure of 25 fibre years had not been reached by the plaintiff, his finding was not essential to his reasoning. The court in that case was not convinced that the plaintiff would not have contracted cancer if

74 Report of Dr Michale McComish 28/1/2021 Ext D1 p3.

75 [2003] NSWDDT 12.

he had not been exposed to asbestos; and further the court was not persuaded that the cancer caused by smoking had been accelerated by being exposed to asbestos because the low increase of RR=1.1 meant that any contribution from asbestos would have been minimal. Consequently, the claim failed.

[72] It is not clear to me why I should treat the case referred to above as bearing on the question I have to decide in this case. The facts are very different, and as stated by French CJ in *Amaca Pty Limited v Booth*,[76] my duty is to make factual findings on the evidence before me, not on some global consensus. Section 25B of the *Dust Diseases Tribunal Act 1989* provides that an issue decided in the Tribunal, whether between the same or different parties, may not be re-litigated without leave. There is no such provision in this jurisdiction. It follows that I am not bound to make the same findings as were made in *Judd* even if the facts were the same in each case (which they are not).

[73] Next, Senior Counsel for the defendant relied on the unanimous decision of the High Court in *Amaca Pty Ltd v Ellis.*[77] In that case, the late Mr Cotton died from lung cancer. One of the issues was whether his exposure to asbestos was a cause of his cancer and therefore of his death. The facts were that the deceased was a smoker who had been exposed to asbestos whilst working for three different entities. In this case, his smoking averaged 15-20 cigarettes a day for 26 years. Despite succeeding at first instance and on

76 [2011] HCA 53; 246 CLR 36 at [33]; see also at [72] per Gummow, Hayne and Crennan JJ.

77 [2010] HCA 5; 240 CLR 111.

appeal, the High Court found for the defendants on two bases. First, no effort was made to distinguish between the responsibilities of each of the defendants on a causal basis. As the Court pointed out, what needed to be proved was that it was more probable than not that the negligence of each of the defendants caused the plaintiff's death. The Court said that there was no attempt to attribute responsibility to each of the defendants separately. Deciding only that the aggregate of the exposure was a cause did not answer the question of the particular responsibility of any defendant.[78] Secondly, the only evidence relating to causation was epidemiological; no evidence was lead of any medical evidence to explain the cause of his cancer.[79] The argument of the plaintiff was that the epidemiological evidence showed that there was an interdependent operation of two carcinogens. However, the proposition that smoking and asbestos *must* work together to cause cancer was not established on the evidence. Whilst this may be the case in some cases, there was no evidence that this was so in this particular case.[80] Further, it was not shown "to be more probable than not that asbestos was a cause of (a necessary condition for) his cancer. It was not shown that exposure to asbestos made a material contribution to his cancer. Material contribution was not shown because a connection between Mr Cotton's inhaling asbestos and his developing cancer was not demonstrated."[81]

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78 Ibid, at [42].

79 Ibid, at [16].

80 Ibid, at [53]-[54].

81 Ibid at [65].

Knowing that exposure to asbestos can cause cancer does not mean that in the case before the High Court it probably did.[82]

[74] Counsel for the defendant submitted that the arguments of the plaintiff in this case are indistinguishable from the case that was put to and rejected by the High Court. Counsel for the plaintiff sought to distinguish *Ellis* on the basis that Professor Klebe's evidence did not rely solely on epidemiological evidence. Her opinion was also based on pathological biological theory[83] and that in this case there is actual evidence of the cancer being caused by asbestos exposure. As counsel for the plaintiff put it in his written submissions, the plaintiff's case does not depend solely or even predominantly on epidemiological evidence. I note that the submission was made by both parties in *Ellis* that in order to prove causation the proper test was the "but for" test, the High Court observing that in these circumstances the case was not a proper one for considering decisions on that topic in the House of Lords or the Supreme Court of Canada.[84] No similar submission was made in this case, although it may be inferred from defence counsel's reliance upon *Ellis* and other cases cited that this was inherent. In these circumstances it is necessary to return to the evidence of Associate Professor Klebe and the other expert evidence called by the plaintiff and the defendant bearing on the question of whether it has been proven that the deceased's cancer was caused by exposure to asbestos.

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82 Ibid at [68]-[70].

83 Tr p 351.2.

84 *Amaca Pty Ltd v Ellis* [2010] HCA 5; 240 CLR 111 at [12].

Report of Dr Slaughter

[75] An important starting point is what was found radiologically immediately prior to the deceased's death. According to the report of the radiologist, Dr Slaughter, [85] who examined the deceased's X-rays and CT scans covering the period from 9/5/2011 to 11/10/2019,

> Mr Roussos has evidence of bilateral pleural plaques. These have progressed significantly over the period of observation with increased extent of calcification. The appearance is typical of benign asbestos related pleural disease and confirms previous asbestos exposure. Mr Roussos had a left upper lobe mass demonstrated on a CT scan in 2016 and this was shown to enlarge on subsequent imaging. Biopsy showed squamous cell carcinoma and there was a little regression of the lesion following treatment. Adjacent pneumonitis was most likely due to radiotherapy and some airways obstruction from the tumour. The tumour involved the mediastinal and hilar lymph nodes. The tumour is most likely a primary lung carcinoma. The plural effusions at this stage in the left lung are almost likely due to left lung cancer…
>
> In the presence of this underlying cardiac disease, it is very difficult to make a radiological diagnosis of interstitial lung disease due to asbestosis as the early changes before cardiac failure was evident, were extremely mild. Under these circumstances, I do not believe that a diagnosis of interstitial lung disease due to asbestosis can be made even on the balance of probability from the images provided.

Evidence of Associate Professor Klebe

[76] Associate Professor Klebe's opinion was, in short, that exposure to asbestos fibres in the deceased's case was a cause of his cancer because (1) asbestos fibres have the capacity to be involved at all stages of tumour development; (2) all cumulative exposure plays some part in the causation of tumour; (3) asbestos fibres increase the uptake and metabolism of polycyclic aromatic hydrocarbons by lung epithelial cells; (4) the best carcinogens in cigarette

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85 Ext P1 p176 at p6 of the report.

smoke are the polycyclic aromatic hydrocarbons; (5) cigarette smoke increases the binding of asbestos fibres to lung epithelial cells; (6) these cells are being genetically damaged, some of which become malignant; (7) malignant cells proliferate at different times; (8) DNA repair processes are occurring or may be impaired and oncogenes and suppressor genes are being activated or inactivated; (9) As Mr Roussos was a cigarette smoker at the time of his exposure, the smoke would have increased bindings of asbestos fibres, decreased clearance due to the impaired function of cilia, and may also have had a general immunosuppressive effect; (10) this would have impaired the removal of genetically damaged (malignant) cells and promoted survival of malignant cells and hence tumour growth; (11) components of cigarette smoke have been shown to impair clearance of particulates, including asbestos fibres, from the upper airways; (12) the more fibres, the more there are free radicals, the greater is the probability of genetically damaged and proliferating cells at any given time; (13) the cigarette smoke would have enhanced the retention of the fibres.[86]

[77] Ultimately, Mr Roussos' lung cancer was the result of cumulative mutations and epigenetic alterations, induced by smoking and asbestos.[87] The interactive effect cannot be partitioned into the individual effects of

86 Ext P1 report p28, at p7.

87 Ibid, at p8.

smoking and exposure to asbestos[88] which is generally accepted as being multiplicative.[89]

[78] The second part of Associate Professor Klebe's report considered the report of Mr Kottek to see if the statistical analysis made by that occupational hygienist supported her own conclusions. It did not otherwise inform the main basis of her opinion. In cross-examination, she said that these calculations can be "very variable, and quite unreliable in my experience".[90] She maintained that while there is a higher risk of cancer the greater the exposure to asbestos, there is no safe level of exposure.[91] She also maintained that this was not a case where there was a risk of lung cancer from either smoking or asbestos. "We are not talking about a risk here. The risk has materialised. Mr Roussos [had] a lung cancer and that lung cancer [had] a unique set of mutations that are - that have manifested in cancer."[92] It was put that this was her theory to which she replied: "It is not my theory. It is the basis of current scientific evidence."[93]

[79] It was submitted that if Associate Professor Klebe's evidence is accepted, the link between asbestos exposure and cancer has been proved, and that the case of *Ellis* is distinguishable. Before reaching any conclusion about that submission, it is necessary to turn to the rest of the expert evidence.

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88 Ibid.

89 Ibid, p9.

90 Tr p69.6.

91 Tr pps 69-70.

92 Tr p72.5.

93 Tr p 72.6.

Dr James Leigh

[80] This witness is a consultant occupational physician specialising in occupational respiratory medicine and asbestos related disease for 35 years. After referring to the deceased's work history, he noted in his first report[94] that the deceased was more likely to have come into contact with airborne amphibole fibres than chrysotile fibres. He states that all exposure would have contributed cumulatively to the risk and cause of lung cancer. The detail provided in the history is insufficient to arrive at any reliable quantitative estimate of exposure. After referring briefly to the report of Mr Kottek, he observes that the finding of bilateral calcified pleural plaques is objective evidence of significant asbestos exposure. After referring to a number of epidemiological studies of the risk of cancer in carpenters and construction workers, about which he makes no particular comment, he states that a latency of 63 years from his first exposure in 1956 is "not inappropriate…The effect of ceasing exposure to asbestos on lung cancer risk has not been well studied and is confounded by changes in smoking behaviour."[95] After referring to a number of studies, he concludes that "the best designed studies do not show large decreases."[96]

[81] He then deals with an issue important to the plaintiff's case when he says that "the current consensus view is that asbestos and tobacco smoking interact multiplicatively to cause lung cancer. This has been recognized

94 Ext P1, 93 at p2.

95 Ibid, at p4.10.

96 Ibid, at p6.5.

since 1968…"[97] He then goes on to refer to a number of sources (text books and journals) to support this view. He then refers to a number of studies which consider the possible biological effects and concludes:

> "While the precise mechanism for interaction between asbestos and tobacco smoke in causing lung cancer is not fully understood, a great deal of research has been done over the last 20-30 years. It is not possible in my view to separate their causal effects in the individual case when both have acted, as they must do to some extent, from a purely physio-chemical point of view, and it is thus more probable than not, that in this situation the lung cancer was the singular result of the two factors acting together.
>
> Exposure to either factor alone without the presence of the other is capable of causing lung cancer but when both are present, in my opinion, on the basis of the above biological evidence of interaction, both must have been acting to some extent in the process of carcinogenesis. To assume otherwise would be to reject the existence of physical and chemical laws governing biological behaviour...
>
> All types of asbestos can cause lung cancer. All tumour cell types have been associated with asbestos, and tumours may appear at any lung site. It is my opinion on the balance of probabilities that asbestosis is not a necessary prerequisite for attributing lung cancer to asbestos in an asbestos exposed smoker"[98]

[82] The rest of his report deals with relative risk models and he refers to the relative risk in this case as being "of about 3.5 of lung squamous carcinoma, in combination with tobacco smoking which would have caused a squamous cell risk of about 63. The combined asbestos/smoking relative risk of squamous lung cancer on a multiplicative model is about 230".[99]

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97 Ibid, at p6.10-7.1.

98 Ibid, pp 8-9.

99 Ibid, p18.

[83] His next report, dated 17 February 2021[100] deals with his response to reports by the defence experts, Professor Fox and Dr McKenzie and it is best to deal with that after setting out the opinions of those experts.

