Hilary Term
[2011] UKSC 10
On appeal from: [2009] EWCA Civ 1159;
[2009] EWCA Civ 1211

JUDGMENT

Sienkiewicz (Administratrix of the Estate of Enid Costello Deceased) (Respondent) *v* Greif (UK) Limited (Appellant)

Knowsley Metropolitan Borough Council (Appellant) *v* Willmore (Respondent)

before

Lord Phillips, President
Lord Rodger
Lady Hale
Lord Brown
Lord Mance
Lord Kerr
Lord Dyson

JUDGMENT GIVEN ON

9 March 2011

Heard on 26, 27 and 28 October 2010

Appellant (Greif)
Jeremy Stuart-Smith QC
Charles Feeny
(Instructed by Hill Dickinson LLP)

Respondent (Sienkiewicz)
Christopher Melton QC
Richard Pearce
(Instructed by Norman Jones Solicitors)

Appellant (Knowsley)
Jeremy Stuart-Smith QC
Charles Feeny
(Instructed by Berrymans Lace Mawer LLP)

Respondent (Willmore)
David Allan QC
Simon Kilvington
(Instructed by John Pickering & Partners LLP)

LORD PHILLIPS

Introduction

1. Mesothelioma is a hideous disease that is inevitably fatal. In most cases, indeed possibly in all cases, it is caused by the inhalation of asbestos fibres. Unusual features of the disease led the House of Lords to create a special rule governing the attribution of causation to those responsible for exposing victims to asbestos dust. This was advanced for the first time in *Fairchild v Glenhaven Funeral Services Ltd* [2002] UKHL 22; [2003] 1 AC 32 and developed in *Barker v Corus UK Ltd* [2006] UKHL 20; [2006] 2 AC 572. Parliament then intervened by section 3 of the Compensation Act 2006 further to vary this rule. The rule in its current form can be stated as follows: when a victim contracts mesothelioma each person who has, in breach of duty, been responsible for exposing the victim to a significant quantity of asbestos dust and thus creating a "material increase in risk" of the victim contracting the disease will be held to be jointly and severally liable for causing the disease.

2. These two appeals involve cases where the defendant was the sole known source of occupational exposure to asbestos dust. In each case the extent of the exposure found was very small. In each case, the Court of Appeal, applying the special rule, held the defendant liable for causing the disease.

3. In *Willmore v Knowsley Metropolitan Borough Council* the Council contends that the trial judge erred in finding that it was responsible for exposing Mrs Willmore to sufficient asbestos dust to cause a material increase in risk. The appeal involves an attack on findings of fact by the judge, which were upheld by the Court of Appeal, but no issue of principle, albeit that it nicely illustrates the effect of the special rule of causation. I agree that this appeal must be dismissed for the reasons given by Lord Rodger and I have nothing to add to these.

4. In *Sienkiewicz v Greif (UK) Ltd* ("Greif") the respondent is the daughter of Mrs Enid Costello and sues as administratrix of her estate. The appellant, Greif, raises two separate, albeit interrelated, grounds of appeal. The exposure for which the judge found Greif to have been responsible only increased the total amount of exposure which Mrs Costello would have experienced as a result of environmental exposure to asbestos, that is exposure to asbestos in the atmosphere, by 18%. Greif submits that in these circumstances the respondent failed to prove on balance of probability that Greif caused Mrs Costello's mesothelioma; to do this she would have had to prove that the exposure for which Greif was responsible had more than

doubled the environmental exposure. This submission raises the following important issue of principle. Does the special rule of causation that applies in cases of mesothelioma leave any room for applying a test of balance of probability to causation? It also raises a general issue as to the applicability as proof of causation in personal injury cases of a test usually applied to epidemiological evidence that I shall call the "doubles the risk" test. Shortly stated this test attributes causative effect to any factor that more than doubled a risk that would otherwise have been present of the injury that occurred.

5. Greif's alternative submission is that occupational exposure to asbestos dust will only constitute a *material* increase in risk for the purpose of the special rule of causation if it more than doubles the environmental exposure to such dust to which the victim was subject. It did not do so in the case of Mrs Costello.

Methods of proving causation

6. Most claims for death or personal injury result from accidents. In such cases the cause of death or personal injury will seldom be in issue. A body of knowledge which I shall call "medical science" will enable a witness, expert in that science, to describe the precise mechanism by which the accident brought about the death or injury. I shall describe this as the "biological cause" of death or injury. It is sometimes referred to by the more general description of the "cause in fact". In some cases, however, medical science will not yet have identified the precise mechanism by which an injury gives rise to a particular physical consequence. In such cases it may be possible to deduce that there was a causative link between the two by evidence of what usually happens. Epidemiological evidence that a particular injury or disease usually follows a particular type of bodily insult may enable a court to conclude in a particular case, on balance of probability, that the former was caused by the latter. "*Post hoc ergo propter hoc*". A finding of causation based on such evidence is sometimes described as "the cause in law".

7. Where the court is concerned with a speculative question – "what would have happened but for a particular intervention" it is likely to need to have regard to what normally happens. A good example of such a situation is the task of estimating the loss of expectation of life of a person whose death has been caused by negligence or breach of duty. In such a situation the evidence upon which the court will reach its conclusion is likely to be provided, at least in part, by a statistician or an epidemiologist. Medical science will identify whether the deceased had any physical characteristic relevant to his life expectancy. Epidemiology will provide statistical evidence of life expectancy of the group or cohort to which the deceased belonged. With this material the court answers the hypothetical question of the length of the life that the victim would have enjoyed but for the breach of duty of the defendant.

8. Epidemiology can also provide a court with assistance in deciding what actually happened, when the cause of a disease or injury is not clear. For one remarkable example of the use, and ultimate rejection, of epidemiological evidence see *Loveday v Renton* [1990] 1 MLR 1. Another remarkable case, to which I shall make further reference was *XYZ v Schering Health Care Ltd* [2002] EWHC 1420 (QB); 70 BMLR 88.

9. When a scientific expert gives an opinion on causation, he is likely to do so in terms of certainty or uncertainty, rather than probability. Either medical science will enable him to postulate with confidence the chain of events that occurred, ie the biological cause, or it will not. In the latter case he is unlikely to be of much assistance to the judge who seeks to ascertain what occurred on a balance of probability. This reality was expressed by Lord Prosser in *Dingley v The Chief Constable, Strathclyde Police* 1998 SC 548, 603 with a clarity that merits quotation:

> "In ordinary (non-lawyers') language, to say that one regards something as 'probable' is by no means to say that one regards it as 'established' or 'proved'. Yet in the civil courts, where we say that a pursuer must prove his case on a balance of probabilities, what is held to be probable is treated as 'proved'. I do not suggest that any lawyer will be confused by this rather special meaning of the word 'proved'. But speaking very generally, I think that the civil requirement of a pursuer – that he satisfy the court that upon the evidence his case is probably sound – would in ordinary language be regarded as very different from, and less stringent than, a requirement that his case be established or proved. More importantly in the context of such a case as the present, the fact that the two concepts are distinct in ordinary language, but the same in this legal context, seems to me to give rise to a risk of ambiguity or misunderstanding in the expressed opinions of expert witnesses. And this risk will be increased if the expert in question would normally, in the exercise of his profession, adopt an approach to such issues starkly different from that incumbent upon a court. Whether one uses the word 'scientific' or not, no hypothesis or proposition would be seen as 'proved' or 'established' by anyone with any form of medical expertise merely upon the basis that he had come to regard it as probably sound. (Indeed, I think even the word 'probable' would be reserved for situations where the likelihood is thought to be much more than marginal). And even if, in relation to any possible proposition or hypothesis, such an expert even troubled to notice that he had come to the point of regarding it as not merely possible but on balance 'probable', then I think he would regard that point as one from which he must set off on further inquiry, and by no means as

being (as it is in the courts) a point of arrival. Mere marginal probability will not much interest him. But it must satisfy a court."

