SUPREME COURT OF QUEENSLAND

CITATION:	*Cowen v Bunnings Group Limited* [2014] QSC 301
PARTIES:	**JANELLE ELIZABETH COWEN** (plaintiff) **v** **BUNNINGS GROUP LIMITED (ACN 26 008 672 179)** (defendant)
FILE NO/S:	BS634/13
DIVISION:	Trial
PROCEEDING:	Hearing
DELIVERED ON:	16 December 2014
DELIVERED AT:	Brisbane
HEARING DATE:	18, 19, 20 and 22 August 2014
JUDGE:	Alan Wilson J
ORDER:	**1. Judgment for the plaintiff in the sum of $700,000.00;** **2. The defendant pay the plaintiff's costs on the indemnity basis on the District Court scale.**
CATCHWORDS:	TORTS – NEGLIGENCE – ESSENTIALS OF ACTION FOR NEGLIGENCE – DAMAGE – CAUSATION – GENERALLY – where the plaintiff was employed by the defendant – where the plaintiff alleges that she swept up spilt fertiliser for over two hours and inhaled significant amounts of the fertiliser dust – where the plaintiff alleges that her inhalation of that dust was the causative factor for her development of pneumococcal meningitis, encephalitis and septicaemia – where the plaintiff sues the defendant in negligence for damages associated with her illness – where the plaintiff and defendant have agreed on all aspects of the negligence claim except causation – where the respondent alleges that a potential pre-existing respiratory illness, the plaintiff's smoking habit and/or the presence of atmospheric sulphur dioxide mean that the plaintiff cannot prove that it is more probable than not that her inhalation of the fertilisation dust was the causative factor of her primary illness, the meningitis – whether it is more probable than not that the plaintiff's exposure to the fertiliser dust caused her illness, and that the defendant was thereby negligent *Adelaide Stevedoring Co Ltd v Forst* (1940) 64 CLR 538, cited *Chappel v Hart* (1998) 195 CLR 232, cited

EMI (Australia) Ltd v Bes [1970] 2 NSWLR 238, cited
Fernandez v Tubemakers of Australia Ltd (1975) 2 NSWLR 190, cited
March v E & MH Stramare Pty Ltd (1991) 171 CLR 506, cited
National Insurance Co of New Zealand v Espagne (1961) 105 CLR 569, cited
Nicolia v Commissioner for Railways (NSW) (1970) 45 ALJR 465, cited
Ramsay v Watson (1961) 108 CLR 642, cited
Seltsam Pty Ltd v McGuiness & Anor (2000) 49 NSWLR 263, cited
Tabet v Gett (2010) 240 CLR 537, cited

COUNSEL: M Grant-Taylor QC for the plaintiff
G W Diehm QC and A Luchich for the defendant

SOLICITORS: Murphy Schmidt for the plaintiff
BT Lawyers for the defendant

[1] **Alan Wilson J**: Ms Cowen, 50, worked for Bunnings in Mt Isa in March 2008. Her work was primarily in an area called the 'Trade Shed' but her duties included keeping the area in which she worked and its surrounds, both inside and outside, clean and tidy.

[2] She alleged that plastic bags containing a fertiliser based on chicken manure, called 'Rooster Booster', had been stored outside uncovered, and exposed to the weather. Over time, she said, the bags began to deteriorate, bursting and splitting and spilling their contents. The fertiliser 'really stank', and customers were complaining. Although moist when packaged, the fertiliser which had spilled from split bags had dried in the sun and was '*... very fine. It was floating around in the air*'.[1]

[3] On 24 March 2008 she set to cleaning it up. It was, she said, about 40°C, and still. She worked for 2½ hours sweeping, dusting, rebagging and disposing of the fertiliser. She swept and shovelled spilled fertiliser back into less damaged bags, and taped them, and put unsalvageable material into a wheelbarrow and garbage bags and disposed of it. She did not wear any respirator, mask, filter or personal protective equipment (PPE). By the time she had finished she was, she said, '*absolutely covered*' in it.[2]

[4] As she did the work she also, she said, began to sneeze and regularly went back into the shed to get tissues. Her nose began to run. She was coughing. When she went home after work her partner, Henry Clay, would not let her into the house until she took her shoes off, and brushed her clothes. She had a shower. Her nose continued to run and she was still coughing and sneezing after she went to bed.[3] She went to work the next morning around 7am, with a headache. At work, it grew steadily worse. She remembers serving a customer and, next, waking out of a coma in Townsville Hospital.

