Neutral Citation Number: [2016] EWCA Civ 86

Case No: B3/2014/4329

IN THE COURT OF APPEAL (CIVIL DIVISION)
ON APPEAL FROM THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION
MR JUSTICE JAY
HQ12X03121

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 15/02/2016

Before :

THE MASTER OF THE ROLLS
LORD JUSTICE TOMLINSON
and
LORD JUSTICE SALES

- -

Between :

CARL HENEGHAN (SON AND EXECUTOR OF JAMES LEO HENEGHAN, DECEASED) **Appellant**

- and -

(1) MANCHESTER DRY DOCKS LIMITED
(2) 00722056 LIMITED
(3) CARILLION CONSTRUCTION (CONTRACTS) LIMITED
(4) R. BLACKETT CHARLTON LIMITED
(5) S.C. CHEADLE HULME LIMITED
(6) KELLOGG BROWN & ROOT LIMITED **Respondents**

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David Allan QC and Simon Kilvington (instructed by **Slater & Gordon (UK) LLP**) for the **Appellant**
David Platt QC and Peter Houghton (instructed by **BLM LLP**) for the **Respondents**

Hearing dates : 18 & 19/01/2016

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Approved Judgment

Master of the Rolls:

1. This case concerns (i) an individual (Mr James Heneghan) who died of lung cancer; where (ii) it is common ground that the cancer was caused by his exposure to asbestos fibres; (iii) he was exposed to the asbestos inter alia whilst he was employed successively by each of the six defendants; (iv) biological evidence cannot establish which (if any) of the exposures triggered the cell changes in his body which led to his contracting the disease; but (v) epidemiological or statistical evidence can establish by how much the exposure attributable to each defendant increased the risk that he would contract the disease. The question is: how should the law deal with the issue of causation as between the claimant and each defendant in these circumstances? Jay J applied the so-called *Fairchild* exception (*Fairchild v Glenhaven Funeral Services Ltd* [2002] UKHL 22, [2003] 1 AC 32) and awarded damages against each defendant in proportion to the increase in risk for which it was responsible. The claimant appeals with the permission of the judge and says that the judge should have held that each defendant had materially contributed to the cancer and was liable for damages in full. The defendants say that the judge reached the right conclusion for the right reasons.

2. Mr Heneghan was born on 8 March 1938. During the course of his working life, he was exposed to respirable asbestos fibres and dust. He was diagnosed as suffering from lung cancer in early 2012 and died from the disease on 3 January 2013.

3. He was employed by the six defendants successively between 1961 and 1974. There were earlier employers who have not been sued. The parties have agreed that (i) Mr Heneghan's exposure to asbestos over the course of his working life can be quantified as 133 fibres/ml years; (ii) the total exposure for which the six defendants were responsible was 46.9 fibres/ml years (i.e. 35.2% of the whole exposure); (iii) the doses or exposures for which the defendants were responsible range from 2.5% to 10.1%; (iv) cumulative exposure of 25 fibres/ml years is sufficient to enable an inference properly to be drawn that lung cancer in any individual case is attributable to asbestos, provided that there is a minimum ten year interval from first exposure to onset of the cancer; (v) the 25 fibres/ml year criterion remains appropriate for exposure to mixed fibre types with a preponderance of amphiboles: if exposure involved equal quantities of amphiboles and chrysotile, then cumulative exposure of 40 fibres/ml year is probably necessary to double the risk of lung cancer; and (vi) since the deceased's exposure included 114 fibres/ml years of amphibole, the relevant threshold is likely to be below the threshold of 40 fibres/ml years, and the parties have been content to proceed on the agreed basis of a more than five-fold increase in the risk of lung cancer attributable to Mr Heneghan's cumulative exposure to asbestos over the whole of his employments.

4. In these proceedings, damages are claimed on behalf of the estate of the deceased and on behalf of his widow against the six former employers alleging exposure to asbestos in breach of duty and further alleging that the asbestos to which each of the defendants exposed him caused his lung cancer.

5. Judgment was entered by consent against all of the defendants. The consent order identified that there was an issue as to "whether each defendant is liable for damages in full or for only a portion of the damages". It was common ground that (i) if the claimant's case on causation was right, he was entitled to £175,000, being the

damages in full contemplated by the order; and (ii) if the defendants' case on causation was correct, the recoverable damages were £61,600 on the basis of the *Fairchild* exception.

6. In an impressive judgment given on 11 December 2014, Jay J accepted the submissions of the defendants and awarded the claimant £61,600. The appeal is of some general importance because this is the first time that the Court of Appeal has considered whether the *Fairchild* exception should be applied to a case of multiple exposures leading to lung cancer (as opposed to mesothelioma).