10. The issue in *Dingley* was whether the development of multiple sclerosis had been caused by physical injury sustained in a motor accident. Medical science was not able to demonstrate the connection between the two, and reliance was placed on epidemiological evidence. Lord Prosser, at pp 604-605, had this to say about this method of proof:

"I am not much impressed by one argument advanced for the defender to the effect that the pursuer's argument is essentially *'post hoc, ergo propter hoc'*, and therefore unsound. Plainly, one will more readily conclude that B is caused by A, or probably caused by A, if one can identify, or at least envisage, some kind of mechanism whereby B might be caused by A. Equally, if one simply cannot identify or envisage such a mechanism, the mere fact that on one occasion B happened after A (and perhaps very quickly after A) would not, in the absence of other indications, lead one easily to conclude that B was caused by A. But no one, certainly in this case, suggests that such a single coincidence is to be interpreted as involving a causal relationship. And once one moves from single coincidence to a number of occasions when B follows (perhaps quickly) upon A, dismissiveness of *'post hoc, ergo propter hoc'* reasoning seems to me to become less and less appropriate. Indeed, unless and until one can identify or envisage a connecting mechanism, countless conclusions as to causal relationship are reached precisely upon a form of *'post hoc, ergo propter hoc'* reasoning: if B is observed never to occur except shortly after A, the conclusion may be relatively easy – but if B is observed to occur frequently after A, then even if each sometimes occurs without the other, the frequency with which B occurs after A may nonetheless well justify a more or less firm conclusion that A, in certain circumstances, causes B. I do not regard such conclusions as based on false (or indeed simple) logic. The approach is in my opinion inherent not only in conclusions drawn from one's general experience or 'anecdotal evidence'. It is inherent also in much experimental research, and also, as it seems to me, in epidemiology. And while it may always seem somewhat insufficient, until one can find an identifiable possible mechanism, as a basis for claiming that the causal link is proved or established, in either ordinary or scientific terms, that feeling of insufficiency strikes me as much less appropriate if one stops short of such claims and contents oneself with saying that the causal relationship is marginally probable (or is proved or established only as required in civil litigation)."

11. Knowledge about mesothelioma is based in part on medical science and in part on statistical analysis or epidemiology. These appeals raise the question of whether, and if so to what extent, the court can satisfactorily base conclusions about causation on the latter, both in mesothelioma cases and more generally.

Principles of causation in relation to disease

12. Many diseases are caused by the invasion of the body by an outside agent. Some diseases are caused by a single agent. Thus malaria results from a single mosquito bite. The extent of the risk of getting malaria will depend upon the quantity of malarial mosquitoes to which the individual is exposed, but this factor will not affect the manner in which the disease is contracted nor the severity of the disease once it is contracted. The disease has a single, uniform, trigger and is indivisible.

13. The contraction of other diseases can be dose related. Ingestion of the agent that causes the disease operates cumulatively so that, after a threshold is passed, it causes the onset of the disease. Lung cancer caused by smoking is an example of such a disease, where the disease itself is indivisible. The severity of the disease, once it has been initiated, is not related to the degree of exposure to cigarette smoke.

14. More commonly, diseases where the contraction is dose related are divisible. The agent ingested operates cumulatively first to cause the disease and then to progress the disease. Thus the severity of the disease is related to the quantity of the agent that is ingested. Asbestosis and silicosis are examples of such diseases, as are the conditions of vibration white finger and industrial deafness, although the insults to the body that cause these conditions are not noxious agents. For this reason it is important to distinguish between asbestosis and mesothelioma when considering principles of causation.

15. Mesothelioma is an indivisible disease. As I shall explain there is uncertainty as to whether its contraction is related to the amount of asbestos fibres ingested.

16. It is a basic principle of the law of tort that the claimant will only have a cause of action if he can prove, on balance of probabilities, that the defendant's tortious conduct caused the damage in respect of which compensation is claimed. He must show that, *but for* the defendant's tortious conduct he would not have suffered the damage. This broad test of balance of probabilities means that in some cases a defendant will be held liable for damage which he did not, in fact, cause.

Equally there will be cases where the defendant escapes liability, notwithstanding that he has caused the damage, because the claimant is unable to discharge the burden of proving causation.

17. There is an important exception to the "but for" test. Where disease is caused by the cumulative effect of the inhalation of dust, 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 has made a material contribution to the disease - *Bonnington Castings Ltd v Wardlaw* [1956] AC 613. The disease in that case was pneumoconiosis. That disease is divisible. The severity of the disease depends upon the quantity of silica inhaled. The defendant did not, however, argue that, if held liable, this should only be to the extent that the dust for which it was responsible had contributed to the plaintiff's symptoms. It was held liable for 100% of the disease. There have, however, been a series of cases at first instance and in the Court of Appeal in which it has been recognised that where there has been a number of exposures of a claimant to bodily insults that have cumulatively caused a divisible disease, responsibility should be apportioned so that an individual defendant is liable for no more than his share of the disease. This apportionment may necessarily be a rough and ready exercise: see Mustill J's analysis in *Thompson v Smiths Shiprepairers (North Shields) Ltd* [1984] QB 405 at pp 437-444 and the cases cited in *McGregor on Damages*, 18th ed (2010) at 8-010 to 8-016

What is known about mesothelioma

18. The special rule of causation applied to mesothelioma was devised because of ignorance about the biological cause of the disease. It was accepted in *Fairchild* and *Barker* that this rendered it impossible for a claimant to prove causation according to the conventional "but for" test and this caused injustice to claimants. It is not possible properly to consider the issues raised by this appeal without reference to what is known about mesothelioma. This has been summarised in many cases, and much of my own summary in *Bryce v Swan Hunter Group plc* [1988] 1 All ER 659 of what was known 25 years ago remains true today. The cases under appeal did not involve the introduction of detailed evidence of what is known today about mesothelioma, proceeding on the basis that findings in previous cases could be taken as read. There was, however, introduced in evidence a case control study by Peto and Rake, published in 2009 by the Health and Safety Executive, on "Occupational, Domestic and Environmental Mesothelioma risks in Britain" ("the Peto Report"), which is said to be the first representative study to quantify the relationship between mesothelioma risk and lifetime occupational and residential history in this country.

19. In these circumstances I have turned to recent judicial authority in order to augment the information contained in Peto and Rake's study. It has not been necessary to look further than the collation of data about mesothelioma set out by Rix LJ in his judgment in the series of appeals collectively described as *Employers' Liability Insurance "Trigger" Litigation* [2010] EWCA Civ 1096. I shall set out in an annex to this judgment details of the current knowledge about mesothelioma that I have obtained from these sources. I can summarise the effect of the material in the Annex as follows:

i) Mesothelioma is always, or almost always, caused by the inhalation of asbestos fibres.

ii) A significant proportion of those who contract mesothelioma have no record of occupational exposure to asbestos. The likelihood is that in their case the disease results from inhalation of asbestos dust that is in the environment. There is, however, a possibility that some cases of mesothelioma are "idiopathic", ie attributable to an unknown cause other than asbestos. Mr Stuart-Smith QC for Greif submitted that the Peto Report indicates that this is more than a possibility, but I do not so read it. I do not, however, think that it matters whether some cases of the disease are idiopathic.

iii) The more fibres that are inhaled, the greater the risk of contracting mesothelioma.

iv) There is usually a very long period between the exposure to asbestos and the development of the first malignant cell. Typically this can be at least 30 years.

v) There will be a lengthy period between the development of the first malignant cell and the point at which the disease can be diagnosed. At the time of *Fairchild* this was thought to be 10 years, but is now thought to be at least 5 years. During this period, further exposure to asbestos fibres will have no causative effect.

vi) The mechanism by which asbestos fibres cause mesothelioma is still not fully understood. It is believed that a cell has to go through 6 or 7 genetic mutations before it becomes malignant, and asbestos fibres may have causative effect on each of these.

vii) It is also possible that asbestos fibres have a causative effect by inhibiting the activity of natural killer cells that would otherwise destroy a mutating cell before it reaches the stage of becoming malignant.

20. These propositions are based in part on medical science and in part on epidemiological studies. They represent the current understanding of a disease about which much remains to be discovered.

The development of the special rule of causation that applies to mesothelioma

21. The starting point in tracing the development of the special rule of causation is the decision of the House of Lords in *McGhee v National Coal Board* [1973] 1 WLR 1, an appeal from the First Division of the Court of Session. The pursuer claimed against his employers for dermatitis which he alleged had been caused by breaches of their common law duties. He was employed in a brick kiln, where he got covered in brick dust. This, so it was held, involved no breach of duty on the part of the defenders. They were, however, held in breach of duty for failure to provide a shower which would have enabled him to wash off the dust as soon as he finished work. He had to cycle home covered in dust and sweat. Two medical experts were called. The effect of their evidence was that the brick dust caused the dermatitis but that the precise mechanism whereby it did so was not known. It was agreed, however, that the risk of contracting the disease would have been reduced had the pursuer been able to wash off the dust before he cycled home. The cycle ride home in his unwashed state increased his risk of getting dermatitis.

22. Lord Reid at p 4 summarised this evidence as follows:

> "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."

23. The nature of the evidence of that experience is not apparent. It does not appear to have been based on epidemiological research. Neither witness was able to quantify the extent to which failure to wash increased the risk, and one said that it was not possible to do so. If epidemiological data had existed it might have been possible to make a quantitative assessment based upon it of the extent to which delay in washing off brick dust increased the risk of dermatitis.

24. On the very limited evidence available it was possible that the dermatitis had already been triggered by the time that the pursuer stopped work. It is equally possible that the additional exposure while he cycled home caked in dust had a critical incremental effect in triggering the disease. The defenders' failure to provide showers increased the hazard posed to their workforce by brick dust and it was impossible on the evidence to determine whether this increase in hazard was

or was not the critical factor in causing the pursuer's dermatitis. Thus the facts of *McGhee* were not on all fours with those of *Bonnington.* In *Bonnington* it was possible to say that the inhalation of the silica attributable to breach of duty had contributed to causing the plaintiff's pneumoconiosis. In *McGhee* it was not possible to say whether or not the lack of a shower had in fact contributed to the contraction of the dermatitis.