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1 Transcript p 1-36.30-31.
2 T1-36.24.
3 Henry Clay, T1-52.9-15.

[5] She had been in that coma for some days. She had been admitted at Townsville after an urgent transfer from Mt Isa with pneumococcal meningitis, encephalitis and septicaemia.

[6] It seems that she had worked into the afternoon of 25 March, by which time she was so obviously unwell that she was instructed to go home. There, her partner also observed that she was unwell, and unsteady. She was taken to Mt Isa Base Hospital where her condition was diagnosed, and she was aerially transferred to intensive care at Townsville. Her convalescence was stormy, with serious complications requiring a long stay, and rehabilitation. She has some residual cognitive dysfunction.[4]

[7] She sued Bunnings for damages associated with her illness. Damages were agreed before trial at $700,000 clear of any refund to WorkCover Queensland.

[8] Bunnings admits a breach of its duty of care to Ms Cowen as her employer, and that it was foreseeable that the work she performed on this occasion might cause her some injury.

[9] Bunnings denies, however, that its breaches caused Ms Cowen's serious illness which, it says, was simply coincidental with the work she did that day. Other causes – a pre-existing respiratory illness, smoking, the presence of atmospheric sulphur dioxide in Mt Isa (or some combination of these factors) – are, Bunnings says, at least equally likely to have caused the illness. Those other possible causes mean, Bunnings argues, that Ms Cowen cannot prove that it is more probable than not that her exposure to Rooster Booster dust was causative. That, it happens, is the question upon which the case turns.

[10] The physical process by which Ms Cowen came to develop her illness is not in dispute. It is common ground that the illness was caused by a streptococcus pneumoniae, and that she presented with a severe manifestation of infection with that organism. The likely course initially involved the colonisation, by this bacterium, of Ms Cowen's upper respiratory tract – a very common event, such that 10-40% of the population have a similar colonisation at any given time. What made her condition dangerous was the relatively rare event that the bacterium descended into her lungs and thence, via her bloodstream, to her meninges and brain, and made her very ill.[5]

[11] As her case is pleaded she accepts that, before she began cleaning up the fertiliser, the offending bacterium was already colonised in her upper respiratory tract. Its presence is plainly probable, in light of the subsequent nature and progress of the illness she developed. Secondly, she alleges that the dust she inhaled caused irritation of her respiratory tract mucosa, and that irritation lead to the introduction of the bacterium into her blood stream, and brain. Thirdly, she alleges that this process lead to her severe illness.

[12] Neither the defendant nor any of the expert medical witnesses called in the case dispute the first proposition: i.e., that the bacterium was present in Ms Cowen's

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4 Dr McCormack's report 21 December 2009, Exhibit 1, p. 2.

5 Three of the medical experts – Drs McCormack, Bartley, and Mitchell – prepared a joint report recording their consensus about the progress of the infection in this way: Exhibit 7. In his evidence, Dr Clarke agreed: his report, Exhibit 5.

upper respiratory tract when she did the work of cleaning up the fertiliser dust. Nor is there any dispute that once the bacterium reached her blood stream, and then her meninges and brain, she became very ill with the disease which required her admission to hospital, and intensive care there.

[13] Rather, it is the second part of the process – what it was that caused the bacterium to travel from her upper respiratory tract to her bloodstream and, thence, her brain – which is in issue. Was it caused by her exposure to the dust or was it coincidental, with other possible causes outweighing or negating the possible effects of the dust exposure such that it cannot properly be categorised as causative, in law?

[14] Four eminent medical specialists gave evidence upon the point. Three are consultant physicians specialising in infectious diseases, and the fourth is a thoracic physician specialising in respiratory and sleep disorders. Two, Drs Clarke and McCormack, thought there was a causal connection on the balance of probabilities; the other two, Drs Mitchell and Bartley, agreed it was '*certainly not implausible*',[6] but were not persuaded to a level which would allow them to concede causation on the 'probability' test.