Some medical common ground

7. The judge summarised some medical common ground in these terms:

> "15. On 12 November 2013 the medical experts produced a Joint Statement to the Court, points 2, 3 and 5 of which are relevant for present purposes:
>
> > '2. His risk of lung cancer was increased by smoking and by asbestos exposure.
> >
> > 3. On the balance of probabilities he would not have developed the lung cancer if he had not been exposed to asbestos.
> >
> > …
> >
> > 5. The risk that lung cancer will develop increases with the amount smoked and with the dose of asbestos received. If a lung cancer does develop, then the duration and severity of symptoms and the eventual outcome are not affected by the amounts of smoking or asbestos exposure which led to that increase in risk.'
>
> 16. The medical experts are also agreed about the multiple mechanisms involved in the carcinogenesis of asbestos-related lung cancer. This involves a consideration of the microbiological processes occurring in cells, in particular cellular DNA, of the inhibitive effect asbestos has on programmed cell death, and of the reduction in the effectiveness of the body's natural defences against cancer caused by the presence of asbestos fibres in lung tissue. The precise detail does not matter for present purposes; it is sufficient to record that the experts are in general agreement that anything between four and seven stages in cellular mutation is required before the emergence of a cancer cell which may potentially progress to become a clinically apparent carcinoma of the lung.''

The two stages of the causation question

8. It is not in dispute that the first question (the "what" question) is what probably caused the lung cancer. Was it asbestos, smoking or something else? It is not in dispute that epidemiology can be deployed to answer this question. Nor is it in dispute that the epidemiological evidence shows on the balance of probabilities that

the deceased's exposure to asbestos was a cause of his lung cancer. That is because it establishes that the relative risk of the cancer having been caused by exposure to asbestos was greater than 2:1: i.e. the risk was "doubled". The "doubles the risk" test is one that applies epidemiological data to determining causation on the balance of probabilities where medical science does not permit determination with certainty of how an injury was caused. If statistical evidence shows that a tortfeasor more than doubled the risk that the victim would suffer the injury, it follows that it is more likely than not that the tortfeasor caused the injury. Some doubt has been expressed as to the validity of the doubling the risk test, at any rate for mesothelioma cases: see, for example, per Lord Phillips in *Sienkiewicz v Greif UK Ltd* [2011] UKSC 10, [2011] 2 AC 229 at paras 94 to 106. But there can be no doubt as to the validity of applying it to answer the first question. The deceased's lifetime risk of developing lung cancer had he not smoked and not been exposed to asbestos fibres was of the order of 0.5%. As I have said, the parties proceeded on the agreed basis that the cumulative effect of the deceased's exposure to asbestos over the whole of his employments resulted in a more than fivefold increase in the risk of his contracting the disease. I leave out of account the complication of the extent to which, having regard to the multiplicative effect of the smoking, the risk attributable to the exposure to asbestos was even greater. On any view, the exposure to asbestos more than doubled the risk of the deceased contracting the disease. That is why it was common ground that it was established in this case that the cancer was caused by that exposure.

9. The second question (the "who" question) arises in a multi-contributor case where the issue is which contributor's asbestos caused the cancer. Medical science is unable to determine this issue. The question in this case is whether epidemiological evidence can provide the answer. It is common ground that it could not be proved that the exposure attributable to any individual defendant had doubled the risk that Mr Heneghan would develop lung cancer. None of the defendants exposed him to an amount of asbestos close to 25 fibres/ml years (see para 3 above). Only one employer, W Blackwell (who had not been sued), was responsible for exposure in excess of 25 fibres/ml years (56% of the total exposure).

The Fairchild exception

10. In *Fairchild,* all the claimant employees had been exposed to asbestos dust during periods of employment with more than one employer. They contracted mesothelioma. This is an indivisible disease, i.e. one whose severity does not depend on the extent of the exposure to asbestos. In that sense, it is similar to lung cancer and differs from diseases such as pneumoconiosis and silicosis. But it was accepted that the *risk* of developing mesothelioma increased in proportion to the quantity of asbestos dust and fibres inhaled: the greater the quantity of dust and fibres inhaled, the greater the risk. There was no way of identifying, even on a balance of probabilities, the source of the fibre or fibres which initiated the genetic process which culminated in the malignant tumour. Lord Bingham referred to this at para 7 as the "rock of uncertainty".

11. In order to surmount this difficulty, the House of Lords fashioned a modified approach to proof of causation: proof that a defendant employer had materially contributed to the risk of contracting the disease was sufficient to satisfy the causal requirements for his liability. This approach had been heralded earlier in *McGhee v National Coal Board* [1973] 1 WLR 1.