25. I have not found it possible to identify in *McGhee* reasoning that is common to all members of the House. The analysis of the decision that is now generally accepted is that the majority of their Lordships adapted the approach in *Bonnington* to the facts of *McGhee* by equating contribution to the risk of contracting dermatitis with contribution to the disease itself. They did so in circumstances where it was impossible to say whether, on balance of probability, the absence of shower facilities had been critical. What four of their Lordships did not consider was what the position would have been if there had been epidemiological evidence that gave a negative answer to that question. Lord Salmon did, however, expressly confront this question at p 12. After observing that the expert evidence did not enable one to place a percentage figure on the extent to which the lack of shower facilities had increased the risk of contracting dermatitis, he added:

> "It is known that some factors materially increase the risk and others materially decrease it. Some no doubt are peripheral. Suppose, however, it were otherwise and it could be proved that men engaged in a particular industrial process would be exposed to a 52 per cent risk of contracting dermatitis even when proper washing facilities were provided. Suppose it could also be proved that that risk would be increased to, say, 90 per cent when such facilities were not provided. It would follow that if the decision appealed from is right, an employer who negligently failed to provide the proper facilities would escape from any liability to an employee who contracted dermatitis notwithstanding that the employers had increased the risk from 52 per cent to 90 per cent. The negligence would not be a cause of the dermatitis because even with proper washing facilities, ie without the negligence, it would still have been more likely than not that the employee would have contracted the disease – the risk of injury then being 52 per cent. If, however, you substitute 48 per cent for 52 per cent the employer could not escape liability, not even if he had increased the risk to, say, only 60 per cent. Clearly such results would not make sense; nor would they, in my view, accord with the common law."

26. In the example given by Lord Salmon the lack of shower facilities did not quite double the risk of contracting dermatitis. Thus, if one applies the "doubles

the risk" test, the lack of shower facilities could not be shown to be the cause of any individual workman's dermatitis. I can understand why Lord Salmon considered that to base a finding of causation on such evidence would be capricious, but not why he considered that to do so would be contrary to common law. The balance of probabilities test is one that is inherently capable of producing capricious results. Nor do I understand his cryptic comment:

> "I think that the approach by the courts below confuses the balance of probability test with the nature of causation".

The "doubles the risk" test is one that, as I shall show in due course, has been adopted in subsequent cases as a valid method of deciding causation on balance of probabilities, and one which Mr Stuart-Smith has sought to apply on these appeals.

27. In *Hotson v East Berkshire Area Health Authority* [1987] AC 750 causation again caused a problem. The plaintiff, aged 13, had fallen out of a tree and sustained injury which reduced the flow of blood to cartilage in his hip joint. In breach of duty the defendants failed to diagnose this for five days. He suffered permanent disability of the hip joint. The issue was whether the injury itself was so severe that the subsequent disability of the hip joint was inevitable or whether, but for the five day delay, it would have been possible to prevent that disability. The medical evidence was that there was a 75% likelihood that the former was the case, but a 25% possibility that the delay in treatment was critical. At first instance [1985] 1 WLR 1036 Simon Brown J held that the defendant's breach of duty had robbed the plaintiff of a 25% chance of avoiding the disability. The House of Lords held that this analysis was erroneous. The plaintiff was not robbed of a chance of avoiding the disability. The die was cast as soon as he had sustained his injury. Either the disability was inevitable or it could, with due skill and care, have been avoided. On balance of probability, estimated at 75/25, the former was the position, so the plaintiff had failed to prove causation.

28. The particular interest of *Hotson* in the present context is the consideration given by Lord Mackay of Clashfern to *McGhee*, a case in which he had appeared as leading counsel for the employers. Like Lord Salmon, he took an epidemiological example. He said, at p 786:

> "In *McGhee v National Coal Board* [1973] 1 WLR 1 this House held that where it was proved that the failure to provide washing facilities for the pursuer at the end of his shift had materially increased the risk that he would contract dermatitis it was proper to hold that the failure to provide such facilities was a cause to a material extent of his contracting dermatitis and thus entitled him to damages from his

> employers for their negligent failure measured by his loss resulting from dermatitis. Material increase of the risk of contraction of dermatitis is equivalent to material decrease in the chance of escaping dermatitis. Although no precise figures could be given in that case for the purpose of illustration and comparison with this case one might, for example, say that it was established that of 100 people working under the same conditions as the pursuer and without facilities for washing at the end of their shift 70 contracted dermatitis: of 100 people working in the same conditions as the pursuer when washing facilities were provided for them at the end of the shift 30 contracted dermatitis. Assuming nothing more were known about the matter than that, the decision of this House may be taken as holding that in the circumstances of that case it was reasonable to infer that there was a relationship between contraction of dermatitis in these conditions and the absence of washing facilities and therefore it was reasonable to hold that absence of washing facilities was likely to have made a material contribution to the causation of the dermatitis."

29. In contrast to Lord Salmon's figures, Lord Mackay's figures demonstrate that, statistically, the lack of washing facilities more than doubled the risk of contracting dermatitis. Had evidence supporting such figures been given, it would have enabled the House, by application of the "doubles the risk" test, to conclude that the lack of shower facilities had, on balance of probabilities, caused Mr McGhee to contract dermatitis. I do not at this stage comment on whether or not it would have been appropriate for the House to apply that test.

30. Lord Mackay went on to comment:

> "Although neither party in the present appeal placed particular reliance on the decision in *McGhee* since it was recognised that *McGhee* is far removed on its facts from the circumstances of the present appeal your Lordships were also informed that cases are likely soon to come before the House in which the decision in *McGhee* will be subjected to close analysis. Obviously in approaching the matter on the basis adopted in *McGhee* much will depend on what is know of the reasons for the differences in the figures which I have used to illustrate the position. In these circumstances I think it unwise to do more than say that unless and until this House departs from the decision in *McGhee* your Lordships cannot affirm the proposition that in no circumstances can evidence of loss of a chance resulting from the breach of a duty of care found a successful claim of damages, although there was no suggestion that the House regarded such a chance as an asset in any sense."

Once again I find this comment cryptic. Lord Mackay seems to be treating epidemiological evidence as evidence of "loss of a chance", but it is not clear whether he is suggesting that such evidence might lead to a partial recovery rather than a full recovery in a case such as *McGhee.*

31. The next step in the story is *Wilsher v Essex Area Health Authority* [1988] AC 1074. A baby boy, born three months prematurely, developed a condition of the retina which rendered him blind. There were five possible causes of the condition. One was the negligent administration of an excessive quantity of oxygen. The other four involved no fault on the part of the defendant's medical staff. The expert witnesses were unable to say which caused the disease. The Court of Appeal, purporting to apply the principle in *McGhee*, held in favour of the infant. Mustill LJ expressed the principle, as he understood it, as follows [1987] QB 730, 771-772:

> "If it is an established fact that conduct of a particular kind creates a risk that injury will be caused to another or increases an existing risk that injury will ensue; and if the two parties stand in such a relationship that the one party owes a duty not to conduct himself in that way; and if the first party does conduct himself in that way; and if the other party does suffer injury of the kind to which the risk related; then the first party is taken to have caused the injury by his breach of duty, even though the existence and extent of the contribution made by the breach cannot be ascertained."

32. This analysis of *McGhee* was principled and coherent, but it was of wide general application and fundamentally rewrote the law of causation. It opened the floodgates and, I suspect, this may, in part, be the reason why it was subsequently rejected.

33. In *Wilsher,* Sir Nicolas Browne-Wilkinson V-C, dissenting in the Court of Appeal at p 779, did not accept Mustill LJ's analysis:

> "To apply the principle in *McGhee v National Coal Board* [1973] 1 WLR 1 to the present case would constitute an extension of that principle. In the *McGhee* case there was no doubt that the pursuer's dermatitis was physically caused by brick dust: the only question was whether the continued presence of such brick dust on the pursuer's skin after the time when he should have been provided with a shower caused or materially contributed to the dermatitis which he contracted. There was only one possible agent which could have caused the dermatitis, viz, brick dust, and there was no doubt

> that the dermatitis from which he suffered was caused by that brick dust. In the present case the question is different. There are a number of different agents which could have caused the RLF. Excess oxygen was one of them. The defendants failed to take reasonable precautions to prevent one of the possible causative agents (eg excess oxygen) from causing RLF. But no one can tell in this case whether excess oxygen did or did not cause or contribute to the RLF suffered by the plaintiff. The plaintiff's RLF may have been caused by some completely different agent or agents, eg hypercarbia, intraventricular haemorrhage, apnoea or patent ductus arteriosus. In addition to oxygen, each of those conditions has been implicated as a possible cause of RLF. This baby suffered from each of those conditions at various times in the first two months of his life. There is no satisfactory evidence that excess oxygen is more likely than any of those other four candidates to have caused RLF in this baby. To my mind, the occurrence of RLF following a failure to take a necessary precaution to prevent excess oxygen causing RLF provides no evidence and raises no presumption that it was excess oxygen rather than one or more of the four other possible agents which caused or contributed to RLF in this case. The position, to my mind, is wholly different from that in the *McGhee* case [1973] 1 WLR 1, where there was only one candidate (brick dust) which could have caused the dermatitis, and the failure to take a precaution against brick dust causing dermatitis was followed by dermatitis caused by brick dust. In such a case, I can see the common sense, if not the logic, of holding that, in the absence of any other evidence, the failure to take the precaution caused or contributed to the dermatitis. To the extent that certain members of the House of Lords decided the question on inferences from evidence or presumptions, I do not consider that the present case falls within their reasoning. A failure to take preventative measures against one out of five possible causes is no evidence as to which of those five caused the injury."