[15] In undertaking that exercise the medical practitioners were applying a legal test, not one that was scientific in the traditional sense or of a kind usually associated with epidemiology (which is the study of the incidence and distribution of diseases, and of their control and prevention). That implies no criticism of the doctors – they were invited, by the lawyers, to undertake the exercise and proffer an opinion.

[16] The scientific approach to causation, using what the Macquarie Dictionary calls the 'scientific method', customarily involves the identification of a problem; the collection of relevant data; the formulation of a hypothesis on the basis of this data; and, finally, empirical testing of the hypothesis to prove its validity.

[17] As Spigelman CJ observed in *Seltsam Pty Ltd v McGuiness & Anor*,[7] a number of well-known High Court decisions[8] show how, and why, the way the common law approaches causation is quite different from accepted, and customary, scientific methods. He said:

> The common sense approach to causation at common law is quite different from a scientist's approach to causation …. An inference of causation for the purposes of the tort of negligence may well be drawn when a scientist, including an epidemiologist, would not draw such an inference.[9]

[18] The task confronting a court, adopting this legal approach to causation, had earlier been explained by Mahoney JA in *Fernandez v Tubemakers of Australia Ltd*:[10] medical science may say in individual cases that there is no possible connection between the alleged causative event and the injury, and in that event the court cannot act as if there were a connection; but, if medical science is prepared to say

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6 Dr Mitchell: T2-65.29; Dr Bartley: T3-5.39-46.

7 (2000) 49 NSWLR 262.

8 *National Insurance Co of New Zealand v Espagne* (1961) 105 CLR 569, at p. 591; *March v E & MH Stramare Pty Ltd* (1991) 171 CLR 506, and *Chappel v Hart* (1998) 195 CLR 232.

9 (2000) 49 NSWLR 262, at p. 286, para [143].

10 (1975) 2 NSWLR 190 – Glass JA agreeing, Reynolds JA dissenting.

the connection is *possible* then it is up to the court to decide if it is probable, to the requisite degree.[11]

[19] This emphasis in the law of tort upon the process of making findings about causation based upon degrees of probability rather than scientific precision has a long and respectable pedigree. As Dixon CJ and others[12] explained in *Ramsay v Watson,*[13] a medical expert may express an opinion as to the nature and cause, or probable cause, of an ailment, but it is for the jury to weigh and determine the probabilities; and, in undertaking that exercise, the Court is not to transfer the task to the experts but, rather, to ask itself: '*Are we on the whole of the evidence satisfied on a balance of probabilities of the fact?*'.[14]

[20] So it has been said, for example, that a finding of a causal connection may be open even if there is no medical evidence to support it,[15] or when the medical evidence does not rise above the opinion that a causal connection is possible.[16]

[21] More recently the High Court again both explained this approach to causation, and confirmed its continuing validity, in *Tabet v Gett*.[17] Kiefel J (with whom Hayne, Crennan and Bell JJ agreed) said that the purpose of proof at law, unlike science or philosophy, is to apportion legal responsibility – and that requires the courts, by a judgment, to '*reduce to legal certainty questions to which no other conclusive answer can be given*'.[18]

[22] Earlier, Kiefel J had spoken of the way the courts undertake this exercise. The common law, her Honour said, requires proof by the person seeking compensation that the negligent act or omission caused the loss or injury constituting the damage; but all that is necessary for that purpose is for the plaintiff to show that, according to the course of common experience, the more probable inference arising from the evidence is that the defendant's negligence caused the injury or harm. '*More probable*' means, she said, no more than that upon a balance of probabilities such an inference might reasonably be considered to have some greater degree of likelihood. But it does not, as Kiefel J emphasised, require certainty.[19]

How bad was the dust?

[23] The medical evidence, discussed later, focused in part on the nature and severity of the effects the fertiliser dust might have had upon Ms Cowen's upper respiratory tract, and the mucosa there.