12. In *Fairchild,* the House of Lords did not address the question of apportionment between defendants. That issue was confronted squarely by the House in *Barker v Corus UK Ltd* [2006] UKHL 20, [2006] 2 AC 572. In that case, the deceased who died of mesothelioma had been exposed to asbestos during three periods of his working life: while working for a company which had become insolvent; while working for the defendant; and while he was self-employed. It was held that the defendant was liable only in proportion to his own contribution to the exposure to the asbestos and therefore to the risk that the deceased would contract mesothelioma. It was irrelevant whether the other exposure was tortious, non-tortious, by natural causes or caused by the employee himself.

Summary of the judgment of Jay J

13. The claimant's case was that it was more likely than not that the deceased's exposure to asbestos when employed by each of the defendants contributed to the causation of his cancer: fibres from each source of exposure must have contributed to the carcinogenesis.

14. The judge rejected Dr Rudd's reliance on the epidemiological evidence to support this case (Dr Rudd was the claimant's expert). At para 30, the judge said:

> "Whether material contribution may be inferred in relation to any of the Defendants raises difficult questions. At one stage during the course of the trial, I was inclined to think that Dr Moore-Gillon [the defendants' expert] – in refusing to accept that such an inference may be drawn – was applying a higher standard of proof than the probabilistic standard that the common law recognises, and on that account I considered that I could properly embrace Dr Rudd's thesis into its furthest reaches. Upon further reflection, and mindful that no other court has been so bold as to infer material contribution to the damage in a lung cancer or similar case where there has been no doubling of the risk, I have concluded that even if Dr Moore-Gillon did err in that respect, he was right to baulk at drawing such an inference. Although the causation of lung cancer is dose-related, it does not follow from the proposition that because 2.5% of the dose may be attributed to the Fourth Defendant, that Defendant has materially contributed to the carcinoma. Asbestos burden cannot be equated with the silica dust which causes pneumoconiosis. The greater the accumulation of such dust in the lungs; the greater the damage that is being caused to the lung tissue of an individual patient with that disease. The dust is directly contributing to the disease process. The greater the exposure to asbestos fibres, on the other hand, the greater the risk that lung cancer may result. Although I would agree that 2.5% represents millions of asbestos fibres with the potential to cause a fatal disease process, whether these fibres actually did so remains an open question. Recourse to epidemiology or statistics does not resolve that agnosticism, nor does recourse to the microbiology. Indeed, an accurate application of the epidemiological and

> statistical data leads to the conclusion that, in relation to the causative potency of the Fourth Defendant's involvement, it cannot be said on the balance of probabilities that this exposure contributed to the deceased's lung cancer. It increased the risk of that cancer occurring, but the quantum of that increase was by a very considerable margin below 51%."

15. At para 60, he accepted that it would be "close to impossible" to devise an epidemiological study which was capable of discriminating between different sequential exposures. On the facts of this case:

> "it is simply a question of doing the basic arithmetic and reaching the conclusion that the contributions of each of the defendants, whether viewed individually or collectively, amount to less than 51%. The relative risk is less than 2 and/or the chances are less than 51%".

16. At para 61, he said that he would have seen no difficulty in principle in concluding that a 56% contribution to Mr Heneghan's overall exposure in the case of W Blackwell should be regarded as sufficient to prove "this hypothetical claim on the balance of probabilities". This was a matter of "basic arithmetic". This observation was *obiter:* it was formed no part of the judge's essential reasoning. Both parties criticise this paragraph of the judgment (for different reasons). It is not necessary to decide whether these criticisms are well-founded.

17. At para 63 he said:

> "In my judgment, it makes no sense in this sort of case to say that each Defendant's exposures materially contributed to the disease process, invoking the second limb of Lord Reid's formulation in *Bonnington Castings*. Adopting Lord Reid's approach in *McGhee*, the evidence does not establish that every asbestos fibre, or exposure, was or must have been implicated in the disease process. The aetiology of lung cancer is different from silicosis or pneumoconiosis. In those cases, which depend on the gradual accumulation of lung dust, the disease process is continuous and the concept of material contribution has an intelligible role. In lung cancer cases, there is no analogue to the gradual accumulation in the lungs of asbestos or cigarette smoke. The risk of the disease eventuating is proportionate to the quantum of exposure, but that is a statistical judgment, not an assessment which may be linked to the physical presence of deposits of dust in the lung."

18. At para 79, he rejected the submission that mesothelioma and lung cancer cases should be treated in different ways. He said that the preconditions for the application of the *Fairchild* exception are satisfied in lung cancer cases. These are most clearly and helpfully set out at para 170 of Lord Rodger's opinion. If that were wrong, the claimant would recover no damages at all. At para 81, he said that, although he accepted that mesothelioma and lung cancer are distinguishable in aetiological terms,

they are legally indistinguishable. Accordingly, he held that apportionment was required and he awarded damages on that basis.