34. In the House of Lords, Lord Bridge of Harwich, reversing, with the agreement of the other members of the House, the decision of the Court of Appeal, approved the Vice-Chancellor's analysis. He went on to hold at p 1090 that *McGhee*

> "laid down no new principle of law whatever. On the contrary, it affirmed the principle that the onus of proving causation lies on the pursuer or plaintiff. Adopting a robust and pragmatic approach to the undisputed primary facts of the case, the majority concluded that it was a legitimate inference of fact that the defenders' negligence had materially contributed to the pursuer's injury. The decision, in my

> opinion, is of no greater significance than that and to attempt to extract from it some esoteric principle which in some way modifies, as a matter of law, the nature of the burden of proof of causation which a plaintiff or pursuer must discharge once he has established a relevant breach of duty is a fruitless one."

This analysis of *McGhee* has fared no better than that of Mustill LJ, cited at para 31.

35. *Bryce v Swan Hunter Group plc* [1988] 1 All ER 659 was heard after the decision of the Court of Appeal and before the decision of the House of Lords in *Wilsher*. It was a claim in respect of mesothelioma against three defendants who, as successive employers, had tortiously exposed the plaintiff to asbestos dust. He had had other exposure to this less than 10 years before the onset of the disease and those responsible for this had not been joined as defendants. *McGhee*, as explained by Mustill LJ in *Wilsher*, was applied, resulting in a finding that each of the defendants was liable. I understand that after this decision insurers of employers who had consecutively subjected workmen to asbestos dust tended to accept joint and several liability for mesothelioma and to agree apportionment. At all events, this Court was not referred to any decision where such an approach was challenged until *Fairchild.*

36. *Fairchild* involved three separate mesothelioma claims, which had been heard together by the Court of Appeal [2002] 1 WLR 1052. In each case the victim had been employed by a series of employers, each of which had wrongly exposed him to asbestos dust. No attempt had been made to prove, by epidemiology or otherwise, that on balance of probabilities, any particular employer had caused the victim to contract the disease. The Court of Appeal ruled against each claim on the ground that it had not been shown on balance of probability that any defendant had caused the disease. Reliance on *McGhee* was rejected on the ground that Lord Bridge in *Wilsher* had held that it established no new principle of causation and that, in *McGhee*, there had been no doubt that the defendant had caused the dermatitis – the only question was whether the defendant had done so in breach of duty. If *McGhee* was applied in the *Fairchild* situation there was a risk that a defendant would be held liable for a disease that it had not caused at all.

37. The House of Lords reversed the Court of Appeal, holding that the principle in *McGhee* was applicable. Lord Bingham at paras 21 and 22 held that Lord Bridge had been wrong in *Wilsher* to hold that *McGhee* represented no more than a robust finding of fact that the defender's negligence had materially contributed to the pursuer's injury. The opinions of at least the majority in *McGhee* could not be read as decisions of fact or orthodox applications of settled law. The House had adapted (rather than adopted) the orthodox test to meet the problem of proving

causation that had arisen on the facts of that case. Lord Nicholls of Birkenhead put the matter this way at para 44:

> "Given the medical evidence in *McGhee*, it was not open to the House, however robustly inclined, to draw an inference that the employer's negligence had in fact caused or materially contributed to the onset of the dermatitis in the sense that, but for that negligence, the dermatitis would not have occurred. Instead, a less stringent causal connection was regarded as sufficient. It was enough that the employer had materially increased the risk of harm to the employee."

38. There is room for debate, and there has been debate, as to the precise basis upon which the House in *Fairchild* applied the *McGhee* principle to the mesothelioma claims under consideration. I do not propose to enter that debate, for it was overtaken by the decision of the House in *Barker*. At this point it suffices to note the following.

39. The House was agreed that the application of the *McGhee* principle was circumscribed by a number of conditions, though not agreed as to what these were. Lord Bingham at para 2 identified 6 relevant factors that applied to the cases under consideration, before going on to hold that they brought into play the *McGhee* principle:

> "(1) C was employed at different times and for differing periods by both A and B, and (2) A and B were both subject to a duty to take reasonable care or to take all practicable measures to prevent C inhaling asbestos dust because of the known risk that asbestos dust (if inhaled) might cause a mesothelioma, and (3) both A and B were in breach of that duty in relation to C during the periods of C's employment by each of them with the result that during both periods C inhaled excessive quantities of asbestos dust, and (4) C is found to be suffering from a mesothelioma, and (5) any cause of C's mesothelioma other than the inhalation of asbestos dust at work can be effectively discounted, but (6) C cannot (because of the current limits of human science) prove, on the balance of probabilities, that his mesothelioma was the result of his inhaling asbestos dust during his employment by A or during his employment by B or during his employment by A and B taken together."

40. At para 7 Lord Bingham explained the shortcomings of medical science:

> "It is not known what level of exposure to asbestos dust and fibre can be tolerated without significant risk of developing a mesothelioma, but it is known that those living in urban environments (although without occupational exposure) inhale large numbers of asbestos fibres without developing a mesothelioma. It is accepted that the risk of developing a mesothelioma increases in proportion to the quantity of asbestos dust and fibres inhaled: the greater the quantity of dust and fibre inhaled, the greater the risk. But the condition may be caused by a single fibre, or a few fibres, or many fibres: medical opinion holds none of these possibilities to be more probable than any other, and the condition once caused is not aggravated by further exposure. So if C is employed successively by A and B and is exposed to asbestos dust and fibres during each employment and develops a mesothelioma, the very strong probability is that this will have been caused by inhalation of asbestos dust containing fibres. But C could have inhaled a single fibre giving rise to his condition during employment by A, in which case his exposure by B will have had no effect on his condition; or he could have inhaled a single fibre giving rise to his condition during his employment by B, in which case his exposure by A will have had no effect on his condition; or he could have inhaled fibres during his employment by A and B which together gave rise to his condition; but medical science cannot support the suggestion that any of these possibilities is to be regarded as more probable than any other. There is 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."

41. Lord Bingham identified at para 23 the problem raised by the facts of *Fairchild* as follows:

> "The problem of attributing legal responsibility where a victim has suffered a legal wrong but cannot show which of several possible candidates (all in breach of duty) is the culprit who has caused him harm is one that has vexed jurists in many parts of the world for many years."

He justified his decision by the following policy considerations set out at para 33:

> "It can properly be said to be unjust to impose liability on a party who has not been shown, even on a balance of probabilities, to have caused the damage complained of. On the other hand, there is a strong policy argument in favour of compensating those who have

> suffered grave harm, at the expense of their employers who owed them a duty to protect them against that very harm and failed to do so, when the harm can only have been caused by breach of that duty and when science does not permit the victim accurately to attribute, as between several employers, the precise responsibility for the harm he has suffered. I am of opinion that such injustice as may be involved in imposing liability on a duty-breaking employer in these circumstances is heavily outweighed by the injustice of denying redress to a victim."

42. Lord Bingham did not expressly consider the approach to be adopted where a claimant had been exposed to asbestos dust both from employers in breach of duty and from sources that did not involve fault, or which involved fault on the part of the claimant himself. At para 34 he expressly limited the special rule of causation that he was endorsing to a situation where all six of the factors that he had identified at the start of his speech were present. At para 22 he underlined why the special rule did not apply on the facts of *Wilsher:*

> "It is plain, in my respectful opinion, that the House was right to allow the defendants' appeal in *Wilsher*, for the reasons which the Vice-Chancellor had given and which the House approved. It is one thing to treat an increase of risk as equivalent to the making of a material contribution where a single noxious agent is involved, but quite another where any one of a number of noxious agents may equally probably have caused the damage."