Professor Richard Fox

[84] Professor Fox is an honorary consultant in the Department of Haematology and Medical Oncology at the Royal Melbourne Hospital. Whilst his qualifications are not in doubt, the information available is limited to a career as an oncologist which included "the medical side rather than the surgical side" of oncology.[101] This witness concludes that the deceased's lung cancer was "a sequela of his cigarette smoking and not asbestos exposure."[102] The reasons for this conclusion are not entirely clear, but appear to be based on inferences drawn from certain published data to which he refers in his report:

- A paper entitled "Lung cancer and cigarette smoking in Europe: an update of risk estimates and an assessment of intercurrent heterogeneity," by L. Simonato and others. After setting out a table from the report, the witness observes that "this indicates the continued high risk [of squamous lung cancer] from cigarette smoking even in ex-smokers."[103]
- [An unnamed] on-line text by Roggli, in the chapter on pleural plaques it was noted that "various epidemiological studies have clearly established the role of inhaled asbestos fibres in the formation of parietal pleural plaques."[104]After referring to other matters, the witness observes that "it is noted that cigarette smoking

100 Ext P1 164.

101 Tr. P142.4.

102 Ext D1.

103 Ext D1 p4 of the report.

104 Ibid, p4.

interacts with asbestos to greatly increase the risk of development of pleural plaques. The issue re the lack of relationship between pleural plaques and lung cancer appears settled by the recent publication from the Perth WA Group on a recent publication "Pleura, plaques and the rick (sic) of lung cancer in asbestos exposed subjects" by Brims FJH et al, 2020. They concluded [that] the presence of pleural plaques does not confer additional increases in the risk of lung cancer. This is also confirmed in the A/P Klebe report."[105]

- The 1997 Helsinki report that "concluded that plaques alone were insufficient to relate lung (sic) to prior asbestos exposure. Presuming clearly the reason for this is that the frequency of pleural plaques and the presence of plural plaques in asbestos exposed workers appears to relate strongly to cigarette smoking. Therefore, the pleural plaques could be a direct measure of smoking and its incredibly high risk of development of lung cancer." [106]

- A paper by Van Oyen SC et al [107] designed to assess asbestos exposure data in Australia. "They noted as an example in the case of [a] carpenter working in the construction industry the annual average exposure was 0.27 f/mL for each year from 1967 to 1986. This is equivalent over a 30 year period of 0.81f/mL years. This is rather low, however I have reviewed several reports from expert Australian epidemiologists determining exposures similar to that of Mr Roussos at about ~ 4 f/mL years."[108]

- A paper by Luberto et al published in "Environmental Health" in 2019 entitled "Cumulative asbestos exposure and mortality from asbestos related diseases in a pooled analysis of 2 asbestos cohorts in Italy". He cites various figures from this report. It is not immediately apparent to me what conclusion relevant to this case he draws from this paper[109].

[85] Professor Fox was asked for his opinion about various issues based on certain assumptions including that the deceased did not have asbestosis. He was asked to consider a variety of levels of cigarette smoking and asbestos exposure. He stated that his answers to these questions were based on "the

105 Ibid, p4.10.

106 Ibid, p5.1-.4.

107 Ibid, p5.5 the paper is not cited by reference to its publication data.

108 Ibid p5.8.

109 Ibid pp 5.10-7.3.

above description of the relationship of squamous cell carcinoma to the development of lung cancer."[110] He concluded that whether the deceased's smoking level was 50 or 150 pack years, he had an extraordinary high risk of lung cancer; it was almost inevitable. He also stated that following the Luberto review "it is now considered that lower exposures, e.g. 10 to 25 f/mL years are not responsible for the development of lung cancer."[111]

Associate Professor David McKenzie

[86] This witness is a highly qualified associate professor of respiratory medicine with, inter alia, extensive training and experience in the treatment of occupational lung disease. At the beginning of his report he sets out, based on his own knowledge and experience, the processes involved in the construction of a dwelling with asbestos cement exterior walls and other structures. Much of what he reports appears uncontroversial. However, it is interesting to note that the type of dwellings to which he refers are either ground level cottages built on a cement slab or brick piers or brick foundations, or built on wooden posts in some rural settings, as opposed to the almost universal construction of such houses on concrete piers, as occurred in Darwin, which, for example, would mean that the first things to be constructed after the erection of the concrete piers would be the stairs followed by the wooden floorboards. He does not comment on the problem of more than one carpenter working together in close proximity causing

110 D1 p7.10.

111 D1 p8.3.

more dust than would be the case if there were only one carpenter working at the time. No comment is made about whether the work is performed indoors or outside in the open air and if this would make a difference. He also refers to the use of Gyprock for internal walls and ceilings from the 1960s, although there is no evidence as to when this became the case in Darwin and it is not mentioned at all by the deceased. He also observes that a carpenter would spend approximately 25% to 35% of his time handling, cutting and fixing fibro cement materials on a typical build. But, if the carpenter was part of a team which was constructing a number of houses simultaneously, it does not necessarily follow that the level of exposure is the same as would be the case if only one house was built to completion at a time, particularly if the tasks done by individual carpenters were divided up so that those who were involved in constructing floors for example were different from those handling walls and ceilings. There is no evidence that the teams were divided into such differing groups. All we know is that Bill Richardson employed up to 150 different people (who presumably were not all carpenters); that the Christou Brothers operation was much smaller “with a small team of five builders”; that Roussos Brothers at its biggest employed 120 people; and that the work was not limited to just building houses, but included government buildings, schools and conversions and additions to the Darwin Hospital and the University of the Northern Territory as it was then called. The point is that making assumptions based on experience in the southern and eastern states does not necessarily translate appropriately to

the circumstances of this case, and therefore does not exclude the possibility that the estimates of exposure to asbestos may be significantly understated.

[87] In his report, Dr McKenzie states that a carpenter's "shift weighted average exposure would usually be less than 0.4 fm/L. When using predominantly electric tools, including roofers handling corrugated asbestos roofing sheets, the shift weighted average exposure to asbestos would be around 1.2 fm/L."[112] How he arrived at these figures is not apparent from his report. He then extrapolates these figures to state that the cumulative exposure would have been between 5.0 fm/L years to 15 fm/L years depending on whether the tradesman was using power tools for the whole period of his shifts (and was exposed for 50% of the shifts that he worked), or some lesser period of time (5.0fm/L if the average shift weighted exposure was .04%; 10 fm/L if he was using hand tools half the time). Again, it was not shown how these figures are calculated.

[88] Then using the van Oyen data, Professor McKenzie arrives at a calculation of 0.68 fm/L years which is describes as "a relatively low estimate for the type of work that Mr Roussos undertook" and that his own estimate of 5.0 to 10 fm/L years are "conservative and overestimates". [113] He then comments on the reports of Mr Kottek using the van Oyen data, concluding that Mr Kottek must have used unrealistic assumptions in his calculations, while the estimate by Mr Pickford was "in the same order of magnitude" as his

112 Ext D1 p3.4 of the report.

113 Ext D1 p.3.10 – 4.1 of the report.

estimate and similar to his own estimate if Mr Roussos was cutting, handling and fixing asbestos cement through every shift that he worked, which he considered to be unlikely.

[89] Associate Professor McKenzie concluded that, using an asbestos exposure of 10 fm/L years and smoking of 50 pack years, the asbestos component is less than 1% and *de minimus*.

Associate Professor Klebe's report of 19th February 2021

[90] In this report, Associate Professor Klebe comments on the reports of Dr Leigh, Professor Fox, Associate Professor McKenzie and Mr Pickford as follows:

- She agrees with with Dr Leigh's opinion that the exposure to asbestos materially contributed to the deceased's cancer.
- As to Professor Fox, she states that she is struggling to put into context his statement about the connection between pleural plaques and smoking, stating that with an "appropriate exposure history and imaging findings [pleural plaques] are considered a reliable marker of asbestos exposure."[114]
- She strongly disagreed with Professor Fox's opinion that the deceased's level of smoking would inevitably lead to lung cancer. She went so far as to state that Professor Fox's statement was a gross misrepresentation of the risks associated with smoking. Associate Professor Klebe referred to a paper by Bach BP that she stated shows a risk of 8%-11% because of his smoking with an adjustment of 1.24 because of his exposure to asbestos. She said that the deceased had radiological evidence of asbestos exposure – pleural plaques - and an occupational history of asbestos exposure in the construction trade and the Bach data describes the deceased's situation very well.

114 Ext P1 p78 p3 of the report.

- She disagrees with Professor Fox's opinion that smoking was the sole cause of the deceased's cancer, because from a pathobiological point of view the interactive effect cannot be partitioned into the individual effects of smoking and asbestos exposure, and she repeats what she said about this in her first report.

- As to Associate Professor McKenzie's acceptance of the proposition that:

  > … there is an interaction between the risks from smoking and asbestos which is more than additive and probably less than multiplicative and is increased at higher exposure levels.

  Associate Professor Klebe states that:

  > I agree that the higher exposures to either cigarette smoke or asbestos increase the chances of mutagenic events, and that smoke and asbestos together have a more than additive effect (although I consider the effect to be multiplicative …). In accepting the joint effect of asbestos and smoking, Associate Professor McKenzie accepts that the effects of the two carcinogens are linked.

- As to the suggestion that the "asbestos component was *de minimus*," she states that this is at odds with Associate Professor McKenzie's concession that the two carcinogens interact which suggest that the effects cannot be separated.

- She points out that while the association between squamous cell lung cancer and smoking is strong, that does not mean that exposure to asbestos does not contribute to the cancer. In the data Associate Professor McKenzie relies on, viz the Simonalto et al data, no adjustment could be made for occupational exposure in their analysis.

- As to the opinion of Mr Pickford, she refers to a study by Cole et al and observes that their findings "would suggest that even an exposure of 3.3 fibre-years/ml made a biologically relevant, measurable contribution to the development of lung cancer in this cohort. This cohort suggests that even the estimate of Mr Pickford of 19 fibres/ml years would be biologically significant and exert a measurable effect on the development of lung cancer.[115]

- Finally, she comments on the various estimates of exposure to asbestos and cigarette smoking and wonders how reliable those

115 Ibid, p6.10.

> estimates are. She states that it is a moot point, given that in this case there is biological evidence in the form of pleural plaques showing a substantial exposure to asbestos, and a history of exposure to asbestos over many years. She says that the real question is not whether he had 19, 25 or 50 fibre/mL years exposure but whether the asbestos made a material contribution to his lung cancer, which she claims it did for the reasons expressed in her report.

[91] Having reviewed the reports of the various experts Associate Professor Klebe stated:

> I remain of the opinion that the asbestos exposure detailed by Mr Roussos made a material contribution to the development of his lung cancer. The biological effect of smoking and asbestos cannot be separated at a molecular level, but a measurable increase in risk has been shown in many studies when the two factors are combined, even in heavy smokers with low levels of asbestos exposure. The complex biological reality in an individual with a unique genetic background can, in my opinion not be meaningfully modelled by considering a theoretical risk, given that the event (lung cancer) has occurred.

Supplementary report of Dr James Leigh of 1 June 2021

[92] Dr Leigh was asked, assuming a cumulative exposure level of asbestos of 19 fibre/ml-yr, if he remained of the opinion that asbestos materially contributed to the development of lung cancer. Part of his answer was not admitted in evidence. The part admitted related to statistical probabilities only. He answered that, having regard to certain published models, the increase in the risk was non-trivial.

Cross-examination of Associate Professor Klebe

[93] At the start of her cross-examination, Associate Professor Klebe was asked about a study made by Jarvholm and Astrom published in 2014 in the Journal of Occupational and Environmental Medicine. The study concluded:

> This study shows that the risk of lung cancer was dependent on exposure to asbestos in the 1970s because those with high exposure had a higher risk than those with low exposure. Nevertheless, when about 20 years have elapsed since last exposure, there was similar risk for lung cancer among those with high and low exposures (Fig. 2). This is a similar time pattern as for lung cancer caused by tobacco smoking, where the risk after about 10 years starts to decrease in ex-smokers compared with current smokers and continues to decrease during several decades.