The appellant's case

19. Mr Allan QC accepts that, if the judge was right to say that mesothelioma and lung cancer are legally indistinguishable, then he was right to apply the *Fairchild* exception qualified by *Barker*. That is because the reversal of *Barker* brought about by section 3 of the Compensation Act 2006 only applies to mesothelioma claims. However, he submits that the evidence established that each of the six defendants materially contributed to the deceased's lung cancer. Whereas in a multi-employer mesothelioma case, the claimant cannot prove that each defendant materially contributed to the disease, the position is otherwise in a multi-employer lung cancer case.

20. He submits that it has been proved that the deceased's lung cancer, attributable to cumulative exposure, was caused by asbestos so that each tortfeasor who contributed to the exposure contributed to the disease. The judge was wrong to hold that it was only if a defendant was responsible for at least 51% of the increase in the risk that causation against an individual defendant could be established. In the case of a cumulative exposure, the court should infer that each source of asbestos contributing to the total exposure materially contributed to the disease.

21. In summary, the judge should have concluded that (i) it was established that the lung cancer had been caused by the deceased's exposure to asbestos; (ii) the causal connection between the lung cancer and the asbestos was established by reason of the cumulative dose; (iii) the asbestos acted in multiple ways to promote carcinogenesis at the cellular level; (iv) the asbestos from each defendant was likely to have been inhaled and distributed in the lungs in a similar way; (v) the fibres from each source were likely to have played a part in the carcinogenic process; and (vi) each defendant, therefore, materially contributed to the contraction of the disease.

Discussion

22. There is no dispute as to the first three of the factors mentioned at para 21 above, but for the reasons that follow (which are substantially the same as those given by the judge and supported by Mr Platt QC), I do not accept factors (iv) to (vi).

23. There are three ways of establishing causation in disease cases. The first is by showing that *but for* the defendant's negligence, the claimant would not have suffered the disease. Secondly, where the disease is caused by the cumulative effect of an agency part of which is attributable to breach of duty on the part of the defendant and part of which involves no breach of duty, the defendant will be liable on the ground that his breach of duty made a "material contribution" to the disease: *Bonnington Castings Ltd v Wardlaw* [1956] AC 613. The disease in that case was pneumoconiosis which is a divisible disease (i.e. one whose severity increases with increased exposure to the agency). Thirdly, where causation cannot be proved in either of these ways, for example because the disease is indivisible, causation may be established if it is proved that the defendant materially increased the risk of the victim contracting the disease: the *Fairchild* exception. Mesothelioma is an indivisible disease.

24. Mr Allan accepted that causation could not be established on the basis of the "but for" test. Indeed, Dr Rudd conceded in cross-examination that, if the deceased had not been employed by the fourth defendant (which had contributed 2.5% of the total exposure), he would still probably have developed lung cancer.

25. Mr Allan's case is not that the exposure attributable to each defendant made a material contribution to the *risk* that Mr Heneghan would contract lung cancer. It is that the exposure attributable to each defendant contributed to the *disease* itself. In short, he says that this is a *Bonnington Castings* case. In *Bonnington Castings*, a steel dresser was exposed in his employment to silica dust emanating from the pneumatic hammer at which he worked and also from swing grinders. The defendant employer was not liable for the dust emanating from the hammer, because no dust extraction plant was known or practicable. But the employer was liable for the dust which emanated from the swing grinders. Lord Reid said at page 621:

> "The medical evidence was that pneumoconiosis is caused by a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years. That means, I think, that the disease is caused by the whole of the noxious material inhaled and, if that material comes from two sources, it cannot be wholly attributed to material from one source or the other. I am in agreement with much of the Lord President's opinion in this case, but I cannot agree that the question is: which was the most probable source of the respondent's disease, the dust from the pneumatic hammers or the dust from the swing grinders? It appears to me *that the source of his disease was the dust from both sources*, and the real question is whether the dust from the swing grinders materially contributed to the disease." (emphasis added)

26. Lord Reid concluded at page 623 that:

> "It is proved not only that the swing grinders may well have contributed but that they did in fact contribute a quota of silica dust which was not negligible to the pursuer's lungs and therefore did help to produce the disease."

27. It was not necessary to rely on statistical evidence in that case to demonstrate that dust emanating from the swing grinders contributed to the disease. It contributed to the disease because its severity was proportionate to the amount of dust inhaled and the amount attributable to the swing grinders was material.