43. The other members of the House did not circumscribe the special rule of causation as tightly as Lord Bingham. In *McGhee* itself the causal competition had been between exposure to dust that involved no fault and exposure that involved fault on the part of the employers, a point made by Lord Rodger at para 153. He also held that Mustill LJ had illegitimately extended the special causation test in *Wilsher*. He held, at para 149:

> "Mustill LJ's extension of the approach in *McGhee* to a situation where there were all kinds of other possible causes of the plaintiff's condition, resulted in obvious injustice to the defendants. In particular, there was nothing to show that the risk which the defendants' staff had created - that the plaintiff would develop retrolental fibroplasia because of an unduly high level of oxygen - had eventuated. That being so, there was no proper basis for applying the principle in *McGhee*. As [Sir Nicolas Browne-Wilkinson V-C] decisively observed, a failure to take preventive measures against one of five possible causes was no evidence as to

which of those five had caused the injury. The reasoning of the Vice-Chancellor, which the House adopted, provided a sound and satisfactory basis for distinguishing *McGhee* and for allowing the appeal."

44. Lord Rodger set out his conclusions at para 170:

"First, the principle is designed to resolve the difficulty that arises where it is inherently impossible for the claimant to prove exactly how his injury was caused. It applies, therefore, where the claimant has proved all that he possibly can, but the causal link could only ever be established by scientific investigation and the current state of the relevant science leaves it uncertain exactly how the injury was caused and, so, who caused it. *McGhee* and the present cases are examples. Secondly, part of the underlying rationale of the principle is that the defendant's wrongdoing has materially increased the risk that the claimant will suffer injury. It is therefore essential not just that the defendant's conduct created a material risk of injury to a class of persons but that it actually created a material risk of injury to the claimant himself. Thirdly, it follows that the defendant's conduct must have been capable of causing the claimant's injury. Fourthly, the claimant must prove that his injury was caused by the eventuation of the kind of risk created by the defendant's wrongdoing. In *McGhee*, for instance, the risk created by the defenders' failure was that the pursuer would develop dermatitis due to brick dust on his skin and he proved that he had developed dermatitis due to brick dust on his skin. By contrast, the principle does not apply where the claimant has merely proved that his injury could have been caused by a number of different events, only one of which is the eventuation of the risk created by the defendant's wrongful act or omission. *Wilsher* is an example. Fifthly, this will usually mean that the claimant must prove that his injury was caused, if not by exactly the same agency as was involved in the defendant's wrongdoing, at least by an agency that operated in substantially the same way. A possible example would be where a workman suffered injury from exposure to dusts coming from two sources, the dusts being particles of different substances each of which, however, could have caused his injury in the same way. Without having heard detailed argument on the point, I incline to the view that the principle was properly applied by the Court of Appeal in *Fitzgerald v Lane* [1987] QB 781. Sixthly, the principle applies where the other possible source of the claimant's injury is a similar wrongful act or omission of another person, but it can also apply where, as in *McGhee*, the other possible source of the injury is a similar, but

> lawful, act or omission of the same defendant. I reserve my opinion as to whether the principle applies where the other possible source of injury is a similar but lawful act or omission of someone else or a natural occurrence."

The conundrum

45. Neither Lord Bingham nor Lord Rodger explained the nature of the principle that justifies restricting the application of *McGhee* to the situation where the competing causes of the injury suffered by the claimant involve the same or a similar noxious substance or agency. There is, however, a more significant conundrum raised by *Fairchild* which is particularly relevant to this appeal. Lord Bingham observed (see para 40 above) that it is accepted that the risk of developing mesothelioma increases in proportion to the quantity of asbestos dust and fibres inhaled. If this is so why should one not determine the probability that a particular defendant caused a claimant's mesothelioma by analysing the extent to which he wrongfully contributed to the exposure of the claimant to asbestos dust and fibres? This conundrum is highlighted by the decision of the House in *Barker.*

Barker

46. The question that Lord Rodger had expressly left open at the end of his speech in *Fairchild* was raised directly in *Barker,* one of three appeals that were heard together. The claimant was the widow of a man who had died of mesothelioma. He had been exposed to asbestos dust on three occasions in his working life. Once when working for a company which had since become insolvent, once when working for the defendant and once when working for himself. On the last occasion he had failed to take reasonable precautions for his own safety. In the courts below the defendant had been held jointly and severally liable with the insolvent company, but the claimant's damages had been reduced by 20% to reflect her husband's contributory negligence. The other two appeals involved employees who had been exposed to asbestos dust by a series of employers, many of whom had since been held insolvent. In the courts below the solvent employers who had been sued were held jointly and severally liable. In each appeal the defendants argued that the special rule of causation that the House had applied in *Fairchild* should be further refined so as to render each employer liable only for that proportion of the damages which represented his contribution to the risk that the employee would contract mesothelioma. This submission was accepted by all members of the Committee with the exception of Lord Rodger, who dissented.

47. Lord Hoffmann gave the leading speech for the majority. He dealt first with the question of whether the *Fairchild* principle could apply in a case where part of the exposure was non-tortious. At para 17 he gave a positive answer to that question:

> "The purpose of the *Fairchild* exception is to provide a cause of action against a defendant who has materially increased the risk that the claimant will suffer damage and may have caused that damage, but cannot be proved to have done so because it is impossible to show, on a balance of probability, that some other exposure to the same risk may not have caused it instead. For this purpose, it should be irrelevant whether the other exposure was tortious or non-tortious, by natural causes or human agency or by the claimant himself. These distinctions may be relevant to whether and to whom responsibility can also be attributed, but from the point of view of satisfying the requirement of a sufficient causal link between the defendant's conduct and the claimant's injury, they should not matter."

48. Lord Hoffmann then turned to deal with apportionment. He did so on the premise that mesothelioma is an indivisible injury caused by a single exposure to asbestos dust. The greater the overall exposure, the greater the risk of an individual fatal exposure: see paras 2 and 26. If, under the *Fairchild* principle exposure had been treated as if it had actually contributed to the disease, the conventional approach would have resulted in all those responsible for exposure being held jointly and severally liable for the injury caused. Lord Hoffmann did not consider it fair to impose such liability on employers "in cases in which there is merely a relatively small chance" that they caused the injury (paras 43 and 46). He avoided this consequence by interpreting the *Fairchild* principle as one that rendered a defendant liable for contributing to risk, not contributing to injury. The risk created was itself the damage, albeit that the principle only applied where injury had been caused. As risk or chance was infinitely divisible, each defendant could be held liable for his contribution to the risk.

49. At the end of his consideration of the issue of causation, Lord Hoffmann made the following finding as to the limit of the *Fairchild* principle or exception:

> "24. In my opinion it is an essential condition for the operation of the exception that the impossibility of proving that the defendant caused the damage arises out of the existence of another potential causative agent which operated in the same way. It may have been different in some causally irrelevant respect, as in Lord Rodger's example of the different kinds of dust, but the mechanism by which it caused the damage, whatever it was, must have been the same. So, for example,

> I do not think that the exception applies when the claimant suffers lung cancer which may have been caused by exposure to asbestos or some other carcinogenic matter but may also have been caused by smoking and it cannot be proved which is more likely to have been the causative agent."

50. In considering how apportionment would work in practice, Lord Hoffmann said this:

> "36. Treating the creation of the risk as the damage caused by the defendant would involve having to quantify the likelihood that the damage (which is known to have materialised) was caused by that particular defendant. It will then be possible to determine the share of the damage which should be attributable to him. The quantification of chances is by no means unusual in the courts. For example, in quantifying the damage caused by an indivisible injury, such as a fractured limb, it may be necessary to quantify the chances of future complications. Sometimes the law treats the loss of a chance of a favourable outcome as compensatable damage in itself. The likelihood that the favourable outcome would have happened must then be quantified: see, for example, *Chaplin v Hicks* [1911] 2 KB 786 and *Kitchen v Royal Air Force Association* [1958] 1 WLR 563.
>
> 37.These are of course cases in which there is uncertainty as to what will be, or would have been, the outcome of a known event; for example, the consequences of a fractured ankle, a beauty contest or a lawsuit. The present case involves uncertainty as to the cause of a known outcome, namely, the mesothelioma. But in principle I can see no reason why the courts cannot quantify the chances of X having been the cause of Y just as well as the chance of Y being the outcome of X."

51. He returned to this theme under the heading of "quantification" at para 48:

> "But when the damage is apportioned among the persons responsible for the exposures to asbestos which created the risk, it is known that those exposures were together sufficient to cause the disease. The damages which would have been awarded against a defendant who had actually caused the disease must be apportioned to the defendants according to their contributions to the risk. It may be that the most practical method of apportionment will be according to the

> time of exposure for which each defendant is responsible, but allowance may have to be made for the intensity of exposure and the type of asbestos. These questions are not before the House and it is to be hoped that the parties, their insurers and advisers will devise practical and economical criteria for dealing with them."

52. These passages raise the conundrum to which I have referred in para 45 above in an acute form. If it is possible, on the basis of responsibility for exposure, to deduce the relative likelihood of a defendant being the employer who actually caused the injury, why should one not resolve liability according to the normal test of balance of probability. If one can determine that there was "a relatively small chance" that a particular employer caused the injury, why should that employer not be absolved from liability on the ground that he can prove, on balance of probability, that he was not responsible?