[24] Ms Cowen's evidence about the strong, intrusive and unpleasant effects of the dust was coincidentally confirmed in a report prepared for Bunnings in 2010 after it attempted, in a scientific way, to recreate the work she was performing. An environmental scientist, Ms Dewar, supervised the exercise and prepared a report in

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11 Ibid, per Mahoney JA at pp. 199-200.
12 McTiernan, Kitto, Taylor and Windeyer JJ.
13 (1961) 108 CLR 642.
14 Ibid, at p. 645.
15 *Nicolia v Commissioner for Railways (NSW)* (1970) 45 ALJR 465.
16 *EMI (Australia) Ltd v Bes* [1970] 2 NSWLR 238.
17 (2010) 240 CLR 537.
18 Ibid, at p. 578.
19 Ibid.

the name of her employer OCTIEF Pty Ltd in August 2013.[20] At the end of the clean up, according to the report, all three of the human participants were covered in fertiliser dust and were all sniffling, and their sinuses were irritated. One, Michelle, had dust which had gone through her suit onto her skin, and clothing. Despite wearing PPE, which included safety eyewear, that employee later developed an infection in her right eye.

[25] Ms Cowen impressed as a direct, forthright, and honest witness when she gave evidence. I saw no reason to doubt the truthfulness of anything she said – but, if, confirmation was needed that the work she performed produced a great deal of dust which seriously irritated her sinuses, the OPTIEF report provided strong corroboration.

[26] I am persuaded the work exposed her to a large amount of dust, some of which she breathed in, and which seriously aggravated her upper respiratory tract.

The medical evidence

[27] All four experts are highly qualified, and experienced. As remarked earlier, the principal difference between them concerned their conclusions on the question of causation, when measured against the 'legal' test – i.e., on the balance of probabilities. Dr McCormack and Dr Clarke were prepared to make that leap, as it were, but Dr Mitchell and Dr Bartley were not.

[28] Three of the specialists (Drs McCormack, Bartley and Mitchell) joined in discussions leading to a joint report.[21] I did not understand Dr Clarke to disagree with anything in it. The report concludes that:

- Ms Cowen's illness was caused by a streptococcus pneumoniae;
- The illness represented a severe manifestation of infection with that organism;
- The likely source was colonisation of her upper respiratory tract;
- The literature suggests that such colonisation occurs in anything between 10% and 40% of the normal adult population at any time;
- Ms Cowen's cigarette smoking was a contributing factor to this colonisation; and
- Her streptococcus pneumoniae infection was a consequence of a change from colonisation by this organism, to infection in her blood stream, brain and meninges.

[29] Like Dr McCormack, Dr Clarke was also influenced by the close proximity in time between exposure to the fertiliser dust and the onset and signs and symptoms of respiratory tract irritation and, within a short period, pneumococcal meningitis.

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20 Exhibit 10.

21 Exhibit 7.

[30] Dr Clarke also thought that the fertiliser product Ms Cowen inhaled, containing endotoxins, was something which had a particular tendency to bring about upper respiratory tract syndrome. None of the other specialists shared this view about the possible relevance or importance of endotoxins and I have not, with respect, given it any weight. That said, Dr Clarke's conclusion about a causal connection between Ms Cowen's work and her illness did not depend upon that aspect of his opinion, and its rejection does not mean his opinions on causation ought, otherwise, be rejected.

[31] Dr McCormack was the first of the specialists to advance a theory involving the three-stage process discussed earlier, about which he remained unshaken during his oral evidence. As noted earlier, that theory was supported by Dr Clarke and, while not accepted outright by Dr Mitchell or Dr Bartley, was conceded by each of them to be '*certainly not implausible*'.

[32] Primarily (putting aside other possible causes for the moment) what stopped Drs Mitchell and Bartley from a more ready acceptance of Dr McCormack's theoretical pathway to the serious illness Ms Cowen eventually developed, on the balance of probabilities, was the absence of any mention in the medical or scientific literature of any similar instance or example recording a link between irritation of the nasopharynx by dust, and invasive pneumococcal disease. I accept that Dr Bartley, in particular, undertook an exhaustive search of the published literature, and found nothing useful.

[33] It was also submitted for Bunnings, against the theory, that Dr McCormack wrongly assumed a temporal connection between Ms Cowen's exposure to the fertiliser, and development of pneumococcal meningitis. In his evidence Dr McCormack said that the interval between introduction of the bacterium into the body and the manifestations of the symptoms may have been as short as 15 hours,[22] and, as I understood their evidence, neither Dr Mitchell nor Dr Bartley disagreed with that. I am satisfied that, as a time-line for the process of invasion Dr McCormack advances, it is not discordant with the sequence and timing of events described by Ms Cowen, and her partner Mr Clay.