28. The *Helsinki Criteria for Diagnosis and Attribution* (1997) have played a part in the argument before us. It states at page 314 that the relative risk of lung cancer is estimated to increase 0.5% to 4% for each fibre/ml year of cumulative exposure. With the use of the upper boundary of this range, a cumulative exposure of 25 fibre/ml years is estimated to increase the risk of lung cancer two-fold. It continues:

> "Estimates of the relative risk for asbestos-associated lung cancer are based on different-sized populations. Because of the high incidence of lung cancer in the general population, it is not

> possible to prove in precise deterministic terms that asbestos is the causative factor for an *individual* patient, even when asbestosis is present. However, attribution of causation requires *reasonable* medical certainty on a probability basis that the agent (asbestos) has caused or contributed materially to the disease. The likelihood that asbestos exposure has made a substantial contribution increases when the exposure increases. Cumulative exposure, on a probability basis, should thus be considered the main criterion for the attribution of a substantial contribution by asbestos to lung cancer risk. For example, relative risk is roughly doubled for cohorts exposed to asbestos fibers at a cumulative exposure of 25 fiber-years or with an equivalent occupational history, at which level asbestosis may or may not be present or detectable. Heavy exposure, in the absence of radiologically diagnosed asbestosis, is sufficient to increase the risk of lung cancer. Cumulative exposures below 25 fiber-years are also associated with an increased risk of lung cancer, but to a lesser extent."

29. The judge described the microbiological processes that lead to lung cancer as "inscrutable". In a malignant cancerous cell, changes in the DNA of the cell cause its behaviour to change. Dr Moore-Gillon describes in his report how there is uncontrolled and often rapid cell division; there are changes in cell structure and function; the cells may invade between the cells of adjacent tissues and organs; there can be distant spread via blood or lymphatic systems so that "seeding" takes place elsewhere and new foci of cancer can develop. He explains that for a cell to begin to behave with these characteristics, it is necessary for it to have acquired a series of mutations. The number is not known with certainty and probably varies from cancer to cancer. Even when a cell in a bronchus does acquire the necessary set of mutations, this does not mean that a clinically significant cancer is inevitable. This is because of the defences afforded by the body's immune system.

30. The judge found as a fact that the percentages of asbestos dust attributable to each of the defendants were insufficient individually to enable him to conclude that they had contributed to the lung cancer. Such causation was not proved by medical evidence. And the statistical or epidemiological evidence, falling short of demonstrating a doubling of the risk, did not overcome this shortcoming in the evidence. This was the finding that he made at para 30 and repeated at para 63: see paras 14 and 17 above.

31. As Mr Platt points out, there was no specific evidence of individual causation directed to individual defendants. Dr Rudd accepted (p 3 of his report) that the current understanding of biological mechanisms "does not form a basis for the practical attribution and apportionment of causation of particular cancers. This still rests upon epidemiological evidence and theories about biological mechanisms should not be used to undermine conclusions based upon epidemiological evidence". He made the point very clearly during his re-examination:

> "Where we have a process which is essentially random, a series of accidents, a stochastic process, it is never going to be appropriate to have a deterministic model of causation. You

> are never going to say this employer contributed to this fibre which had that effect on that cell."

32. In my view, the judge was right to find support for his conclusion in the decision of the High Court of Australia in *Amaca Pty Ltd v Ellis* [2010] HCA 5. The deceased died from lung cancer. During his working life, he had been exposed to respirable asbestos fibres with two employers. He also smoked cigarettes. It is pertinent to note that the *Fairchild* exception has not been recognised by Australian law. The case proceeded on the basis that no medical evidence could say why the deceased had developed lung cancer. The central question was whether it had nevertheless been proved that it was more probable than not that the exposure to asbestos attributable to either defendant had been a cause of the lung cancer. The plaintiff relied on epidemiological evidence as the basis for an inference that the lung cancer had been caused by the fibres attributable to both defendants. All the witnesses agreed that the risk of contracting cancer from smoking was many times greater than the risk from inhalation of asbestos.

33. Between paras 51 and 65 of the judgment of the Full Court, the judges gave their reasons for concluding that causation was not established. The epidemiological evidence did not permit an inference of causation to be drawn unless it could be shown that the particular case under consideration should be treated as conforming to the pattern described by the epidemiological studies. The court held that, even if the risks and probabilities derived from the studies could be treated as revealing what was the probable explanation of what caused the cancer, they demonstrated that the probability of the deceased developing cancer if exposed to asbestos was much lower than the probability of his developing the disease from smoking. In other words, the answer to what I have referred to as the first question (the "what" question) was that it was smoking, not asbestos. That was fatal to the claim.