53. Lord Scott agreed with the reasoning and the result reached by Lord Hoffmann. He recognised, however, that the limitations on medical knowledge rendered it impossible to say whether mesothelioma was caused by a single exposure, and thus a single employer, or by a combination of more than one exposures and thus, possibly, by more than one employer: para 51. His speech also implicitly raised the conundrum. When dealing with apportionment he said this, at para 62:

> "Ascertainment of the degree of risk would be an issue of fact to be determined by the trial judge. The issue would depend upon the duration of the exposure for which each negligent defendant was responsible compared with the total duration of the claimant's exposure to the injurious agent in question. It might depend also on the intensity of the exposure for which the defendant was responsible compared with the intensity of the exposure for which the defendant was not responsible. The exact type of agent might be a relevant factor in assessing the degree of risk. I have in mind that there are different types of asbestos and some might create a greater risk than others. Other factors relevant to the degree of risk might come into the picture as well. The assessment of the percentage risk for which an individual defendant was responsible, and therefore the percentage of the total damage for which that defendant could be held liable, would, as I have said, be an issue of fact to be decided on the evidence in each case."

Why could one not assess the probability of having caused the injury on the same basis as that used to apportion contribution to the risk of causing the injury?

54. The same question is raised by the speech of Lord Walker, who also agreed with the reasoning and result reached by Lord Hoffmann. He observed, at para 109:

> "A rule of law by which exposure to risk of injury is equated with legal responsibility for that injury entails the possibility that an employer may be held liable for an injury which was not in fact caused by that exposure (though in the present state of medical science, that fact can be neither proved nor disproved). This possible unfairness cannot be eliminated, as the House recognised in *Fairchild*, but it is considerably reduced if each employer's liability is limited in proportion to the fraction of the total exposure (measured by duration and intensity) for which each is responsible."

The underlying premise of all three speeches, as of the speeches in *Fairchild*, is that it is not possible to determine causation unless medical science enables one to do so with certainty. But the law of causation does not deal in certainties; it deals in probabilities.

55. Lady Hale agreed in general with the majority, but she did not accept that the gist of the actions was the risk created rather than the mesothelioma. To that extent she shared the reasoning that had led Lord Rodger to dissent.

56. The result of the decision in *Barker* was that, where not all those who were responsible for an employee's mesothelioma were before the court, only a proportion of the relevant damages would be recoverable. This was highly significant having regard to the very long latency period of the disease, for in most cases there was a high likelihood that there would be employers who had contributed to exposure and who had gone into liquidation. Apportionment also dealt with the problem of contributory negligence. The rejoicing with which the insurance industry must have greeted this result was short lived as Parliament intervened.

The Compensation Act 2006

57. The preamble to the 2006 Act includes among its objects "to make provision about damages for mesothelioma". The relevant parts of the provision made are as follows:

> "3. *Mesothelioma: damages*

(1)This section applies where—

> (a) a person ('the responsible person') has negligently or in breach of statutory duty caused or permitted another person ('the victim') to be exposed to asbestos,
>
> (b) the victim has contracted mesothelioma as a result of exposure to asbestos,
>
> (c) because of the nature of mesothelioma and the state of medical science, it is not possible to determine with certainty whether it was the exposure mentioned in paragraph (a) or another exposure which caused the victim to become ill, and
>
> (d) the responsible person is liable in tort, by virtue of the exposure mentioned in paragraph (a), in connection with damage caused to the victim by the disease (whether by reason of having materially increased a risk or for any other reason).

(2) The responsible person shall be liable—

> (a) in respect of the whole of the damage caused to the victim by the disease (irrespective of whether the victim was also exposed to asbestos—
>
> > (i) other than by the responsible person, whether or not in circumstances in which another person has liability in tort, or
> >
> > (ii) by the responsible person in circumstances in which he has no liability in tort), and
>
> (b) jointly and severally with any other responsible person."

58. This provision has grafted onto the *Fairchild/Barker* principle a special rule in relation to liability in damages that applies only to mesothelioma. The 2006 Act, coupled with *Fairchild*, has draconian consequences for an employer who has been responsible for only a small proportion of the overall exposure of a claimant to asbestos dust, or his insurers, but it would be wrong to have regard to that fact when considering the issues raised by these appeals. Parliament has willed it so.

The facts in Greif as found by the judge

59. Mrs Costello died of mesothelioma in January 2006 at the age of 74. She had worked for Greif or their predecessors at their factory at Ellesmere Port, Cheshire, between 1966 and 1984. Greif exposed those working at that factory to

asbestos dust in breach of duty. The greatest exposure was on the factory floor, but to a much lesser extent asbestos dust permeated to other parts of the factory. Mrs Costello's exposure was in those other parts as she moved around the factory.

60. This occupational exposure was very light. The judge, His Honour Judge Main QC, heard expert evidence which quantified this exposure and compared it to the environmental exposure that would be experienced by everyone. While he held that he could "only use the broadest sorts of estimates" as to Mrs Costello's asbestos exposure he none the less based some very specific findings on this expert evidence. He held that her exposure to asbestos over her working life at Greif's factory increased the risk to which environmental exposure subjected her from 24 cases per million to 28.39 cases per million – an increase of risk of 18%.

61. It was on the basis of this finding that the judge held that the claimant's case on causation had not been made out. His starting point was that the special rule in *Fairchild* had no application where there was only one tortfeasor and where the competition as to causation was between an innocent and a tortious source of dust. In that situation he adopted an approach to causation which had been adopted, by agreement between the parties, in an earlier case on similar facts in the Cardiff County Court decided by HH Judge Hickinbottom: *Jones v Metal Box Ltd* (unreported) 11 January 2007:

> "53. (ii) It was common ground that, in order to succeed with the claim, the claimant must show that as a result of her exposure to asbestos dust at work as I have described, Mrs Jones' risk of mesothelioma at least doubled from that which it would have been without that exposure. That in my judgment is a correct analysis of the position with regard to medical causation: because unless the claimant can show that the risk was doubled, then it is more likely than not that the mesothelioma had an idiopathic rather than an occupational cause…"

Thus Judge Hickinbottom applied the "doubles the risk" test.

62. Applying that test Judge Main held:

> "On the facts of this case, the claimant could only succeed if she were able to prove that all Mrs Costello's exposure to asbestos was within the Oil Sites premises, cumulatively, over her 18 years employment exceeded her environmental risks. Here environmental risks are the same as those 'idiopathic' risks referred to by Judge

Hickinbottom. This in my judgment regrettably, she failed to do. Whilst Mrs Costello's risk of contracting mesothelioma increased by 18% the bottom line is that it was caused by her environmental exposure to asbestos. Her claim against the defendants accordingly must be dismissed."

63. In the Court of Appeal Smith LJ gave the leading judgment. In discussing the legal principles applicable she first referred to *McGhee* and *Fairchild*. She then considered the "doubles the risk" test in relation to cases of diseases other than mesothelioma. She reached the following conclusion of general principle:

"20. The theory that causation could be proved on the balance of probabilities by reference to a doubling of the risk of injury was first applied by Mackay J in the oral contraceptive litigation *XYZ v Schering Health Care Ltd* (2002) 70 BMLR 88. As a preliminary issue, the parties agreed that the judge should examine the epidemiological evidence relating to the risk of deep vein thrombosis arising from two different types of oral contraceptive. The claimant group could succeed only if the epidemiology showed that the risk of harm arising from the type of contraceptive they had been taking (which it was assumed they had not been warned about and would not have taken if warned) was at least twice that arising from the type which they had formerly been taking (which it was assumed they had been warned about and which risk they had accepted). The logic behind this was that, if the risk from potential cause A is x% and the risk from the other potential cause B is 2.1x%, it is more likely than not that the condition which has eventuated has been caused by B.

21. Since the oral contraceptive case, this method of proving causation has been applied in cases of lung cancer where the claimant has been tortiously exposed to asbestos and non-tortiously exposed to cigarette smoke, both of which are potent causes of the condition. Expert evidence is received as to the relative risks created by the two forms of exposure and, if, on the individual facts of the case, the risk from the asbestos exposure is more than double the risk from smoking, the claimant succeeds.