[94] Associate Professor Klebe accepted that the deceased's exposure to asbestos dust ended in 1984, which was 35 years before he was diagnosed with lung cancer. Associate Professor Klebe was asked, if in those circumstances and if the findings of Jarvholm and Astrom are applied here, the effect of asbestos in inducing Mr Roussos' cancer might have diminished to zero. The professor replied that she did not refer to the article because it did not deal with the deceased's condition. The study was not relevant to the deceased's case as it excluded people over 75 years of age, and mostly referred to chrysotile whereas the deceased was exposed to amphibole. She said it was largely an "epineuralogical" (?) (most likely- epidemiological) paper best dealt with by Dr Leigh.[116]

[95] Associate Professor Klebe conceded that the length of time that the deceased was exposed to asbestos was important but whether the exposure occurred over 20 years or 40 years was not significant. She said that it was important if one was applying the criteria, presumably referring to the standards used by occupational hygienists. However, this did not neatly fit into the biology. She said that there is no threshold where there is no risk, but there is a

116 Tr p 93.8.

higher risk with higher "doses", by which I take her to mean exposure to significant quantities of asbestos fibres in the air.

[96] She was asked why she referred to smoking in her report, to which she replied, inter alia:

> If you look at the risk of lung cancer from smoking, it is about ten times that for the background population, for asbestos alone, five times. But if you add the two together, the risk is not 15 times. It is 5-0, 50 times. So it is nearly a multiplicative effect. Now, would the smoking have been able to cause lung cancer in Mr Roussos, the smoking alone? Absolutely. Would the asbestos have been able to cause the lung cancer in Mr Roussos alone? Absolutely. But it would not have been the lung cancer that he has died with. He has died from a lung cancer that has been caused by the unique series of mutations, brought about by the interaction of these carcinogens together, and they cannot be separated because that series of mutations brought on by both of these carcinogens, enhancing the action of the other, cannot be separated.[117]

It was put that this was her theory, to which she replied before she was cut off that it is not her theory, it is the basis of current scientific evidence.[118] It was put that there were a lot of researchers that hold a different view, but she said that she did not think so; there are some differences in opinion in the link between cancer and amphiboles but this was a minority view.

[97] She was asked about the Helsinki Criteria to which she had referred in her first report, which was the result of a report published in 1997 for the guidance to health authorities, to which she added insurance companies, as well as other organisations, including compensation tribunals. It was conceded that it was not possible to prove in precise deterministic terms that

117 Tr p71.4

118 Tr p 72.5

lung cancer was caused by asbestos in an individual patient and the purpose of the report was to provide an alternative based on mathematical probabilities. She said that the Helsinki Report did not specifically take into account smoking as a factor in relation to lung cancer. Despite the witness' evidence that this report was out of date, and had been updated by herself and others, the cross-examiner persisted in asking questions about it that the witness regarded as irrelevant. Nevertheless, she answered, in effect stating that the report recommended a 25 fibre m/L year exposure before attributing asbestos as a cause of lung cancer, *assuming that the patient was a non-smoker*. Further cross-examination continued on the report, despite the witness saying that this was not her area of expertise, with a view to establishing that the risk of cancer from exposure to chrysotile purely was .05% and 4% from amphiboles purely.

[98] She was then referred to a paper by Hodgson and Darnton, which stated that the doubling of the risk from chrysotile exposures only occurred at a level of 1600 fibre/mL years. She agreed that this was the result of the report but said there had been updates since then.

[99] Associate Professor Klebe gave evidence that it was not possible to calculate the recommended dose at which it can be inferred that asbestos causes lung cancer. She had made no effort to do so because, in this case, that cannot be achieved. The cause of the cancer is indivisible and assessing the risk is irrelevant when the risk has materialised. The two carcinogens cannot be separately linked.

[100] Associate Professor Klebe was asked why she did not calculate the increased risk due to inhaling tobacco smoke. She replied that she did not think that it would help in this case, nonetheless she had regard to the reports of others and her own experience in similar cases that the risk was at least 25 fibre m/L years.

[101] Associate Professor Klebe was asked if she had leapt to a conclusion that both agents caused the deceased's lung cancer because she knew of no method of deciding which carcinogen caused which mutation. She responded as follows:

> That is not correct. I am referring extensively to data which shows us there's - the two at a biological level act together, in a synergistic way. I have shown in my report that asbestos, independent of smoking, or even in smokers, raises the level of oxidative stress measurable in the blood, which affects the ability of the immune system to deal with mutation. I have shown in my report, based on the literature, that asbestos fibres facilitate longer exposure of epithelium in the lung to the benzo pyrones in the cigarette smoke. The biological effects are not separable, because both of them act together. The carcinogens will bind to the fibre. So the two of them are distinctly linked and cannot be separated.[119]

[102] A point raised with Associate Professor Klebe is whether squamous cell lung cancer is exclusively associated with smoking. She said that was the opinion historically, but with current filter-tipped cigarettes, adenocarcinoma is far more common and all types of lung cancer have been associated with asbestos.[120]

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119 Tr p82.7.

120 Tr p 83.6.

[103] Associate Professor Klebe was asked about Chronic Obstructive Pulmonary Disease (part of the emphysema group of diseases), or COPD. The witness accepted that the deceased had COPD and that studies showed that amongst any group of smokers, the risk of lung cancer was multiplied 3 to 6 times in the event the person also had COPD. It is a "signature illness caused by smoking". The professor said that she did not refer to it in her report, as she did not know the severity of the deceased's COPD. She was not asked to comment on it, and many smokers in the cohort studies suffered from COPD.

[104] During her cross-examination, Associate Professor Klebe accepted that COPD was a factor that increased the risk of Mr Roussos contracting lung cancer from smoking by 3 to 6 times.

[105] It is apparent from the cross-examination of Associate Professor Klebe that, to a significant degree, the defence case relies on three key propositions, which it suggested Associate Professor Klebe ignored or failed to address adequately. First, in accordance with Mr Pickford's, and Associate Professor McKenzie's assessments, the deceased's exposure to asbestos dust over an extended period was quite insignificant. Second, a very long time elapsed from when the deceased stopped using asbestos cement and when he contracted lung cancer, and current research findings indicated that overtime the risk of acquiring lung cancer from asbestos dust reduced to zero. Third, the deceased was a very heavy smoker who suffered from COPD, which is strongly associated with lung cancer caused by smoking cigarettes.

The cross-examination of Dr Leigh

[106] Before being cross-examined, Dr Leigh was asked about the studies by Jarvholm and Astrom. He stated that the report findings were affected by bias in the study that made it difficult to reach an informed opinion, and the study was based on low exposure anyway.

[107] In cross-examination, Dr Leigh confirmed that there were three commercially viable types of asbestos, crocidolite, amosite and chrysotile. There were two classes of asbestos, amphiboles and chrysotile. Crocidolite and amosite were both amphiboles. The fibres in amphiboles are straighter and sharper than those in chrysotile. Crocidolite is very dangerous, amosite is less dangerous and chrysotile is less dangerous again. He agreed that there were various ways in which someone could become exposed to asbestos and a wide range of asbestos products. So far as asbestos cement is concerned, that was a mixture of cement, sand, asbestos and water. The asbestos component might vary anywhere from between 5% and 15% of the mix. Before 1965, some of the asbestos used included crocidolite. After 1965, some of it included amosite. The larger component was typically chrysotile. In some cases, the product contained only chrysotile. Two conditions that arise from asbestos exposure are lung cancer and mesothelioma. The latter occurs in the outer lining of the lung, not the lung itself. The deceased did not suffer from mesothelioma. Asbestosis is a non-malignant fibrotic

condition of the lung. The cross-examination continued on the assumption that the deceased did not have asbestosis.[121]

[108] Next the witness was asked a number of questions about the other conditions from which the deceased suffered and are referred to in his death certificate. The principal purpose of these questions was to connect these conditions and their cause to smoking, for example, heart disease.[122]

[109] Later, it was pointed out to Dr Leigh that the relative risk (RR) for smoking calculated by Professor McKenzie was lower than the RR he calculated, and therefore more favourable to the plaintiff. Further, the RR of 60, which Dr Leigh calculated, would increase significantly if the number of cigarettes smoked increased from 20 per day, which he had used for his calculations, to 40 a day. While he did not agree that it would increase linearly, he agreed it would increase significantly. He noted that the studies do not go over 50 pack years and he agreed that this is a "phenomenally high level of smoking".[123] He was asked if his calculations took into account COPD as a separate factor. He said they did not because COPD was an integral part of the cause of lung cancer and death by smoking and the studies did not separate them out.[124]

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121 See generally tr pp107-114.

122 Tr 114-119.

123 Tr p 121.8.

124 Tr p 125.10 to 126.1.

[110] He was cross-examined about why he chose a figure of 90 fibre/Ml years when he had earlier said, "the detail provided was insufficient to make any reliable quantitative estimate of exposure". He explained that he chose the lesser figure from Mr Kottek's report because he considered that the higher figure was extremely unlikely.[125] The rest of the cross-examination was largely directed to trying to get the witness to accept that he consistently made assumptions or chose criteria from reports that favoured the plaintiff's case as far as it was based on mathematics, which he denied.

The cross-examination of Professor Fox

[111] At the start of his cross-examination, Professor Fox was asked if he agreed with the following statement in an article by Professor Klebe and others, "Asbestos, smoking and lung cancer: an update":

> We conclude that the mechanism of lung cancer induced by the interdependent coaction of asbestos fibres and tobacco smoke at a biological level is a multistage stochastic process with both agents acting conjointly at all times."

He replied that he agreed with it in part. He was then asked what part he disagreed with. He replied, in effect, that the validity of the proposition depended on the dose of asbestos fibre, and when the asbestos exposure took place, and the dose of cigarette smoke.

[112] He was asked the following questions and gave the following answers at 147 – 148 tt:

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125 Tr p 127.5 – 128.10.

Counsel:	You do not accept ... that in every case of lung cancer that has [involved] both cigarette exposure and asbestos exposure, that the two interact biologically?
Prof Fox:	That is when there are appropriate doses and duration of both and when...
...	...
Counsel:	[T]he two carcinogens act at a biological and molecular level. Do you agree with that?
Prof Fox:	All carcinogens act at a molecular level, yes.
Counsel:	When they are both present, they both interact at a biological level, correct?
Prof Fox:	Yes and no. Without diverging, they both have quite different actions, and under some circumstances, which again depend on dose intensity and when it took place, there may be a significant inter-reaction.

[113] He was then directed to another conclusion in the article, namely that the effect of asbestos exposure and cigarette smoking is synergistic, and asked if he agreed with that proposition. Professor Fox replied, “not completely” because some experts hold that it is multiplicative, some additive and some in between.

[114] The Professor was asked the following questions and gave the following answers at 150- 151 tt:

Counsel:	[A]sbestos exposure and cigarette smoke are both carcinogens. Correct?
Prof Fox:	Yes.
Counsel:	Both capable of causing lung cancer. Correct?
Prof Fox:	Yes.
Counsel:	When they are both present, they both act synergistically. Correct?
Prof Fox:	Yes, but the degree of synergism depends, as I said before, on the dose duration, et cetera, of both factors.

Counsel: Both make a material contribution to the lung cancer tumour. That is correct is it not?

Prof Fox: No, it is not.

Counsel: Your hypothesis is this, in relation to Mr Roussos: His smoking risk is so great that it must, in all the circumstances, overwhelm the asbestos risk, correct?

Prof Fox: Correct.

Counsel: He has been exposed to both carcinogens and the biology means that both carcinogens have acted together to cause his lung cancer. That is correct, is it not?

Prof Fox: Not completely correct, because there is a proportionality of the working together.

Counsel: There is only a proportionality if you import into the biology your mathematical construct. That is correct is it not?

Prof Fox: It is not my mathematical construct, it is the relative risks.

Counsel: So his relative risk as we have discussed, is 100 percent. He has got it, correct?

Prof Fox: We are talking about two different factors contributing to the risk.

Counsel: I am talking about what caused Mr Roussos' lung cancer, and we know that he was exposed to two carcinogens, and just because one is greater than the other, it does not follow, does it, that you can dismiss either of those carcinogens as a causal factor?