[34] To the layperson, the temporal connection is something which is striking, and not easily dismissed. So, too, is Ms Cowen's description of the volume, the intrusive nature, and the immediate unpleasant effects of the dust – i.e., the extreme conditions in which she worked for over two hours, and their immediate and continuing unpleasant effects.

[35] These are things which suggest a '… *presumptive inference which this sequence of events would naturally inspire in the mind of any common-sense person uninstructed in pathology*'[23] – i.e., they raise a powerful impression, in the mind of a layperson, that a causal connection is likely to exist.

[36] For the reasons explored earlier, the opinions of the medical experts are not, with respect, conclusive, and (again, intending no disrespect) their views about causation, measured against the legal test – the balance of probabilities – are relevant, and of interest, but nothing more. It is for the court to decide that matter.[24]

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22 T2-32.40, T2-33.10.

23 *Adelaide Stevedoring Co Ltd v Forst* (1940) 64 CLR 538, per Rich ACJ at p. 563.

24 *Seltsam Pty Ltd v McGuiness* (supra), at pp. 143-4.

[37] The absence of any support in the medical/scientific literature is also not determinative. It does suggest that this event, if it occurred, was unusual and may have been rare or, even, surprising and unanticipated. That is, again, a material factor in determining causation but it is not determinative.

[38] As Spigelman CJ observed in *Seltsam*, the legal concept of causation requires the court to approach the matter in a distinctly different manner from that which may be appropriate in either philosophy or science, including the science of epidemiology.[25] The fact that medical science has not recorded a similar course of infection does not mean that it has not happened – or, importantly for present purposes, in a case where there is medical evidence indicating it is on one view quite possible and, on another, that it is not impossible or implausible, that it could not have occurred.

[39] The compelling features pointing to a causal connection are, firstly, the strong relationship in time between one event, and the other. The second is the nature of the material to which the plaintiff was exposed and the duration of exposure which, on any view, was serious and unpleasant and, in particular, caused immediate and prolonged upper respiratory tract symptoms. Dr McCormack thought the temporal connection weighed against coincidence, and that is also, again compellingly, the common-sense conclusion. The absence of similar examples in the literature cannot mean, as Drs Mitchell and Bartley had to concede, that the internal physical process he postulated is improbable or, in their words, implausible.

[40] In his oral evidence Dr Bartley conceded that the intensity of the reaction brought about by the phenomena of irritation or inflammation of the respiratory mucosa has a bearing on the likelihood of the transference of bacteria into the blood stream, although he said that the reaction would have to be extremely intense.[26] The exposure to dust suffered by the plaintiff, according to the description of the incident which she has given, and which I accept, was intense and unpleasant and involved inhaling significant amounts of dust over a lengthy period. Dr McCormack described the exposure to dust as 'pretty extreme'.[27]

[41] Two of the four experts find the connection persuasive, on the legal test. Two do not, but in reliance, principally, on the absence of evidence of any previous, similar example. For the reasons just explored, to the layperson the connection, both in time and in light of the severe nature of the exposure, is compelling.

[42] Absent any other potential cause, the evidence in its totality is in my view sufficient to warrant the conclusion that there is a causal connection, on the balance of probabilities, between irritation of Ms Cowen's respiratory mucosa and her subsequent grave illness.

Other possible explanations/causative factors?

[43] The plaintiff can only succeed, however, if she can establish her infection occurred by this process on the balance of probabilities despite what Bunnings argue were, at least, equally likely causal factors.

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25 At para [78].
26 T3-7.6-11.
27 T2-33.14.

[44] In its submissions at the conclusion of the trial Bunnings argued that there were five other possible causes which, either alone or in combination, were at least equally possible/probable as explanations for Ms Cowen's illness, and that two of them – a pre-existing respiratory illness, and the fact that she was a smoker – meant her action must fail.

[45] Two of those five factors were not pleaded – that the plaintiff might have had some other unknown condition, like gingivitis, or a mouth ulcer; or that she was colonised by a different serotype of the bacteria.

[46] There was no evidence that the plaintiff had a mouth ulcer or gingivitis, and neither condition was put to her. In the absence of those things, the defendant cannot rely upon either as a putative causative factor.