34. But the court went on to consider the second question (the "who" question). The plaintiff sought to prove the case against both defendants on the basis of the *Bonnington Castings* material contribution principle. He relied on that decision in a way similar to the way in which Mr Allan relies on it in the present case. The court distinguished *Bonnington Castings* at para 68:

> "This description of the issue of causation in *Bonnington Castings* shows how different it is from the issue of causation in this case. The issue in *Bonnington Castings* was whether one source of an injurious substance contributed to a gradual accumulation of dust that resulted in disease. The issue here is whether one substance that *can* cause injury *did* cause injury. Or, to adopt and adapt what Starke J said in *Adelaide Stevedoring Co Ltd v Forst*, was Mr Cotton's cancer "intimately connected with and contributed to" by his exposure to asbestos? Questions of material contribution arise only if a connection between Mr Cotton's inhaling asbestos and his developing cancer was established. Knowing that inhaling asbestos *can* cause cancer does not entail that in this case it probably *did.* For the reasons given earlier, that inference was not to be drawn in this case. Questions of what is a material contribution do not arise. "

35. Like the judge, I agree with this reasoning. Like the judge, I have also derived assistance from the judgment of Swift J in *Jones v Secretary of State for Energy and Climate Change* [2012] EWHC 2936 (QB). This case is referred to as *Phurnacite* because it concerned eight claimants who had all worked at the Abercwmboi Phurnacite Works in South Wales which produced a smokeless fuel with the trade name "Phurnacite". They claimed damages for a range of conditions including lung cancer and bladder cancer. They alleged that their conditions had been caused by their exposure to two carcinogens which are present in coal tar pitch. The three lead claimants with claims for lung cancer were exposed to carcinogens both from their occupational exposure at the Phurnacite Plant and as a result of their smoking. In addition, each of them would have been subject to other factors such as environmental exposure to carcinogens. The judge said (para 8.50) that all these factors were likely to have played some part in the carcinogenic processes going on in the claimants' bodies, processes which were, by their nature, random.

36. The battle lines in that case were similar to those in the present case. As the judge recorded at para 8.13 of her judgment, Dr Rudd (for the claimants) said (as in the present case) that in any individual case where a victim develops lung cancer after exposure to two sources of asbestos, both exposures will on the balance of probabilities have contributed materially to the carcinogenic process which resulted in the development of the cancer. Every exposure to a carcinogen will play a part in the carcinogenic process going on in an individual's body. Dr Rudd accepted that, in any individual case, it was not possible to say what had "caused" the ultimate step that had resulted in the formation of the malignant cell leading to the cancer. At para 8.21, the judge recorded that Dr Rudd had said that, if he had been asked the same questions now as he had been asked during the *Fairchild* case, he would have said that it was probable that the asbestos fibres from each source had contributed to the carcinogenic process. This would mean that there was no need for the *Fairchild* exception at all: in a claim arising from mesothelioma, the claimant should succeed 100% on the basis of the material contribution to damage principle.

37. Professor Jones (the defendants' expert) took the same view as Dr Moore-Gillon in the present case: he accepted that both exposures would have contributed to the *risk* of a lung cancer developing. But he did not consider that it was possible to determine whether one, both or neither of the exposures had actually been involved in the causal sequence of the formation of an individual cancer.

38. The whole of the judge's discussion and conclusions between para 8.49 and para 8.65 merits careful reading. The following summary will suffice. Having referred to the agreed medical evidence (similar to the agreed medical evidence in the present case), she said that it was "not possible to say, in relation to any individual cancer, which factor or factors have caused or contributed to its development" (para 8.52).

39. She questioned whether the term "cumulative" could really be applied to the part played by the claimants' occupational exposure to carcinogens when taken in conjunction with their exposure to carcinogens contained in cigarette smoke and other possible factors. In respect of a cancer suffered by any individual claimant, the occupational exposure might or might not have contributed. It was a very different situation from *Bonnington Castings,* where the additional dust exposure caused by the defendant's breach of duty did have a cumulative effect by adding to the total dust exposure. It was also different from *Bailey v Ministry of Defence* [2009] 1 WLR 1052

where there was clear *medical* evidence that the defendant's negligence had added to the claimant's weakness which had in turn resulted in her cardiac arrest and consequent brain damage.

40. Swift J therefore decided that she should not approach a case of lung cancer by applying the *Bonnington Castings* principle. She concluded that the obvious alternative was the "doubling of risk" test.

41. Mr Allan does not submit that the decision in *Phurnacite* is wrong. In my view, he is right not to do so. I find Swift J's analysis compelling and I agree with the reasons she gave for holding that the *Bonnington Castings* principle could not be invoked. For present purposes, what matters is that it is based on her acceptance of agreed medical evidence as to the aetiology of lung cancer which was not materially different from the agreed medical evidence in the present case. Mr Allan seeks to distinguish *Phurnacite.* As I understand it, he relies on the fact that in *Phurnacite* it was not established whether the carcinogens resulted from smoking, occupational exposure or both. On the other hand, in the present case it has been established that the carcinogens were caused by exposure to asbestos fibres. But that does not affect the validity of Swift J's reasoning for not applying the *Bonnington Castings* test for the purposes of the present case. In my view, the judge was right to adopt the reasoning in *Phurnacite.* In short, *Bonnington Castings* cannot be pressed into service to bridge the gap left by the evidence.