22. The only case of which I am aware in which this approach has been applied or approved in the Court of Appeal is *Novartis Grimsby Ltd v Cookson* [2007] EWCA Civ 1261. That was a case of bladder cancer, in which the claimant had been tortiously exposed to carcinogens in the course of his employment in a dye works. He had also been a regular smoker. Both were potential causes of bladder cancer. At trial, the defendant's case was that the tortious exposure at

> work had been minimal. The recorder held that it was not minimal and applied the *Bonnington* case [1956] AC 613*;* he held that the tort had made a material contribution to the disease. On appeal, the appellant employer argued, correctly, that that was wrong as the tort could not be said to have made a contribution to the disease, only to the risk of the disease developing. The claimant argued that the case ought to come within the *Fairchild* exception so that all that was necessary was to prove a material increase in risk. The appellant employer contended that the *Fairchild* case [2003] 1 AC 32 should not be extended to cover such a case. In the event, the Court of Appeal observed that there was expert evidence, which the recorder had accepted, to the effect that the tortious exposure had more than doubled the risk arising from smoking. The court held that that was sufficient for the claim to succeed.
>
> 23. In my view, it must now be taken that, saving the expression of a different view by the Supreme Court, in a case of multiple potential causes, a claimant can demonstrate causation by showing that the tortious exposure has at least doubled the risk arising from the non-tortious cause or causes."

64. Smith LJ went on to consider whether the "doubles the risk" test could be applied in relation to mesothelioma. She held that it could not. She did so on the basis that by enacting section 3(1)(d) of the Compensation Act 2006 Parliament had laid down a rule that causation in a mesothelioma case could be established by proof of a material increase in risk: para 34. This precluded a defendant from averring, in a case of mesothelioma, that the claimant had to satisfy the "doubles the risk" test. She held, at para 27, that the judge should have applied the test of material increase of risk, ie the *Fairchild/Barker* rule, and in consequence the appeal should be allowed:

> "it is not now possible for this court to consider whether, at common law, the *Fairchild* exception should be limited in application to cases in which it is quite impossible for a claimant to prove causation by reference to a more than twofold increase in risk. That is because Parliament has intervened by enacting section 3 of the Compensation Act 2006 so that the common law simpliciter no longer governs claims for damages in mesothelioma cases."

65. Had Smith LJ held that the "doubles the risk" test could be applied to mesothelioma, she would not have allowed the decision of the judge to stand. She held that the "doubles the risk" test had been advanced without adequate notice, so that Sienkiewicz had been wrong footed and denied a fair chance to deal with the expert evidence. The case would have to be remitted for a new trial.

66. Scott Baker LJ and Lord Clarke agreed with the judgment of Smith LJ. Lord Clarke held that the *Fairchild* test had to be applied by reason both of common law and the construction of section 3 of the 2006 Act.

Submissions

67. I can summarise the arguments advanced by Mr Stuart-Smith on behalf of Greif as follows, adopting a different order to that adopted in his printed case:

i) The Court of Appeal erred in holding that section 3 of the 2006 Act mandated the application of the *Fairchild/Barker* rule of causation in mesothelioma cases.

ii) The *Fairchild/Barker* rule does not apply in this case because this is a "single exposure" case.

iii) It is possible in this case to adopt a conventional approach to causation by applying the "doubles the risk" test. This approach demonstrates that Mrs Costello contracted mesothelioma as a result of environmental exposure and not as a result of the slight additional exposure to which she was subjected by Greif.

iv) The claim also fails because the exposure to which Greif subjected Mrs Costello was not *material*. Occupational exposure is not material unless it more than doubles the amount of environmental exposure to which a claimant is subject. In the case of Mrs Costello the exposure for which Greif was responsible was insignificant. The findings of exposure made by the trial judge could not be supported by the evidence and there was no justification for a fresh trial.

68. Mr Melton QC for Mrs Costello's estate challenged all these submissions. He submitted that the *Fairchild* test was applicable and attacked the application of the "doubles the risk" test. He further submitted that the asbestos dust to which Mrs Costello was subjected materially increased the risk that she would contract mesothelioma and that, applying the *Fairchild* test and section 3 of the 2006 Act, the Court of Appeal had properly held the claim to be made out in full.

Discussion

69. I propose to discuss the problems raised by this appeal under the following five headings:

i) The effect of section 3 of the Compensation Act 2006.

ii) Epidemiology and the nature of the "doubles the risk" test.

iii) Can the "doubles the risk" test be applied in multiple cause cases involving diseases other than mesothelioma?

iv) Can the "doubles the risk" test be applied to mesothelioma cases.

v) What constitutes a material increase in risk?

vi) The result in this case.

The effect of section 3 of the Compensation Act 2006

70. The Court of Appeal treated section 3(1) as enacting that, in cases of mesothelioma, causation can be proved by demonstrating that the defendant wrongfully "materially increased the risk" of a victim contracting mesothelioma. This was a misreading of the subsection. Section 3(1) does not state that the responsible person *will be* liable in tort if he has materially increased the risk of a victim of mesothelioma. It states that the section applies *where* the responsible person is liable in tort for materially increasing that risk. Whether and in what circumstances liability in tort attaches to one who has materially increased the risk of a victim contracting mesothelioma remains a question of common law. That law is presently contained in *Fairchild* and *Barker*. Those cases developed the common law by equating "materially increasing the risk" with "contributing to the cause" in specified and limited circumstances, which include ignorance of how causation in fact occurs. The common law is capable of further development. Thus section 3 does not preclude the common law from identifying exceptions to the "material increase of risk" test, nor from holding, as more is learned about mesothelioma, that the material increase of risk test no longer applies. The *Fairchild/Barker* rule was adopted in order to cater for the ignorance that existed at the time of those decisions about the way in which mesothelioma is caused. Section 3 does not preclude the courts from reverting to the conventional approach of balance of probabilities in mesothelioma cases should advances in medical science in relation to this disease make such a step appropriate.

71. Greif contend that the Court should identify an exception to the *Fairchild/Barker* rule where there has been only one occupational exposure to risk and that, in those circumstances, the Court can and should apply the "doubles the risk" test. Section 3 poses no bar to that contention; it must be considered on its merits.

Epidemiology and the nature of the "doubles the risk" test

72. The "doubles the risk" test is one that applies epidemiological data to determining causation on balance of probabilities in circumstances where medical science does not permit determination with certainty of how and when an injury was caused. The reasoning goes as follows. If statistical evidence indicates that the intervention of a wrongdoer more than doubled the risk that the victim would suffer the injury, then it follows that it is more likely than not that the wrongdoer caused the injury. I propose first to consider the authorities to which Smith LJ referred to see the extent to which they support the general proposition that she stated at para 23 of her judgment.

73. Smith LJ founded the general proposition in para 23 of her judgment (see para 63 above) on one decision of Mackay J, one decision of the Court of Appeal and on unspecified cases of exposure both to asbestos and to cigarette smoke. When these are examined it becomes apparent that they exemplify the application of the "doubles the risk" test in three quite different circumstances. I propose to look at these before considering the nature of the epidemiological principle applied in each of them.

74. *XYZ* is a lengthy and complex judgment devoted exclusively to a preliminary issue on the effect of epidemiological evidence. The issue was whether a second generation of oral contraceptives more than doubled the risk of causing deep vein thrombosis (DVT) that was created by the first generation of oral contraceptives. It was common ground that, if the claimants in this group litigation could not establish this, their claims under the Consumer Protection Act 1987 were doomed to failure. I do not believe that Smith LJ has correctly identified the relevance of this issue. It was not whether the DVT suffered by the claimants had been caused by the second generation of oral contraceptives which they had taken. It was whether the second generation of contraceptives created a significantly greater risk than the first. The experts appear to have been in agreement that the "doubles the risk" test was the proper one to apply in order to resolve this issue. Thus I do not believe that that decision affords any direct assistance to the question of whether the "doubles the risk" test is an appropriate test for determining causation in a case of multiple potential causes. It does, however, contain a detailed and illuminating discussion of epidemiology and I shall revert to it when considering that topic.

75. *Shortell v BICAL Construction Ltd* (Liverpool District Registry, 16 May 2008), another decision of Mackay J, was a claim in relation to a death caused by lung cancer where there were two possible causes of the cancer. One was occupational exposure to asbestos and the other was cigarette smoke. The defendant was responsible for the former but not for the latter. Applying the

Bonnington test of causation, the issue was whether the asbestos to which the victim had been exposed had made a material contribution to the cause of the victim's lung cancer. The expert evidence, given by both medical and epidemiological experts, but based in the case of each, I suspect, on epidemiological data, was that asbestos and cigarette smoke not merely combined cumulatively to cause lung cancer, but that they had a synergistic effect in doing so. This evidence was enough, as I see it, to satisfy the *Bonnington* test of causation, as the victim had been exposed both to significant quantities of asbestos fibres and to significant cigarette smoke.

76. In these circumstances, I am puzzled by the following passages in the judgment:

> "49.The causation of lung cancer as opposed to mesothelioma is dependent on an aggregate dose either of asbestos fibre or smoke. Mr Feeny for the defendants rightly in my view concedes that if the claimant proves on a balance of probabilities that the risk factor created by his client's breach of duty more than doubled the deceased's relative risk of contracting lung cancer then the claimant's case is proved, and the only remaining issue is contributory negligence. For the reasons I have advanced above I am satisfied on the balance of probabilities that once the estimate of 99 fibre/ml – years is accepted as I have accepted it the relative risk is on any view more than doubled.
>
> 51. Where, as here, it is the case that the claimant has proved causation against this defendant by showing a more than doubled relative risk it is not relevant as between the claimant and the defendant to argue that another agent (tortious or otherwise) may also have contributed to the occurrence of the disease."