[47] The medical evidence showed that there are different serotypes of the pneumococcal bacterium – different sub-species, some of which will be more virulent than others. Using another bacterium as an example, HIV-1 is (Dr McCormack said) more aggressive and more virulent than HIV-2, and that variability amongst different strains is true for the pneumococcal germ as well.[28]

[48] Colonisation with a serotype with which a person's body is unfamiliar, and to which it has not developed a level of immunity, can cause serious problems. Dr McCormack said a new serotype could '… *cause a problem in the sense that you don't have immune memory for that particular strain … you have a new strain to which your immunity is not in tune*'.[29]

[49] What Bunnings argues, again as I understand it, is that the sudden presence of a different serotype in Ms Cowen's body explains her illness, because its novelty meant that she lacked immunity to it. But that would not, it seems to me, provide an effective defence, for two reasons. First, if the causal theory advanced by Dr McCormack is plausible – and, for reasons already explored at length, I accept that it is – then the nature of the serotype which entered her blood stream as a consequence of her mucosal distress is irrelevant. Secondly, the proposition that she coincidentally, but as a matter of very bad luck, was infected with a novel serotype for which she lacked immunity is something which lacks a credible evidentiary basis. Dr McCormack was, fairly, prepared to accept that other casual explanations (like a different serotype) were 'quite plausible', but he went on to describe Ms Cowen's exposure to the fertiliser as an 'acute' and 'big' event and, absent of that, 'it wouldn't be an issue at all'.[30]

[50] Nothing in the evidence here suggests that this is what happened to Ms Cowen, and the question was never investigated. Nor was it put to Dr McCormack that the introduction of a different serotype would have made any difference to his theory about the mechanism, and the progress or advance of the infection, which he postulated.

[51] The primary question is whether the plaintiff's exposure to dust was causative, in terms of the development of her illness. As I understand Dr McCormack's

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28 T2-47.37-45.
29 T2-49.15-19.
30 T2-49.44-2-50.4.

evidence, the process that led him to discern a causal connection is not affected by the nature of the serotype.

[52] At the highest, the possibility that Ms Cowen's illness was caused not by the process described by Dr McCormack and accepted by Dr Clarke but, rather, by the unfortunate but highly coincidental introduction of a new serotype into her body suffers the same disadvantages that attach to Bunnings' arguments against the theory itself, as an explanation of causation of Ms Cowen's illness: the possibility is remote, improbable, and extraordinarily coincidental when compared with a simple, but plausible, explanation advanced by those doctors.

[53] The next ground argued is exposure to sulphur dioxide in the air in Mt Isa. There was evidence that, in 2008, Mt Isa had 'issues' with respect to atmospheric sulphur dioxide and it is recognised in the literature that this is a risk factor for invasive pneumococcal disease. It was submitted that the plaintiff, as a resident of and worker in that town, had been exposed to that gas on a chronic rather than an acute basis.

[54] There is evidence of a 'spike' in airborne sulphur dioxide concentrations on 24 March, but that reading was taken at a point which is not useful in terms of determining levels at the location of the Bunnings store.[31]

[55] The plaintiff had also been smoking cigarettes for nearly three decades, since her mid-teens. All the specialists acknowledge it is a risk but, as Dr McCormack said[32] in a previously healthy woman of Ms Cowen's age, the likelihood of streptococcus pneumoniae colonisation progressing to more invasive disease '… *would be extremely low, less than 1%*'. Dr McCormack also observed, in his oral evidence, that most people who smoke do not develop pneumococcal meningitis.[33]

[56] It would be extraordinarily coincidental, to an extent which would make it highly surprising, if either of these potential causes happened to come into effect at, or during, this very time; and, again, they have to be weighed against the compelling temporal connection between Ms Cowen's heavy exposure to dust one afternoon, and a development of a grave illness the next day.