42. He was right to reject the opinion of Dr Rudd that every period of exposure in fact contributed to the development of the deceased's cancer. This was not a medical opinion. It was an opinion that an inference of causation could be drawn from the epidemiological evidence. But for the reasons stated in *Phurnacite* and by the judge in this case, it was wrong to draw this inference. The epidemiological evidence permitted the contribution to the risk of cancer attributable to an individual defendant to be quantified. But it went no further than that. That was the finding made by the judge at paras 30 and 63 of his judgment. We should not interfere with it.

43. There is also support for this conclusion in the important decision of *McGhee v National Coal Board*. A workman contracted dermatitis. The issue was whether this was attributable to (i) his work in the dusty brick kilns where he worked without adequate washing facilities (for which his employer was responsible) or (ii) the effect of his cycling home caked with sweat and dust (for which his employer was not responsible). The pursuer relied on *Bonnington Castings* and argued that the first source of noxious dust (lack of adequate washing facilities) had made a material contribution to his injury. At p 4F, Lord Reid said:

> "In the present case the evidence does not show – perhaps no one knows – just how dermatitis of this type begins. It suggests to me that there are two possible ways. It may be that an accumulation of minor abrasions of the horny layer of the skin is a necessary precondition for the onset of the disease. Or it may be that the disease starts at one particular abrasion and then spreads, so that multiplication of abrasions merely increases the number of places where the disease can start and in that way increases the risk of its occurrence.

> I am inclined to think that the evidence points to the former view. But in a field where so little appears to be known with certainty I could not say that that is proved. If it were then this case would be indistinguishable from *Wardlaw's* case. But I think that in cases like this we must take a broader view of causation. The medical evidence is to the effect that the fact that the man had to cycle home caked with grime and sweat added materially to the risk that this disease might develop. It does not and could not explain just why that is so. But experience shows that it is so. Plainly that must be because what happens while the man remains unwashed can have a causative effect, though just how the cause operates is uncertain. I cannot accept the view expressed in the Inner House that once the man left the brick kiln he left behind the causes which made him liable to develop dermatitis. That seems to me quite inconsistent with a proper interpretation of the medical evidence. Nor can I accept the distinction drawn by the Lord Ordinary between materially increasing the risk that the disease will occur and making a material contribution to its occurrence.
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> There may be some logical ground for such a distinction where our knowledge of all the material factors is complete. But it has often been said that the legal concept of causation is not based on logic or philosophy. It is based on the practical way in which the ordinary man's mind works in the everyday affairs of life. From a broad and practical viewpoint I can see no substantial difference between saying that what the defender did materially increased the risk of injury to the pursuer and saying that what the defender did made a material contribution to his injury.
>
> I would therefore allow this appeal."

44. In other words, the House of Lords rejected the submission that *Bonnington Castings* should be applied in the situation that arose in that case. That was because so little was known about the aetiology of dermatitis that it was not possible to say with any certainty which source had caused or contributed to the disease. I can see no distinction between the facts of that case and those of the present case which would justify applying the *Bonnington Castings* test in the latter when the House refused to apply it in the former.

45. As was said by Lord Hoffmann in *Barker* at para 13, *McGhee* was an application avant la lettre of the *Fairchild* exception. The gap in biological evidence (Lord Bingham's "rock of uncertainty") meant that, if the conventional rules of causation were applied, the claimant would not prove his case and would recover no damages for the defendant's breach of duty at all, notwithstanding that the defendant had materially increased the risk that the claimant would suffer injury. These conventional rules included the *Bonnington Castings* material contribution test. Thus it was that the House in *McGhee* adopted what Lord Reid called a "broader view of causation". This broader view of causation was adopted as a matter of policy in order to arrive at a just solution. Mr Allan's invocation of the *Bonnington Castings* test ignores the fact that there is a fundamental difference between making a material contribution to an injury and materially increasing the risk of an injury. If the two were the same, *Fairchild* would not have been seen to be the ground-breaking decision that it was,