Prof Fox: You can discuss the relative contribution.

Counsel: You can discuss the mathematics, but the fact is that they have both acted together and you cannot dismiss either the asbestos or the cigarette smoke as a contributor can you?

Prof Fox: You certainly cannot dismiss the cigarette smoke, but you make a discussion (sic) and consideration of what might have been the asbestos exposure adding to his lung cancer risk.

[115] He was referred to another conclusion in the article of Associate Professor Klebe and others that the synergistic effect meant that the cause of the deceased's cancer could not be apportioned between the two individual

carcinogens and asked if he agreed with this proposition. Professor Fox disagreed. He repeated his earlier answer that it depended on the dose, or level, of the exposure, and when it occurred. [126] The remainder of the cross-examination related to statistical propositions that are largely unhelpful because Professor Fox did not accept them, or at least qualified them with the same factors as he mentioned previously.

The cross-examination of Associate Professor McKenzie

[116] The cross-examination of Associate Professor McKenzie started by establishing that he did not have the deceased's statutory declaration at the time he prepared his report and calculated the risk of cancer from asbestos exposure. He said that in the course of his work, he had interviewed many carpenters from all over Australia, including one or two from the Northern Territory, and he based his assumptions about the level of exposure on his own background knowledge.

[117] He was asked to comment on the same passage from the article by Associate Professor Klebe and others quoted in paragraph [110] above. He replied that this was largely a theoretical conclusion based on a variety of studies many of which are in vitro studies and many of which are animal studies. "The bottom line is that we don't know how lung cancer is initiated."[127] He was then asked, "Do you agree with what the authors of this study concluded. He

126 Tr p145

127 Tr p191.1.

replied, “I think it’s a reasonable hypothesis.”[128] The witness later said that while it is a possibility and it is “an attractive and plausible mechanism for at least some of the cancers attributable between asbestos and cigarette smoke,”[129] ultimately, in Mr Roussos’ case, we do not know if the asbestos exposure contributed. His cancer was overwhelmingly likely to have been caused by smoking alone and it is possible that exposure to asbestos made a small contribution.

[118] He was later asked to comment about whether or not he accepted certain findings that were presumably from the same article, although this is not clear. In short, he accepted that there was evidence for synergism between asbestos and cigarette smoke in lung cancer causation at a biological level; that “any asbestos exposure, even in a heavy smoker contributes to causation”; that the data conformed best with a multiplicative model with no requirement for asbestosis.[130]

[119] He also gave the following evidence at 192 and 193tt:

> Counsel: (inaudible) so the synergistic approach that I put to you is correct, in the broad. Cigarette smoking is a carcinogen. Asbestos fibre is a carcinogen. They are both present in Mr Roussos’ history, correct?
>
> McKenzie: Correct.
>
> Counsel: Your opinion is that they both act together interdependently, correct?

128 Tr p 191.2.

129 Tr p 191.5.

130 Tr p193.4.

McKenzie: Yes.

Counsel: But in your opinion, the asbestos exposure made a *de minimus* contribution, correct?

McKenzie: Yes, correct.

Counsel: And that depends does it not, on your opinion as to what his cumulative fibre burden was?

McKenzie: Yes.

Counsel: The higher the fibre burden determined by this court, the higher the contribution from the asbestos component, correct?

McKenzie: Correct.

Counsel: But there is no argument that both are acting together in Mr Roussos' case, correct?

McKenzie: Once again, we come back to – keep asking this question, and my answer will remain the same. In Mr Roussos' case, we do not know. All right... smoking alone. It could have been asbestos alone. But, statistically, it is overwhelmingly likely that it was the smoking. And if we accept that that there is a contribution from the interaction between smoking and asbestos, then that is a small contribution.

Counsel: And you accept that there is a contribution, correct?

McKenzie: Yes.

[120] He was asked about pleural plaques and he agreed that they were a marker for asbestos exposure[131] and are not generally found as a marker for cigarette smoking.[132] He went on to explain that the cigarette smoking "impairs the ability of the lungs to remove particles or exogenous materials from the lungs, such as dust particles and asbestos fibres." It was put:

131 Tr p 193.9.

132 Tr 194.1.

"And if the smoking causes the asbestos fibres to remain in the lung for longer periods of time or prevents the lung from clearing the asbestos fibre, it stands to reason, does it not, that the lung---that the smoking has enhanced the prospect of damage to the lung caused by asbestos fibre?

He answered:

Yes.[133]

[121] Associate Professor McKenzie was asked to comment on certain passages taken from Associate Professor Klebe's first report. Without going through them all, he accepts much of her report as a reasonable hypothesis, and he accepts that if the asbestos exposure contributed to the cancer one could never apportion the individual effects of smoking and asbestos exposure.[134] There was the following exchange between Senior Counsel for the plaintiff and Associate Professor McKenzie.

Counsel: But we know that he was exposed to asbestos. We know that he was exposed to cigarette smoke. And we know that he got a lung cancer. Those – it follows, does it not, that the lung cancer was caused by the cigarette smoking and the asbestos exposure?

McKenzie: Well he may have been exposed to other things as well. But, you know, those are two things we know about, and those are two things that are potentially causal. So, yes.

Counsel: And on the history and the facts we have, the probability is that those two things caused Mr Roussos' lung cancer, correct?

McKenzie: Yes.

133 Tr 194.9.

134 Tr 197.

Ultimately, it seems that Associate Professor McKenzie accepted that the probability in this case was that "those two things [smoking and asbestos] caused Mr Roussos' lung cancer".[135]

[122] Finally, the witness was referred to the 1997 Helsinki criteria, and accepted that the criteria were alternatives: either exposure of 25 m/L years; or an occupational history of more than one year's exposure; or 5 to 10 years of moderate exposure in construction or ship building. He accepted that the deceased fitted into the last category, although he said that he did not see how the last category would be the equivalent to 25 m/L years.[136]

Consideration

[123] I am satisfied that the plaintiff has proven that the deceased's exposure to asbestos cement fibre, caused or contributed to his cancer, and therefore to his death, for the following reasons.

[124] I accept the evidence of Associate Professor Klebe as to the cause of the deceased's lung cancer, namely that at a biological level cigarette smoke and asbestos fibres acted together in a synergistic way to cause his cancer and cannot be partitioned off into the individual effects of smoking and asbestos. Asbestos fibres facilitate longer exposure of epithelium in the lung to benzo pyrones in the cigarette smoke. The biological effects are distinctly linked. In the deceased's case, there is radiological evidence of significant exposure to asbestos in the form of pleural plaques.

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135 Tr p 198.5.

136 Tr p199.7

[125] Asbestos causes chronic inflammatory changes, which release cytokines (including growth factors), providing a selective growth advantage for cells, which have undergone cancerous mutation due to carcinogens in the tobacco smoke or due to asbestos itself. Asbestos fibres facilitate longer exposure of epithelium in the lungs to the benzo pyrones in the cigarette smoke. The biological effects are not separable because both of them act together.

[126] As to the various estimates of the deceased's exposure to asbestos, Associate Professor Klebe expressed concerns about the reliability of such estimates. She stated that it was a moot point, given that in this case there is biological evidence in the form of pleural plaques showing a substantial exposure to asbestos over many years. As stated above, she stated that the real question is not whether the deceased had 19, 25 of 50 fibres/mL years exposure but whether the asbestos made a material contribution, which it did for the reasons specified in her reports. The cause of the cancer is indivisible and assessing the risk is irrelevant when the risk has materialised. The two carcinogens cannot be separately linked.

[127] The evidence before the Court establishes that there is now wide acceptance of the opinion expressed by Associate Professor Klebe. The evidence of Dr Burdon and Dr Leigh also supports the evidence of Associate Professor Klebe. Dr Leigh also gave evidence that a latency of 63 years from the deceased's first exposure to asbestos in 1956 and until his diagnosis of cancer in 2019 was not inappropriate. I accept his evidence.

[128] Associate Professor McKenzie also gave evidence that the two carcinogens act together interdependently. He further conceded that the probability was that the two carcinogens acted together to cause Mr Roussos' lung cancer. However, he maintained that asbestos made a very small contribution to the deceased's cancer.

[129] However, in Associate Professor McKenzie's opinion the contribution of asbestos fibres to the deceased's lung cancer was *de minimus*. He based his conclusion on his own assessment of the deceased's cumulative fibre burden. I do not accept his evidence in this regard. His opinion failed to take into account the deceased's evidence about how long he spent cutting and manipulating asbestos fibre. As I have stated, I have found that the deceased would have spent 40 minutes a day cutting and manipulating asbestos with power tools for most of his working life, which would have resulted in a fibre burden of more than 25 fibre/mil years.

[130] I do not accept the evidence of Professor Fox. In particular, I reject his evidence that the deceased's pleural plaques were caused by smoking. His evidence to that effect was not supported by the evidence of any other expert witness.

[131] As to the nub of the defendant's case, which is set out at [106] I find as follows. As I have stated I do not accept the evidence of Associate Professor McKenzie and Mr Pickford about the deceased's exposure to asbestos dust. I prefer the evidence of Dr Leigh about the consequences of the latency in the

development of the deceased's lung cancer. I also accept the evidence of Associate Professor Klebe about that many of the subjects in the studies on which she relied would have suffered from COPD and that condition does not exclude asbestos from making a material contribution to the deceased's cancer.

Contributory Negligence

[132] The defendant submitted that the deceased was guilty of contributory negligence for two reasons. First, because he continued to smoke cigarettes notwithstanding the warnings that were given to the public that smoking was dangerous to a person's health. Second, because the deceased failed to take adequate precautions for his own safety in his use of the defendant's products.

[133] So far as the first argument is concerned, the defendant submitted that the dominant cause of the deceased's cancer was his smoking; that the deceased took up smoking at a time when it was already known that smoking was dangerous, continued to smoke for nearly 40 years after the warnings first appeared on cigarette packets, and continued long after graphic warnings were given a public airing. Counsel for the defendant did not refer to any evidence led at the trial in support of these contentions. The evidence that was led to show what warnings were given, when they were given, by whom and to whom is contained in Exhibits D3 and D7. One of the problems with this evidence is that there is no evidence that the articles in Ext D3 were published in the Northern Territory. While the articles in Ext D7 may have

been published in the Northern Territory, counsel for the plaintiff submitted that they did not adequately communicate the risk to the smoking population and in some cases appeared to have celebrated smoking. For example, an editorial in the NT News of 7 January 1981 did nothing to deter smokers by stating that "smokers have every right to puff away to their lungs content."

[134] Further, from 1972 onwards a number of Acts came into force in the Northern Territory that required certain warnings to be on cigarette packets. On 5 December 1972 the *Cigarette Containers (Labelling) Ordinance 1972* (NT) was assented to. The Ordinance came into operation on 1 January 1973. Subsection 4(1) of the Ordinance states:

> A person shall not sell, or expose for sale, cigarettes otherwise than in a container on which the statement "WARNING – SMOKING IS A HEALTH HAZARD" appears, as provided by this section, in two positions on the exterior of the container.

[135] In 1992 the *Tobacco Act 1992* (NT) came into operation. Section 6 of the Act and Regulation 4(1) and (2) of the Tobacco Regulations required cigarette packets to have on them the warning, SMOKING CAUSES LUNG CANCER.

[136] In 1992, the Commonwealth Government enacted the *Tobacco Advertising Prohibition Act* (Cth) which operated in conjunction with the Northern Territory Legislation to control tobacco advertising

[137] In 2002, the *Tobacco Control Act 2002* (NT) repealed the *Tobacco Act 1992* and provided a much more comprehensive regime warning of the very serious dangers of cigarette smoking.