[57] The other potential causative factor most vigorously advanced on Bunnings' behalf was that Ms Cowen was suffering from cold or flu-like symptoms *before* 24 March 2008. Dr McCormack accepted that, if she had an upper respiratory tract infection at the time of her exposure, that of itself was an '*…entirely sufficient and plausible explanation for her progress to suffering from pneumococcal meningitis, regardless of the exposure to dust*'.[34]

[58] The plaintiff strongly denied that she had a cold or flu before 24 March, and so did her partner Mr Clay. Fellow workers said the same thing. Ms Cowen conceded that she did not like going to doctors and, it might be inferred, would not have sought medical treatment, and would have continued to work, even if she had an illness of that kind. There is, however, evidence that she did go to Mt Isa Hospital in August 2007 complaining of cold and flu-like symptoms and a history of a cough and

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31 Ms Dewar, T1-73.17-23.
32 Exhibit 1, p. 2.
33 T2-44.41-42.
34 T2-44.02-04.

running nose for two days, and was diagnosed with an upper respiratory tract infection. She said that she was, at the time, concerned about the swine flu that was going around and felt that she could not control her symptoms. It cannot be said, then, that her personal reluctance to seek medical treatment was irrational, or pig-headed, or of such a nature that it was more likely than not that, even if she was ill before she undertook this task, she would not have sought medical assistance.

[59] As noted earlier, the plaintiff impressed as a reliable and honest witness. The only evidence that she was suffering from anything resembling an upper respiratory tract infection came from her supervisor, Ms Elaine Joseph, who gave evidence that Ms Cowen had been suffering from the flu for at least three weeks before 24 March.[35]

[60] Evidence was produced from Ms Joseph's general practitioner, Dr Hewage, who had made a note of being consulted by Ms Joseph on 28 March, after Ms Cowen had been diagnosed with meningitis and was in a coma, and the notes recorded that '*... she had the same symptoms following her by one week*'. The inference, it was submitted for the defendant, is that the doctor's notes corroborate Ms Joseph's recollection.

[61] Cross-examination of Dr Hewage confirmed, unsurprisingly, that she had no independent recollection of the consultation and could not say to which person – the plaintiff, or Ms Joseph – her use of the words 'she' and 'her' in the note refer. Dr Hewage properly conceded that her note is equally consistent with the notion that Ms Joseph had told her that her workmate had the same symptoms after her, i.e., one week after Ms Joseph. When that is appreciated, Dr Hewage's evidence does not corroborate that of Ms Joseph and, indeed, possibly consistent with what the plaintiff said.

[62] There were other inconsistencies between Ms Joseph's evidence, and that of Ms Cowen and her fellow employees, Ms Rebecca Hornberg, Mr Graham Thompson and Ms Phillipa Caldwell. Ms Joseph had claimed that the Rooster Booster was covered with a sheet or plastic to keep the elements off it, but all of these employees gave quite contrary evidence that it was never covered at all. She said that two of the workers in the trade shed, Mr Thompson and Mr Rodney McCarthy, applied the covering of black plastic, but both denied it.

[63] Ms Joseph also made a statement on 6 August 2013[36] in which she said that before Ms Cowen became ill all of the staff, including her, were sick with the flu. In cross-examination she at least partially retracted this: Bunnings had around seven employees at the time, but she could ultimately name only three – herself, Ms Cowen and Ms Rice – who, she said, had the flu. There was no evidence in the way of timesheet or attendance records suggesting any staff absenteeism in this three week period.

[64] None of Ms Cowen's fellow employees had any recollection that she was ill for any period before 24 March. She says she was not. Ms Joseph's evidence must, for the reasons just explored, be discounted.

[65] I am satisfied that Ms Cowen was not suffering from any cold or flu-like symptoms, or symptoms suggesting an upper respiratory tract infection, before the incident in

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35 Exhibit 28, para [28]; T2-57.47-T2-58.01.

36 Exhibit 28.

which she was exposed to dust at Bunnings on 24 March. It is not, then, a potential causative factor to be taken into account.

Conclusion

[66] This is a case in which the plaintiff suffered a serious illness shortly after a severe exposure to dust which, I am satisfied, led her to develop symptoms which can instigate the internal physical process of infection accepted by two of the four medical experts called in the case as providing an explanation for her subsequent serious illness.

[67] While the event is unusual, the close relation in time between exposure and the development of the illness means that explanation accords with common sense and provides an acceptable causative pathway, according to the appropriate legal test.

[68] While other causes had been advanced as possibilities, one (a pre-existing illness) is exploded by the evidence, and all the others involve such a high degree of coincidence – and, indeed, awfully bad luck – that they are considerably less plausible, to a degree which means that the attribution of causation to them would, with respect to the doctors, defy common sense.

[69] The plaintiff, for these reasons, must succeed in the case and have judgment against the defendant.