and the decision in *Barker* would have been difficult to understand. *Fairchild* could hardly have been described by Lord Hoffmann at para 1 in *Barker* as "an exceptional and less demanding test for the necessary causal link between the defendant's conduct and the damage" than requiring proof that the defendant did cause the damage in fact. Moreover, it is clear that the House of Lords in *Fairchild* did not proceed on the basis of the fiction that a defendant who had created a material risk of mesothelioma was deemed to have caused or materially contributed to the contraction of the disease itself: see per Lord Hoffmann at paras 31 to 33 in *Barker*. As Lord Hoffmann said at para 34, if the basis of liability is the wrongful creation of a risk or chance of causing damage, "the damage which the defendant should be regarded as having caused is the creation of such risk or chance". It seems to me that these statements are fatal to Mr Allan's submission that the creation of a material risk of injury is to be equiparated with making a material contribution to the injury. At para 40, Lord Hoffmann said that the *Fairchild* exception was created because the alternative of leaving the claimant with no remedy was thought to be unfair. If Mr Allan is right, it is difficult to see why the claimant in *Fairchild* did not succeed on the *Bonnington Castings* test.

46. Mr Allan seeks to invoke policy considerations of justice and to persuade the court to adopt a broader view of causation in this case. He says that such a view requires the application of the *Bonnington Castings* test. I do not agree. That test is to be applied where the court is satisfied on scientific evidence that the exposure for which the defendant is responsible has in fact contributed to the *injury*. This is readily demonstrated in the case of divisible injuries (such as silicosis and pneumoconiosis) whose severity is proportionate to the amount of exposure to the causative agent.

47. The response of the law to the problem posed in a case where the scientific evidence does not permit a finding that the exposure attributable to a particular defendant contributed to the injury is to apply the *Fairchild* exception. The factors identified in *Fairchild* for the application of this solution exist in the present case: (i) all the defendants concede their breach of duty; (ii) all increased the risk that the deceased would contract lung cancer; (iii) all exposed the deceased to the same agency that was implicated in causation (asbestos fibres); but (iv) medical science is unable to determine to which (if any) of the defendants there should be attributed the exposure which actually caused the cell changes which initiated the genetic changes culminating in the cancer.

48. In short, I can see no reason not to apply the *Fairchild* exception to the facts of the present case. There can be no objection in principle to extending it to situations which are not materially different from *Fairchild.* Indeed, principle requires that in a situation which is truly analogous to that considered in that case, the *Fairchild* exception should be applied. Otherwise, the law in this area would be inconsistent and incoherent.

49. There is some support for this view in the Supreme Court decision in *International Energy Group Ltd v Zurich Insurance Plc UK* [2015] UKSC 33, [2015] 2 WLR 1471. Thus, Lords Neuberger and Reed said at para 191 that the *Fairchild* exception is "applicable to any disease which has the unusual features of mesothelioma". The possibility of its application in cases concerning other injuries or diseases was also expressly contemplated by Lord Hodge (para 109) and Lord Sumption (para 127).

Conclusion

50. To summarise, Mr Allan concedes that causation cannot be established against any of the defendants on the conventional "but for" test. For the reasons that I have given, I do not accept his submission that it is possible to infer from the epidemiological evidence that all or any of the defendants made a material contribution to Mr Heneghan's contracting of lung cancer. All of the defendants did, however, materially contribute to the risk that he would contract lung cancer. The judge was, therefore, right to apply the *Fairchild* exception.

51. I would dismiss the appeal.

Lord Justice Tomlinson:

52. I agree.

Lord Justice Sales:

53. I agree that the appeal should be dismissed for the reasons given by the Master of the Rolls.

54. He points out that both parties were critical of an observation by Jay J at para 61, where the judge mentioned a feature of the case which could give rise to an interesting question in an appropriate case, but which did not arise for decision. The judge suggested that on the evidence before him it could have been said that if the appellant had sued W. Blackwell the claim against them could have been proved on the conventional balance of probabilities approach without recourse to the extended *Fairchild* approach. The judge's observations in para 61 did not form part of the critical reasoning in the case. Mr Platt QC for the respondents proposed that we should expressly disapprove what the judge said there, to provide guidance in other cases.

55. It is unnecessary for us to say anything definitive about this and it would not be appropriate to do so, since we have not heard adversarial argument on the point. It is not immediately obvious to me that the judge was wrong (albeit I was unsure what he meant by another observation in para 61, namely that his suggestion regarding W. Blackwell was not "primarily based on any epidemiology", since the arithmetic on which he based his suggestion was derived from the epidemiological evidence). I would wish to reserve any concluded opinion on the question whether one in a series of employers might be held liable on ordinary principles on the basis of epidemiological evidence showing that that employer was responsible for a doubling of the relevant risk (which the judge took to be the position of W. Blackwell) alongside other employers whose liability depended upon application of the *Fairchild* approach (as with the defendant employers in these proceedings) and, if so, what the effect might be in terms of the recoverability of damages against each of them.