[138] Relevant to an assessment of the deceased's understanding of the risks of cigarette smoking, are the matters referred to in the two reports of Dr Wayne Hall, a psychologist and an expert in the field of drug addiction, including tobacco addiction, whose credentials are not in doubt. For various reasons, Dr Hall is of the opinion that it is more probable than not that the deceased was not aware, or not fully aware, of the risks of cancer from cigarette smoking. Dr Hall was also of the opinion that the deceased was less informed about those risks and, because of his addiction, less likely to be able to succeed in quitting smoking. His opinion was influenced by the following factors. The deceased had a limited education, and a limited ability to speak and read English. He arrived in Australia at aged 18 speaking only Greek, and mixed primarily with Greek migrants in Darwin. He worked as a carpenter and mixed with other carpenters, most of whom were probably smokers, and he did not work in a non-smoking environment. The Northern Territory funded fewer anti-smoking campaigns than other jurisdictions. The Northern Territory's population was younger than other jurisdictions and smoking was more prevalent than elsewhere in Australia. Warning labels on cigarette packets such as "smoking causes lung cancer and heart disease" did not appear in Australia until 1987. As far as newspaper articles were concerned, only three articles appeared in the

Northern Territory prior to 2000, two of these were in the Centralian Advocate and the other in the NT News was only 48 words long, albeit it was on page 1. The chance of the deceased being properly appreciative of the risks of smoking depends more on the social environment in which he lived and worked, than on the occasional newspaper article he may or may not have read. As far as TV ads are concerned, the first national program was not until 1990s.

[139] As against this, I note that the deceased signed his affidavit before his death without it being translated, which suggests that perhaps he could read English better than expected. In addition, I note that when he had his operation in 2010, he was able to give up smoking. There is nothing to suggest that this then caused him any particular difficulty.

[140] The defendant referred to the decision of the Court of Appeal of Western Australia in *The State of South Australia v Ellis*[137] where the majority of the Court allowed an appeal from a finding that the plaintiff's contributory negligence was 10%, and increased it to 50%. The facts of that case are different from the present case but the principles applied by the court are important. Steytler P and McLure JA stated:[138]

> A plaintiff will be guilty of contributory negligence when the plaintiff exposes himself or herself to a risk of injury which might reasonably have been foreseen and avoided and suffers an injury within the class of risk to which the plaintiff was exposed: *Joselyn* (McHugh J). Once

137 [2008] WASCA 200; (2008) 37 WAR 1.

138 At [491]-[492].

contributory negligence is proved, a just and equitable proportion must be made.

The relevant principles are addressed in *Podrebersek v Australian Iron and Steel Pty Ltd* [1995] HCA 34; (1985) 59 ALJR 492, 493-494. The court (Gibbs CJ, Mason, Wilson, Brennan and Deane JJ) said that the making of an apportionment involves a comparison both of 'culpability, ie the degree of departure from the standard of care of the reasonable man…and of the relative importance of the acts of the parties in causing the damage…' The court also said that it was 'the whole conduct of each negligent party in relation to the circumstances' of the damage 'that must be subjected to comparative examination' (see also *Wynbergen v Hoyts Corporation Pty Ltd* [1997] HCA 52; 72 ALJR 65, 68 (Hayne J, with whom Gaudron, McHugh Gummow and Kirby JJ agreed)). It is, of course, important to bear in mind that a finding on a question of apportionment is one upon a 'question not of principle or positive findings of fact or law, but of proportion, of balance, and of relative emphasis, and of weighing different considerations… [involving] an individual choice or discretion as to which there may well be differences of opinion by different minds': *Owners of Steamship or Vessel 'British Fame' v Owners of Steamship or Vessel 'McGregor'* (the '*British Fame'*) [1943]AC 197, 201 quoted in *Podrebersek* (532).

[141] The second matter put by way of contributory negligence raises two questions. The first question is whether there is any evidence that the deceased knew the defendant's products were dangerous. According to the deceased's statement, he first became aware that asbestos fibres were dangerous at some time in the last 15 or 20 years, i.e. between 1999 and 2004. There is no evidence other than the warning labels attached to the defendant's products that might have alerted the deceased to the danger. As noted previously, these labels made no appearance until the later 1970s, and the warning about the danger of getting cancer was not provided until 1982. No specific warning was given about the dangers to smokers, yet these dangers were well known to the defendant, from at least 25 January 1968. I

have previously found that the warnings given were inadequate. I do not make any finding against the deceased based on the warnings.

[142] The second and more contentious submission is that the deceased should have complied with the *Construction Safety Rules 1978* (NT) that came into force in February 1979, and that if he had complied, it would have resulted in a substantial reduction in his exposure to asbestos cement dust.[139] The requirement to comply with the rules existed because he was as one of the partners in Roussos Brothers, an employer of other men, who were similarly exposed. This partnership lasted until 1983. Thus, the period during which it might be argued that the deceased should have complied with the rules by using masks and protective clothing etc. is limited to the period between February 1979 and the mid-1980s when the defendant removed its products from the market.

[143] Division 3 of Part IV of the *Construction Safety Rules 1978*, (NT) deals with rules for spraying and handling asbestos. Among other things, rule 48(2)(a) stipulates that:

> A person engaged in asbestos process on a construction site shall wear and make full and proper use of protective clothing and protective respiratory equipment provided for his use.

[144] There is no evidence before the Court about whether the *Construction Safety Rules 1979* were well publicised or enforced in the Northern Territory or

139 See the evidence of Mr Kottek at tr pp216-217.

that the deceased had any knowledge of them. In any event, by 1979 the deceased had been exposed to asbestos cement dust for about 23 years.

Consideration

[145] I am not satisfied that the deceased is guilty of contributory negligence for failing to take adequate steps in the protection of his own safety in the use of the defendant's products. Nor am I satisfied that the deceased was guilty of contributory negligence for failing to comply with the *Construction Safety Rules 1978*. I have found that the warnings provided by the defendant were inadequate and I also find that it has not been established that the *Construction Safety Rules 1978* were adequately brought or came to the deceased's attention. The rules extensively cover areas other than working with asbestos.

[146] However, I am satisfied that the defendant has proved that the deceased is guilty of contributory negligence for continuing to smoke cigarettes in the face of warnings that it was dangerous to do so. I apportion the deceased's contributory negligence as 50 percent.

[147] The deceased must have been aware that smoking was a health hazard by 1972 when warnings first appeared on cigarette packets in the Northern Territory. Those warnings became stronger as the years progressed. By 1987 cigarette packets in Australia carried the warning that smoking causes lung cancer and heart disease. In 1992 s 6 of the *Tobacco Act 1992* (NT) and Regulation 4(1) and (2) of the Tobacco Regulations came into operation.

The Regulations required cigarette packets to have on them the warning, SMOKING CAUSES LUNG CANCER. Despite these warnings, and the additional warnings which were introduced in 2002, the deceased continued to smoke until 2010.

Damages generally

[148] Damages in this proceeding are not subject to the *Personal Injuries (Liabilities and Damages) Act 2003*. Damages are at large and the common law applies. However, any damages assessed by the Court must be reduced by the deceased's contributory negligence. Consequently, I have first assessed the amount of damages under the various heads of damages and then adjusted the amount of damages assessed to take into account his contributory negligence.

Damages for pain and suffering

[149] Prima facie, the plaintiff is entitled to damages under ss 5 and 6 of the *Law Reform (Miscellaneous Provisions*) Act *1956* (NT). If the claim falls within the exceptions referred to in s 6(2) of the Act, the plaintiff can recover damages for the deceased's pain and suffering, bodily or mental harm, and for curtailment of expectation of life, but not for loss of future earnings nor for loss of future earning capacity. In order to do so, the action must be for damages for personal injury "arising from a dust disease" which results in the deceased's death before the action is determined. A dust disease is defined by s 6(4) to "mean a pathological condition of the lungs, pleura or peritoneum that is attributable to dust." The defendant maintains that the

claim must fail because the deceased did not die as a result of lung cancer, and therefore did not fall within the definition of "dust disease".

[150] Counsel for the defendant submitted that, although lung cancer was present as one of the antecedent conditions leading to the deceased's death, his death occurred because of a "community acquired" infection, i.e. a bacterial infection, and each of the conditions of liver failure and kidney failure "were probably caused by the heart or other cardiac conditions".

[151] Counsel for the plaintiff submitted that the principal cause of death was lung cancer. In his report of 20 February 2021,[140] Dr Burdon gave evidence to this effect. Dr Burdon was cross-examined on this issue but did not waiver in his opinion.[141] Professor Fox, who gave evidence for the defendant, said that his death was "largely due to pneumonia, a sequel of either progressive cardiac failure or progressive tumour."[142] Professor Fox was not cross-examined on this issue. In his report, Dr McComish stated, "from the Death Certificate it looks likely that he succumbed to the complications of chest infection in his debilitated state. So, from the information I have it would appear that lung cancer contributed to his death but was not the isolated cause."[143] However, Dr McComish did not receive any of the hospital records after the

140 Ext P1 Item 2, p 8.

141 Tr pp 166-167.

142 Report, Ext D1 Item 2, p8.

143 Report, Ext D1, Item 3, p4.

deceased's admission on 4 July 2019, which limited his ability to comment.[144]

[152] The expression "arising from a dust disease" is wider than "caused by a dust disease", cf. the expression "caused by or arising out of his employment" in worker's compensation legislation. In my opinion, it is sufficient if a dust disease was a contributing cause of his death. In my opinion, the major cause of his death can be directly attributed to his lung cancer. If I am wrong in that, his death arose from lung cancer which in turn arose from a dust disease.

[153] Alternatively, the defendant submitted that, in this case, any award of damages for pain and suffering should be quite small, maybe $30,000, because there was no evidence of what the symptoms of his lung cancer were until it became evident in May 2019 that he had lung cancer. Counsel for the plaintiff maintained that the deceased's pain and suffering "was extreme, torrid and unrelenting over in excess of 5 years". In my opinion, the evidence supports neither contention completely, although the evidence supports a finding that the deceased did have a particularly nasty experience especially in the last 6 months of his life.

[154] According to the evidence of the deceased's daughter, Helen Roussos, prior to 2016, the deceased was well and independent in all of his activities of daily living, he was happy and enjoyed a good quality of life. Between 2016

144 Ibid, pp1,2.

and 2017, she observed that the deceased became increasingly fatigued and short of breath, and suffered from spells of dizziness. Despite this, he remained relatively independent. Towards the end of 2017, she and her siblings began to provide assistance around the house. This included looking after the deceased's wife, Irini, who suffered from a major depressive disorder, type II diabetes, and is frail and prone to falls. Up until then, the deceased had been Irini's carer and did most of the work around the house. A roster was set up between Helen Roussos and her sisters Koula, Maria and Anna who assisted with grocery shopping, meal preparation, clothes and linen washing, sweeping the home, doing welfare checks and transporting the deceased to and from hospital and other community appointments. Her best estimate is that she and her siblings provided on average 10 hours of care per week during this period. Throughout 2019, the deceased's health deteriorated. He became increasingly frail and short of breath. He frequently vomited. In the final six months, he required very high levels of care and assistance. The sisters drew up a new roster to ensure that he and Irini received the help and care that they needed. According to Helen, they averaged about 4 hours per day assissting the deceased during this period until his final hospitalisation. The help and assistance included, in addition to what he had been provided previously, managing his medication, changing surgical dressings, feeding the deceased towards the end, supervising the deceased in the bathroom and assisting him with washing and showering, managing his financial and legal affairs and assisting him to

pay his bills at the post office, and frequent cleaning of bathrooms due to vomiting. In cross-examination, she confirmed that the deceased complained of dizziness quite often when climbing stairs and that towards the end this became a constant theme and quite disabling. She confirmed that, in 2018, the deceased was still able to drive a car to go to the shops or to the bank, and he could make a sandwich for himself and prepare breakfast of Weet-Bix. However, he was not able to cook up a healthy meal. She said that he attempted to mow the lawn on one occasion that she knew of in 2018 when he was unable to get help. She said that the deceased continued to drive until he lost his licence at some time in 2019. She also confirmed that she, her siblings, and the deceased assisted Irini with household chores for many years prior to the deceased's death.

[155] One of the difficulties in this case is deciding whether the deceased's lung cancer was a contributing cause of his shortness of breath and dizziness. The evidence is that the deceased was also suffering from Chronic Obstructive Pulmonary Disease (COPD), the principal cause of which is smoking, and which would account for his symptoms. Although, in June 2019, Dr Hewitt described his vomiting as likely to be a result of recently commencing radiotherapy for his cancer. In the circumstances, it is difficult to attribute much of the deceased's need for assistance, at least until 2018 to 2019, to his cancer.

[156] However, in 2019 the deceased consulted various specialists, including respiratory, oncology and radiation oncology, attended outpatient clinics,

underwent numerous invasive surgical procedures and investigations including a lung biopsy, had multiple admissions to hospital including to the ICU, underwent several blood transfusions and 5 courses of radiation therapy and immunotherapy with consequent serious and painful side-effects. He spent his last days at the Royal Darwin Hospital with immediate and extended family at his bedside and was on IV pain medication.

[157] Counsel for the plaintiff referred the Court to a number of decisions where significant damages awards were made for pain and suffering in dust disease cases.[145] All are from other jurisdictions. It seems that there are no similar cases from this jurisdiction. I accept that those decisions are of some assistance. Doing the best I can, I determine that, but for his contributory negligence, the deceased would have been entitled to $300,000 for pain and suffering. As a result of his contributory negligence the amount of damages for pain and suffering is reduced to $150,000.

Damages for loss of expectation of life

[158] The plaintiff claimed $8,000 for loss of expectation of life, based on the submission that the Dust Diseases Tribunal conventionally awards $1,000 for each year of life lost. Whatever else might be said about this sum, it is certainly not a large amount of money. I have no doubt that if one were to offer $1000 for a year's life, the offer would be universally rejected. It was

145 *Kennedy v CIMIC Group Limited and CPB Contractors Pty Ltd* [2020] NSWDDT 7; *Webber v Comcare* [2018] NSWDDT 10; *Dunning v BHP Billiton Limited* [2014] NSWDDT 3; *Amaca Pty Limited v Tullipan* [2014] NSWCA 269; *Williams v Amaca Pty Limited* [2020] NSWDDT 2; *Parkin v Amaca Pty Limited* [2020] WASC 306.

submitted that the plaintiff's life expectancy was the same as an 81 year old male and in accordance with life tables, he had a life expectancy of 8.68 years at the time of his death. I think that overlooks the other serious conditions from which he was suffering. The evidence suggests that but for the cancer, the deceased's future life expectancy was between 5 and 10 years. In accordance with *Watts v Rake* and *Purkess v Crittenden*, which are discussed below, I am unable to find that his other conditions would have ended his life less than 8 years from the time of his death. But for the finding of contributory negligence, I would have awarded the full amount of $8,000 claimed. As a result of the deceased contributory negligence, the amount is reduced to $4,000.

Funeral Expenses

[159] Funeral expenses may be claimed in accordance with s 6(1)(c)(i) of the *Law Reform (Miscellaneous Provisions) Act 1956* (NT). The amount claimed is $17,042.40 being the amounts paid for funeral services, the burial plot and to the Litchfield Council for internment fees.[146] The defendant denies liability for the same reasons previously discussed, but says that in any event it should not be for the full amount claimed given that there were other causes as well. No authority was cited for the defendant's proposition. The deceased's lung cancer was the principal cause of his death. But it is clear that there were other causes as well which were not the fault of the defendant. The High Court dealt with this type of problem first in *Watts v*

146 P5 pp3-7.

Rake[147]. In that case, the plaintiff suffered personal injury as a result of the defendant's negligence. The injury exacerbated a pre-existing condition which it was found would have led to the same disability anyway after a period of 13 years. The Court held that, so far as the original injury was concerned, the defendant was fully responsible because he must take the victim as he finds him. However, once the period after the 13 years had passed, the defendant was no longer liable on the basis that the vicissitudes of life, which the court is bound to take into account once they have become a reality, brings to an end the defendant's liability. This is because the disability can no longer be attributed to the accident. Leaving aside any question of the burden of proof, in the present case it is clear that there were other causes not related to the lung cancer and that these other causes were likely to have caused, by themselves, the deceased's premature death as some future period of time likely to be in the order of 5-10 years.[148] In those circumstances, I consider that the evidence does not permit me to make a finding that the deceased would have died from his other health problems so close to his actual death that any allowance should be made for that possibility. Accordingly, there will be an award for $17,042.40 for funeral expenses reduced by 50 percent to $8,521.20 for the deceased's contributory negligence.

Medical Expenses

147 [1960] HCA 58; 108 CLR 158; see also *Purkess v Crittenden* [1965] HCA 34; 114 CLR 164.

148 Dr McComish, report Ext. D1 item 3 p5.

[160] The plaintiff claims out of pocket expenses in the sum of $94,727.40 for statutory refunds to Medicare Australia pursuant to the *Health and Other Services (Compensation) Act 1955* (Cth), for the Royal Darwin Hospital pursuant to the *Medical Services Act 1982* (NT), and miscellaneous costs. The amounts claimed are $88,227.40 for the RDH; $1500 for Medicare, and $5000 for "miscellaneous costs." The amount claimed by the RDH is set out in detail in Ext P5, p2. The defendant points out that it is described as a "cost estimate" which does not appear to have been paid, and that it might be negotiable. These factors do not detract from the probability that prima facie that is the amount which the estate will be required to reimburse the RDH. However, the defendant submits that the costs do not all appear to be related to the deceased's lung cancer. The first claim relates to an admission on 20 June 2019. The RDH notes[149] indicate that this admission was for "an upper GI bleed".[150] However, the RDH notes also indicate that he had been receiving radiotherapy treatment for his cancer only 2 days before and that the radiotherapy had been put on hold on 21 June 2019, with a review on the following Monday.[151] The evidence does not support a finding that the upper GI bleed was causally related to the cancer or his treatment for cancer. I would therefore deduct $12,980 from this claim. The only other complaint relates to an amount of $7,788 for an admission on 10 October 2019 that it is suggested related to his admission for community-acquired pneumonia.

149 Ext D4 p197.

150 This refers to gastrointestinal bleeding.

151 See generally Ext D5 pps61-72.

However that may be, it is evident that the pneumonia is also causally related to his cancer. There is no evidence as to what the miscellaneous amount of $5000 is for and I do not allow anything in respect of that amount. I would therefore allow the sum of $70,247.40 under this head that is to be reduced by 50 percent for the deceased's contributory negligence, resulting in an amount of $35,123.70.

Griffiths v Kerkemeyer damages

[161] The plaintiff claims $82,626.85 for *Griffith v Kerkemeyer* damages. This comprises a claim of $41,040.00 for the period from 1 January 2018 to 26 May 2019; and $41,586.85 for the period from 26 May 2019 to 26 November 2019.

[162] The relevant rate for assessing *Griffith v Kerkemeyer* damages is the gross standard or market rate that would be paid to secure the services from outside help of the relevant kind, not the net (after-tax) rate.[152]

[163] The defendant submitted that the plaintiff's claim should be rejected in its entirety because the evidence was that he would have needed the care in any event. Dr McComish was asked to advise in his report if, besides his lung cancer, the deceased would require care and assistance as the condition progressed. He stated that the answer was yes, and he would need assistance with the bathroom and toilet each morning and with meals, probably 2 to 3 hours per day. However, it is not clear when Dr McComish was saying that

152 *Griffith v Kerkemeyer* (1977) 139 CLR 161 at 193; *GIO (NSW) v Planas* [1984] 2 NSWLR 671; *CSR Ltd v Bowditch* (1991) 7 NSWCCR 223.

the assistance the deceased needed would have commenced. The fact that Dr Burdon agreed with these comments does not advance the defendant's submission. Given that Dr McComish also stated that the deceased had a life expectancy of 5 to 10 years from his other conditions, he may have been thinking in terms of an average over this latter period. Furthermore, this assistance of 2 hours a day is a lot less than the assistance of 4 hours a day which is claimed as having been provided, and which in my opinion appears to have been eminently reasonable.

[164] It was put that, on the evidence, the care that was provided was occasioned by Mr Roussos' dizziness and shortness of breath and feelings of tiredness. It was put that this was all caused by the myriad of cardiac conditions from which the deceased suffered. There is no evidence of this. The evidence of Dr Burdon is that he suffered from squamous lung carcinoma, and that this is a consequence of his exposure to asbestos because of the presence of the pleural plaques and associated calcification.[153] This form of lung cancer can bring about dizziness, shortness of breath and tiredness.

[165] The total claim is for the period between 1 January 2018 and 26 November 2019, excluding any periods of time when the deceased was in hospital. As stated above the claim is for two periods. The first period is from 1 January 2018 to 26 May 2019, six months before his death. The amount claimed for this period of 72 weeks is for $41,040.00 and is based on an average of 10

153 Report Ext P1 page 13, p 7 of the report.

hours of care and assistance per week according to the evidence of Helen Roussos. The rate claimed is $57 per hour, based on the day rates provided by the report of Therapy Solutions[154] for a support worker. I note that rates for evening work and weekend work and public holidays are higher. I would allow a claim for the period from 20 September 2018 after he was discharged from the Royal Darwin Hospital to 26 May 2019. Prior to 20 September 2018 the deceased's needs were primarily due to his heart condition. However, on 20 September 2018 Mr Roussos was admitted to the Royal Darwin Hospital with shortness of breath. Professor Kenneth Thomson, a radiologist, viewed an x-ray of the deceased's chest of 20 September 2018 and found there to be a well-defined 4 cm in diameter mass in the left upper lobe that he considered indicated a malignant tumour. I find that from the time onwards the deceased's lung cancer created the needs that are the subject of the *Griffith v Kerkemyer* claim. I assess the damages for the period from the deceased's release from the Royal Darwin Hospital in September 2018 to 26 May 2019, doing the best I can, to be $19,314.00 less 50 percent for contributory negligence which comes to $9,656.47.

[166] The amount claimed in respect of the period from 26 May 2019 is based on the rates identified by Therapy Solutions. For this period, higher rates are claimed for care provided over weekends and one hour per weekday is claimed at the evening rate, but otherwise the day rate is applied. The total claim amounts to $41,587.85 for this period. I hold that the plaintiff is

154 Ext P1 Item 15.

entitled to the payment of this amount, reduced by 50 percent for contributory negligence, which comes to $20,793.93.

[167] The defendant submits that the rates are only claimable at the post tax rate payable to a comparable person and not at commercial rates, citing *Amaca Pty Ltd v Werfel,*[155] a decision of the Full Court of the Supreme Court of South Australia. In that case, the Court held that, given that the services were unskilled and that the plaintiff had the capacity to retain carers, a rate of $20 an hour was appropriate until "after remission and the recurrence of illness until the end of life, when professional care at agency rates was appropriate." The Court referred to the well-known decision of the High Court in *Van Gervan v Fenton* [156], where it was held that the normal measure of the loss is determined by reference to commercial rates, because the loss is calculated on the basis of the plaintiff's (or deceased person's) need for the services, and not on some other basis. Some exceptions to this were recognized in the majority judgment of Mason CJ, Toohey and McHugh JJ[157], where for example, the market cost may be too high because of the remote location of the services which were being provided by the wife or husband of the plaintiff. Such exceptions are not applicable to this matter. Furthermore, no allowance is to be made for the fact that some of the services were provided to the deceased as a matter or ordinary domestic relations before the accident:

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155 [2020] SASCFC 125 at [517]-[518].

156 [1992] HCA 54; 175 CLR 327 at 337-338.

157 Ibid, at para [17].

> "A defendant is no more entitled to have the pre-accident voluntary contribution of a spouse taken into account than a defendant would be entitled to have the pre-accident value of a paid housekeeper taken into account."[158]

[168] The approach in *Amaca v Werfel* is not entirely consistent with the decision of the majority in *Van Gervan*, and some of the observations of the Court would require the parties to enter into meticulous evidence gathering and record-keeping. This is not consistent with the nature of the claim, which is to compensate the plaintiff, or the deceased's estate, for the need for the services. In the present case, there is no basis for finding that the amount for the services should be otherwise than on a commercial rate. No evidence was led to suggest that the rates claimed were too high or could be obtained more cheaply elsewhere, for example by the deceased's family engaging someone themselves and thereby avoiding the supplier's profit margin. In any event, such an approach is fraught with unnecessary difficulties as the family would then have to deal with such other issues as income tax, GST and superannuation, a nightmare for an ordinary family already doing its best in difficult circumstances.

[169] Accordingly, I award the plaintiff $30,450.40 under this head of damages.

Damages for wrongful death

[170] Section 10 of the *Compensation (Fatal Injuries) Act 1974* (NT) permits the Court to make an award for (a) loss of consortium to the deceased's spouse, (b) the reasonable costs of engaging services to provide for household

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158 Ibid, at para [26]; cf Brennan J; and Deane and Dawson JJ who held a different opinion on this point. However Gaudron J agreed with the majority.

services previously performed by the deceased, and (c) solatium for the deceased's widow and for each of the deceased's children.

[171] The amount claimed for loss of consortium for Mrs Irini Roussos is $20,000. This is a modest sum which reflects the fact that she could not give evidence because of her own ill-health. As Helen Roussos noted in her evidence, Irini was unable to do domestic work by the beginning of 2018 and some of the assistance provided was also provided to Irini. According to her evidence, her sister Maria mainly cared for her mother. Prior to 2018, the deceased supported his wife by working as a handyman and he did most of the work around the house, including the housework because Irini was unwell as she suffered from a major depressive disorder and type II diabetes, and was prone to falls. However, the children also helped with domestic chores, cleaning, washing, vacuuming, sweeping and doing the laundry. In cross-examination, Ms Roussos said:

> My mum would do what she could do and she's a very proud lady who would always try. I don't want to paint the picture that my mother was completely useless because she wasn't and she isn't. But truth be said that a lot would fall on the family, especially the children and *my father had to pick up where- to pick up what she couldn't do*. So, quite often, part of her condition, she wouldn't be able to finish tasks like laundry. She wouldn't be able to go to the shop to buy her food or to do shopping. So, a lot of the responsibility fell on the children in Darwin.

[172] Altogether, there were seven children, four of whom lived in Darwin and who were involved in supporting the deceased or Irini. These were Helen, Koula, Maria and Anna. In addition, Michael lived in Milperra, and was a qualified builder. He gave evidence but only about the deceased's smoking

habit. There was another son, Manolis or Manoli, who lived in New South Wales. He is a medical practitioner who was consulted about whether or not the deceased should undergo surgery or radiotherapy, which suggests that he was in contact with the deceased and other family members.[159] There is evidence that Manoli was present in Darwin and attended the RDH on 24 May 2019 with the deceased, Helen, and Koulla when the deceased was interviewed about the options for his future care.[160] On the day of the deceased's death, the RDH notes show that there were 4 family members present and another one expected to arrive shortly. One of the family members was Irini who the notes record 'was sitting on a chair in the corridor" and according to the family was "having a major panic attack."[161] There is also a note that on 16 November 2019, at the deceased's request, a doctor spoke with his son Manolis over the phone about his care plan.[162] Although the hospital notes refer to the family being present on a number of occasions, apart from Helen and Irini most of the other notations indicate that, while there were family present at various times during the last period of his hospitalisation, no other names are mentioned, except I note that on at least two occasions a son is also mentioned but not named. None of the other family members gave evidence. Who the seventh child is remains a mystery.

159 Ext P5 p190 file note by Dr Charakidis dated 7 June 2019.

160 Ext P 5 p50 Letter by Dr Charakidis dated 24 May 2019.

161 Ext P 5 pp 109-110; 190.

162 Ext p5 p135.

[173] So far as the claim for loss of consortium is concerned, it would appear from the limited information available that the deceased and his wife Irini were a close couple, as is evidenced by Irini's concern to be present at the hospital on a number of occasions and the evidence that the deceased assisted her with domestic chores. I find that the amount claimed of $20,000 is reasonable and 50 percent of that amount, being $10,000, is awarded for the benefit of Irini Roussos.

[174] So far as the claim for the hire of help to perform household services is concerned, I make no allowance for the period up to the deceased's death. This is because no deduction was made for the fact that the *Griffith v Kerkemeyer* damages were not reduced, in recognition that the assistance given not only benefited the deceased but also benefited Irini and an award has been made in respect of that assistance. However, an amount should be awarded on the basis that, but for the cancer, the deceased would have lived another 5 to 10 years during which he would have, at least for some of that time, performed household services for the benefit of Irini in accordance with his customary behaviour. Undoubtedly at some time the deceased himself would have needed support had he lived, perhaps for as much as 2 hours per day as Dr McComish suggests. In the circumstances the amount claimed of $50,000 is excessive. I would award an amount of $10,000 which must be reduced by 50 percent to $5000.

[175] So far as the claims for solatium are concerned, although the evidence is largely circumstantial, I find that the family were close and were very

saddened by the deceased's death. As I know nothing about the seventh child there will be no award for that person. I determine that the members of the deceased's family are entitled to awards of the following amounts for this head of damages, which are to be reduced by 50 percent because of the deceased's contributory negligence:

Irini: $10,000 reduced to $ 5,000

Helen: $10,000 reduced to $ 5,000

Koula: $10,000 reduced to $ 5,000

Anna: $10,000 reduced to $ 5,000

Maria: $10,000 reduced to $ 5,000

Michael: $10,000 reduced to $ 5,000

Manolis: $10,000 reduced to $ 5,000

Total Damages

[176] The total award of damages is $278,095.30.

Interest on past damages

[177] Since the judgement of Gibbs J (as he was then) in *Cullen v Trappell*[163] (with whom the majority of the Court agreed), interest on damages, including damages for pain and suffering up to the date of trial, and other losses incurred up to the date of trial, have been able to be awarded at commercial rates, to the extent that this is justified by legislation. In this jurisdiction the relevant provision is s 84 of the *Supreme Court Act 1979.* I note that s 84(1A) states that the provision is subject to part 4 of the

163 [1980] HCA 10; 146 CLR 1.

Personal Injuries (Liabilities and Damages) Act 2003. Section 29 of the latter Act precludes the court from awarding interest on damages awarded for non-pecuniary loss or gratuitous services. However, s 4(3)(c) exempts a claim for damages for personal injury that is a dust related condition. This includes a fatal injury (s 3 definition of "personal injury") and a "dust - related condition" includes "asbestos induced carcinoma and asbestos related pleural disease." (s 3).

[178] As far as I have been able to ascertain, there is no rule of court, practice direction or other statutory provision which fixes the rate of interest. Commercial rates of interest have varied considerably over the last few years and much depends on whether the rate is to be applied to a secured or unsecured loan. In the absence of any evidence as to what is an appropriate rate, I will hear the parties further as to the amount of interest to be awarded and costs.

ADDENDUM

Delivered: 13 April 2024

Interest

[1] On 28 March 2024, damages were assessed for the plaintiff with interest and costs to be determined. The approach taken in this regard is similar to the approach taken by Mildren AJ in *TTG Nominees Pty Ltd v Aileron Pastoral*

Holding Pty Ltd[164] where his Honour delivered judgment on 31 January 2020 and his decision on interest and costs on 15 April 2020.

[2] I awarded the plaintiff damages in the sum of $278,095.30 comprised of the following amounts:

HEAD OF DAMAGES	**AMOUNT**
Damages for pain and suffering	$150,000
Damages for loss of expectation of life	$4,000
Funeral expenses	$8,521.20
Medical expenses	$35,123.70
Griffiths v Kerkemeyer damages	$30,450.40
Damages for wrongful death	• $10,000 (loss of consortium) • $5,000 (costs of engaging household services) • $35,000 (solatium, being $5,000 x 7)

164 [2020] NTSC 15 at [2].

[3] On 2 April 2024, I made directions about the filing of submissions on interest and costs. On 10 April 2024, the plaintiff filed her submissions on interest and costs. On 19 April 2024, the defendant filed its submissions on interest and costs. On 24 April 2024, the plaintiff filed her submissions in reply.

[4] Following are my reasons for decision on interest and costs.

Plaintiff's submissions on costs and interest

[5] The plaintiff claims interest under s 84(1) of the *Supreme Court Act 1979* (NT) on the damages awarded by the Court under various heads of damages at a rate of 10.35 percent. Subsection 84(1) states:

> In any proceeding in respect of a cause of action that arises after the commencement of this Act the Court may order that there shall be included in the sum for which judgment is given interest at such rate as it thinks fit on the whole or any part of the sum for the whole of any part of the period between the date when the cause of action arose and the date of the judgment.

[6] A claim for damages for personal injury that is a dust-related condition is excluded from the application of the *Personal Injuries (Liabilities and Damages) Act 2003* (NT), other than Part 4, Division 5 that deals with orders for structured settlements. As such, Part 4, Division 5 that deals with interest of the *Personal Injuries (Liabilities and Damages) Act 2003* (NT) has no application to this action for damages. Consequently, it was submitted that interest may be awarded on damages for non-pecuniary (non-economic) loss and gratuitous services.

[7] In support of the plaintiff's submission on interest the plaintiff relied on *TTG Nominees Pty Ltd v Aileron Pastoral Holding Pty Ltd*.[165] In that case, Mildren AJ stated:

> The Court has a discretion to allow interest at ordinary commercial rates. It was open to the plaintiff to call evidence about relevant commercial interest rates, but it has elected not to do so. In the absence of such evidence, the practice is to apply the rates from time to time applicable to post judgment interest. Under Supreme Court Rule 59.02(3), the rate of interest fixed for post judgment interest is the rate of interest fixed for s 52(2)(a) of the *Federal Court of Australia Act 1976* (Cth).

[8] However, *TTG Nominees Pty Ltd v Aileron Pastoral Holding Pty Ltd* is not a personal injuries case. It concerned unpaid rent, and breach and repudiation of contract.

[9] As was the case in *TTG Nominees Pty Ltd v Aileron Pastoral Holding Pty Ltd*, the plaintiff in this proceeding did not call evidence about interest rates. Instead the plaintiff submitted that the Court should adopt the usual practice and order that the rate at which interest is to be paid on the amounts awarded in the judgment be the rates applicable from time to time for post judgment.

[10] Rule 39.06 of the *Federal Court Rules 2011* (Cth) states:

> The prescribed rate at which interest is payable under section 52(2)(a) of the Act is:

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165 [2020] NTSC 15 at [2].

(a) for the period from 1 January to 30 June in any year – the rate that is 6% above the cash rate last published by the Reserve Bank of Australia before the period commenced; and

(b) for the period 1 July to 31 December in any year – the rate that is 6 percent above the cash rate last published by the Reserve bank of Australia before the period commenced.

[11] On 6 December 2023, the Reserve Bank of Australia cash rate was 4.35 percent. Consequently, the plaintiff submitted that the rate at which interest is fixed by the *Federal Court Rules* and may be payable on damages in this proceeding is 10.35 percent.

[12] If interest was awarded at the above rate, the plaintiff calculated that she was entitled to a total award of interest of $128,119.24 calculated as follows:

Type of damages	**Period claimed for**	**Rate applied**	**Calculation and sum**
General damages	20 September 2018—28 March 2024	10.35%	$150,000 x 0.1035 x (2017 days ÷ 365) = **$85,791.58**
Loss of expectation of life	26 November—28 March 2024	10.35%	$4,000 x 0.1035 x (1584 days ÷ 365) = **$1,796.65**
Past gratuitous care	20 September 2018—26 November 2019	10.35%	$9,656.47 x 0.1035 x (1767 days ÷ 365) = **$4,838.41** $20,793.93 x 0.1035 x (1595 days ÷ 365) =

			$9,404.70
Funeral expenses	25 November 2019 (date of payment)—28 March 2024	10.35%	$8,521.20 x 0.1035 x (1585 days ÷ 365) = **$3,829.81**
Medical expenses	No claim made	No claim made	No claim made
Loss of consortium	26 November 2019 (date of death)—28 March 2024	10.35%	$10,000 x 0.1035 x (1584 days ÷ 365) = **$4,491.62**
Hire of help to perform household services customarily provided by the late Mr Roussos to his wife	26 November 2019 (date of death)—28 March 2024	10.35%	$5,000 x 0.1035 x (1584 days ÷ 365) **= $2,245.81**
Solatium for the family (claimed for total sum of $35,000 on behalf of Irini, Helen, Koulla, Anna, Maria, Michael, and Manolis)	26 November 2019 (date of death)—28 March 2024	10.35%	$35,000 x 0.1035 x (1584 days ÷ 365) = **$15,720.66**
Total sum of interest claimed		**$128,119.24**	

The defendant's submissions

[13] The defendant submitted that the Court's power to award interest under s 84 of the *Supreme Court Act 1979* was discretionary. Guidance about the

exercise of the discretion as to interest on damages is found in the previous decisions of the Court in personal injury cases.

[14] As to general damages, the defendant submitted that the settled practice of the Court was to apply a rate of 4 percent for damages for pain and suffering. This practice is consistent with the High Court's decision in *MBP (SA) Pty Ltd v Gogic*[166] which considered awards of interest on damages for non-economic loss up to the date of judgment. The High Court stated:

> ... it is equally fallacious to hold that a plaintiff will be properly compensated for the delay in obtaining damages for pre-trial pain and suffering only if the award of damages for pre-trial pain and suffering only if the award of damages contains an amount for interest calculated at the commercial rate or rates. The function of an award of interest is to compensate aa plaintiff for the loss or detriment which he or she has suffered by being kept out of his or her money during the relevant period:... Damages for pre-trial non-economic loss, however, are assessed in accordance with the value of money as at the time of the award. In no way is any loss which a plaintiff incurs by reason of being deprived of his or her damages for pre-trial non-economic loss brought about by inflationary factors. In those circumstances, to award interest on damages for non-economic loss during the pre-trial period by reference to commercial rates is to compensate the plaintiff for a "loss" which he or she has not sustained.
>
> ...
>
> In the circumstances, the use of the 4 percent figure seems to us to be more likely to achieve fair and reasonable compensation for plaintiff's than the use of the real rate of interest figure...[167]

[15] The 4 percent rate has been consistently applied in the Northern Territory to awards of general damages since the High Court's decision in *MBP (SA) Pty*

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166 (1991) 171 CLR 657.

167 Ibid at pp 663 to 666.

Ltd v Gogic.[168] In *Rosecrance v Rosecrance*[169] the Full Court rejected a ground of appeal challenging an award of interest on non-economic loss at 4 percent per annum. The Court stated that the 4 percent figure "has been consistently applied in the Northern Territory Supreme Court" and that it was a "settled practice".[170] That position was expressly affirmed by the Court of Appeal in *Coull v Nationwide News Pty Ltd*.[171]

[16] I accept the defendant's submission. Accordingly, the interest on $150,000 at 4 percent per annum from 20 February 2018 to **13 May 2024** is:

$150,000 x 0.04 x (2063 ÷ 365) = $33,912.33

[17] Likewise, I determine that an interest rate of 4 percent should be applied for **loss of expectation of life**. Therefore interest for this head of damages is assessed as:

$4000 x 0.04 x (1630 ÷ 365) = $714.52

[18] As to **interest on funeral expenses**, the defendant accepted that interest runs from 25 November 2019 and that the general approach for pecuniary heads of damages such as funeral expenses is that the Court will apply "commercial" or "pecuniary" rates of interest. The defendant submitted that the Court should follow Mildren J's approach in *Young v Northern*

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168 (1991) 171 CLR 657.

169 (1998) 8 NTLR 1.

170 Ibid at p 7.

171 [2008] NTCA 10; (2008) 155 NTR 1 at [100].

Territory[172] to arrive at a commercial rate of interest. His Honour explained his approach as follows at p 278:

> Over the relevant period, 1984 to 1992, [the rates] have fluctuated considerably. No evidence was called allowing for any precision, and accordingly it has been left by the parties for the Court to apply its own knowledge as to these rates. I consider that a reasonable fair average over the whole period is about 12 percent, but, as the loss has occurred over most of the whole of that period on a week by week basis, I consider that the correct approach is to reduce the rate to 6 percent to allow for that factor: see *Volmer v Northern Territory Electricity Commission* (1985) 34 NTLR 12.

[19] The defendant accepted that the relevant rates are the rates identified in my Associate's email to the parties dated 12 April 2024. Those rates, which are the post judgment interest rates under Rule 39.06 of the *Federal Court Rules 2011* (Cth), are as follows:

Period	**Post-judgment interest rate**
1/07/2016—31/12/2016	7.75
1/01/2017—30/06/2017	7.50
1/07/2017—31/12/2017	7.50
1/01/2018—30/06/2018	7.50
1/07/2018—31/12/2018	7.50
1/01/2019—30/06/2019	7.50
1/07/2019—31/12/2019	7.25
1/01/2020—30/06/2020	6.75
1/07/2020—31/12/2020	6.25
1/01/2021—30/06/2021	6.10
1/07/2021—31/12/2021	6.10

172 (1992) 107 FLR 264.

1/01/2022—30/06/2022	6.10
1/07/2022—31/12/2022	6.85
1/01/2023—30/06/2023	9.10
1/07/2023—31/12/2023	10.1
1/01/2024—30/06/2024	10.35

[20] However, the defendant disputed that a rate of interest of 10.35 percent is the rate that should be applied for the entire period that interest is payable by the defendant. In support of this submission, the defendant submitted that two important points arose from Mildren J's decision in *Young v Northern Territory*.[173] First, where the rate has fluctuated during the relevant period, selecting the prevailing interest rate at the date of judgment is incorrect. In this proceeding, the rate has fluctuated between 6.10 percent and 10.35 percent from 29 November 2019 to 13 May 2024. The defendant submitted that it was unnecessary to be precise in calculating an average of those rates; an appropriate method is to identify a reasonably fair average over the whole period: *Young v Northern Territory*[174]; *Public Trustee as Administrator of the Estate of Matthew Leonard v Atileo*.[175] This is consistent with the overarching principle that an award of interest should always be approached in a broad and practical way and should not be allowed to assume disproportionate importance: *Cullen v Trappell*.[176] Based

173 (1992) 107 FLR 264

174 Ibid at [278]

175 [2023] TASSC 33 at [368]

176 (1980) 148 CLR 1 at 21 – 22

on those principals the defendant submitted that 7 percent was an appropriate rate.

[21] Second, for some heads of damages the rate should be reduced by 50 percent. Those are the heads of damages where loss occurs over the whole period on a "week by week" basis. That does not apply to special damages for funeral expenses, which were a one-off expense.

[22] Additionally, selecting the date of judgment as fixing the interest rate is arbitrary. Apart from anything else, that date falls outside the time period for which interest is awarded.

[23] I accept the defendant's submissions as to the approach to be taken in determining the relevant interest rate. **However, I find that rate to be 7.5 percent. Consequently, I determine that the interest on <u>funeral expenses</u> is as follows**.

$8,521.20 x 0.075 x (1631 ÷ 365) = $2,855.77

[24] As to past gratuitous care (***Griffith v Kerkemeyer***[177]), the defendant accepts that following *Grincelis v House*[178] it has been settled that, subject to exclusion by specific statutory provisions, pre-judgment interest can be awarded on *Griffith v Kerkemeyer* damages at commercial rates of interest. However, because the loss occurs gradually over the relevant period, the customary technique in assessing interest is to apply an interest rate to the

177 (1977) 139 CLR 161.

178 (2000) 201 CLR 321.

total damages and divide it by two, or alternatively to halve the interest rate.[179] I accept the defendant's submission.

[25] The plaintiff's claim for *Griffith v Kerkemeyer* damages covered two periods. The first period was from 1 January 2018 to 26 May 2019 for which the plaintiff was awarded $9,656.47 damages. The second period was from 27 May 2019 to 26 November 2019 for which the plaintiff was awarded $20,793.93 damages.

[26] Accordingly, I determine interest on the *Griffith v Kerkemeyer* damages to be as follows:

For the period from 1 January 2018 to 26 May 2019 -

($9,656.47 ÷ 2) x 0.075 x (1813 days ÷ 365) = $1,798.68

For the period 27 May 2019 to 12 May 2024

($20,793.93 ÷ 2) x 0.075 x (1641 ÷ 365) = $3505.76

[27] As to loss of consortium and solatium, the defendant made the following submissions. It is trite that interest is only to be awarded for past loss not future loss.[180] Further, damages for loss of consortium and solatium are damages for non-economic loss, they compensate a plaintiff for future loss of the deceased and go beyond merely compensatory damages. Therefore,

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179 *Grincelis v House* (2000) 201 CLR 321 at [22]; *Gill v Ethicon Sarl (No 5)* [2019] FCA 1905 at [513].

180 *Cullen v Trappell* (1980) 146 CLR 1 at [18]-[19]; *Amaca Pty Ltd v Wefel* (2020) 138 SASR 295 at [685].

the defendant submitted that no interest should be awarded for these heads of damages. Alternatively, the plaintiff submitted that, if interest was to be awarded, it should be awarded at a rate of 4 percent because the damages were for non-economic loss.

[28] In my opinion, interest on damages for loss of consortium and solatium should be awarded at a rate of 4 percent. These damages are similar in nature to damages for pain and suffering. They are assessed as follows:

1. Loss of consortium – Mrs Roussos –

   $10,000 x .04 x (1630 ÷365) = $1,786.30

2. Solatium – total sum of $35,000 on behalf of Irini, Koulla, Anna, Maria, Michael and Manolis

   $35,000 x .04 x (1630 days ÷365) = $6,252.05

[29] As to hire of help to perform household domestic services, I accept the defendant's submissions. These damages encompass both past and future losses. I previously found that Mrs Roussos would have a life expectancy of another five to 10 years. Consequently, the same approach should be taken to assessing interest on these damages as was taken with *Griffith v Kerkemeyer* damages. Accordingly I assess interest on these damages as follows:

($5,000 ÷ 2) x 0.075 x (1630 days ÷365) = $831.16

[30] Accordingly, I award interest in the sum of $51,656.57.

Costs

[31] The plaintiff seeks the following costs orders.

(a) The defendant pay the plaintiff's standard costs of these proceedings up to 20 May 2021; and

(b) The defendant pay the plaintiff's costs on an indemnity basis from 21 May 2021.

[32] In support of the application for indemnity costs, the plaintiff relied on a Notice of Offer of Compromise for $250,000 plus costs on the standard basis made on 21 May 2021 under rule 26.02 and 26.03 of the *Supreme Court Rules*; and an offer of settlement pursuant to *Calderbank v Calderbank*.[181] The defendant did not oppose the plaintiff's application.

[33] Given that the plaintiff's offer of compromise involved a real and genuine element of compromise and the plaintiff obtained a judgment significantly more favourable than the offers of compromise, the plaintiff is entitled to the costs orders claimed above. I order that:

(1) The defendant pay the plaintiff's costs of the proceeding on the standard basis up to 20 May 2021; and

181 (1975) 3 All ER 333.

(2) The defendant pay the plaintiff's costs of the proceeding on an indemnity basis from 21 May 2021.

Formal Orders

[34] I make the following orders:

1. Judgment is entered for the plaintiff in the sum of $329,751.87 for damages and interest.

2. The defendant is to pay the plaintiff's costs of the proceeding on the standard basis up to 20 March 2021 and thereafter on an indemnity basis.