Epidemiological evidence indicated that, had the victim not been a smoker, his exposure to asbestos would have more than doubled the risk that he would get lung cancer. I do not, however, see that it was essential for the claimant to prove this. For this reason I question whether Smith LJ's endorsement of the "doubles the risk" test is correct in cases where asbestos and tobacco smoke have combined to cause lung cancer.

77. *Novartis Grimsby Ltd v Cookson* [2007] EWCA Civ 1261, which was the third case to which Smith LJ referred, was an appeal in which she gave the only reasoned judgment. The claimant sought damages against his employers for causing him bladder cancer. It is known that bladder cancer is caused by exposure

to amines and the claimant had been so exposed from two sources. One was his employment, which wrongfully exposed him to amines used in the manufacture of dyes. The other was smoking, for cigarette smoke contains amines. There was expert evidence, which the recorder accepted, that the occupational exposure had more than doubled the risk caused by smoking. There was an issue as to whether *Bonnington* applied or whether the claimant had to prove that "but for" the occupational exposure he would not have suffered the cancer. Smith LJ did not find it necessary to resolve this issue, for at para 74 she held that the "but for" test was satisfied:

> "In terms of risk, if occupational exposure more than doubles the risk due to smoking, it must, as a matter of logic, be probable that the disease was caused by the former."

78. On analysis, it is only this last proposition that supports Smith LJ's general statement that a claimant can prove causation where there are a number of potential causes of a disease or injury by showing that the tortious exposure had at least doubled the risk arising from the non-tortious cause or causes. I agree with her that, as a matter of logic, if a defendant is responsible for a tortious exposure that has more than doubled the risk of the victim's disease, it follows on the balance of probability that he has caused the disease, but these are statistical probabilities and the issue in this case is whether a statistical approach to determining causation should be applied in place of the *Fairchild/Barker* test.

79. I have derived assistance in relation to the next section of this judgment from the judgment of Mackay J in *XYZ.* He there set out a careful and detailed introduction into the discipline of epidemiology and I shall gratefully borrow some of the clear language that he used.

80. Epidemiology is the study of the occurrence and distribution of events (such as disease) over human populations. It seeks to determine whether statistical associations between these events and supposed determinants can be demonstrated. Whether those associations if proved demonstrate an underlying biological causal relationship is a further and different question from the question of statistical association on which the epidemiology is initially engaged.

81. Epidemiology may be used in an attempt to establish different matters in relation to a disease. It may help to establish what agents are capable of causing a disease, for instance that both cigarette smoke and asbestos dust are capable of causing lung cancer, it may help to establish which agent or which source of an agent, was the cause, or it may help to establish whether or not one agent combined with another in causing the disease.

82. Epidemiological data can be obtained by comparing the relevant experience (eg contraction of a disease) of a group or cohort that is subject to exposure to a particular agent with the experience of a group or cohort that is not. Where an agent is known to be capable of causing a disease, the comparison enables the epidemiologist to calculate the relevant risk (RR) that flows from the particular exposure. An RR of 1 indicates that there is no association between the particular exposure and the risk. An RR of 2 indicates that the particular exposure doubled the chance that the victim would contract the disease. Statistically the likelihood that the victim would have contracted the disease without the particular exposure is then equal to the likelihood that the victim would not have contracted the disease but for that exposure. Where the RR exceeds 2 the statistical likelihood is that the particular exposure was the cause of the disease. The greater the RR the greater the statistical likelihood that the particular exposure caused the disease.

83. An RR of just over 2 is a tenuous basis for concluding that the statistical probable cause of a disease was also the probable biological cause, or cause in fact. The greater the RR the greater the likelihood that the statistical cause was also the biological cause. One reason why an RR of just over 2 is a tenuous basis for determining the biological cause is that the balance of that probability is a very fine one. Another is that the epidemiological data may not be reliable. One reason for this may be that the relevant survey or surveys have been insufficiently extensive to produce data that is truly representative. Epidemiologists conventionally seek to indicate the reliance that can be placed on an RR by determining 95% confidence limits or intervals (C1) around it.

84. The approach that I have been describing focuses on one specific causal agent or a number of specific causal agents. There may well, however, be other causal factors that operate in conjunction with the agent exposure to which is the particular object of investigation, eg the age or genetic susceptibility of the victim. The identification of one probable cause of a disease does not preclude the possibility that there are other contributory causes.

85. Mr Stuart-Smith in his printed case helpfully referred us to a number of foreign authorities which demonstrate that the weight to be attached to epidemiological evidence can vary significantly according to judicial policy. In America the test of causation in toxic tort cases varies from state to state. The most helpful case in the present context is *Merrell Dow Pharmaceuticals Inc v Havner* (1997) 953 SW 2d 706, a decision of the Supreme Court of Texas, for this gives detailed consideration to the “doubles the risk” test. The claim was one of a large number brought against the manufacturer of the prescription drug Bendectin. The parents of a child born with a limb reduction birth defect alleged that the cause of this was Bendectin, taken by the mother when she was pregnant. The parents sought to establish causation by epidemiological evidence which they contended demonstrated that taking this drug more than doubled the risk of such birth defects.

86. Giving the judgment of the court Phillips CJ remarked, at p 716, that the "doubling of the risk" issue in toxic tort cases had provided "fertile ground for the scholarly plow". He proceeded to refer to much of this, summarising the position as follows, at p 717:

> "Some commentators have been particularly critical of attempts by the courts to meld the more than 50% probability requirement with the relative risks found in epidemiological studies in determining if the studies were admissible or were some evidence that would support an award for the claimant. But there is disagreement on how epidemiological studies should be used. Some commentators contend that the more than 50% probability requirement is too stringent, while others argue that epidemiological studies have no relation to the legal requirement of 'more likely than not'."

87. The Chief Justice went on to hold that, although there was not a precise fit between science and legal burdens of proof, properly designed and executed epidemiological studies could form part of evidence supporting causation in a toxic tort case and that there was a rational basis for relating the requirement that there be more than a "doubling of the risk" to the more likely than not burden of proof.

88. At p 718 the Chief Justice commented:

> "But the law must balance the need to compensate those who have been injured by the wrongful actions of another with the concept deeply imbedded in our jurisprudence that a defendant cannot be found liable for an injury unless the preponderance of the evidence supports cause in fact. The use of scientifically reliable epidemiological studies and the requirement of more than a doubling of the risk strikes a balance between the needs of our legal system and the limits of science.
>
> We do not hold, however, that a relative risk of more than 2.0 is a litmus test or that a single epidemiological test is legally sufficient evidence of causation. Other factors must be considered. As already noted, epidemiological studies only show an association."

He then emphasised the need for the design and execution of epidemiological studies to be examined in order to identify possible bias.

89. At pp 720-721 he made a comment that is particularly pertinent in the context of this appeal:

> "Finally, we are cognizant that science is constantly re-evaluating conclusions and theories and that over time, not only scientific knowledge but scientific methodology in a particular field may evolve. We have strived to make our observations and holdings in light of current, generally accepted scientific methodology. However, courts should not foreclose the possibility that advances in science may require re-evaluation of what is 'good science' in future cases."

Can the "doubles the risk" test be applied in multiple cause cases involving diseases other than mesothelioma?

90. For reasons that I have already explained, I see no scope for the application of the "doubles the risk" test in cases where two agents have operated cumulatively and simultaneously in causing the onset of a disease. In such a case the rule in *Bonnington* applies. Where the disease is indivisible, such as lung cancer, a defendant who has tortiously contributed to the cause of the disease will be liable in full. Where the disease is divisible, such as asbestosis, the tortfeasor will be liable in respect of the share of the disease for which he is responsible.

91. Where the initiation of the disease is dose related, and there have been consecutive exposures to an agent or agents that cause the disease, one innocent and one tortious, the position will depend upon which exposure came first in time. Where it was the tortious exposure, it is axiomatic that this will have contributed to causing the disease, even if it is not the sole cause. Where the innocent exposure came first, there may be an issue as to whether this was sufficient to trigger the disease or whether the subsequent, tortious, exposure contributed to the cause. I can see no reason in principle why the "doubles the risk" test should not be applied in such circumstances, but the court must be astute to see that the epidemiological evidence provides a really sound basis for determining the statistical probability of the cause or causes of the disease.

92. *McGhee* may have been such a case. The facts were puzzling, for no other workman had ever contracted dermatitis at the defendants' brick kiln, so one wonders what the basis was for finding that the lack of shower facilities was potentially causative. Had there been epidemiological evidence it seems unlikely that this would have demonstrated that the extra ten or fifteen minutes that, on the evidence, the pursuer took to cycle home doubled his risk of contracting dermatitis, or came anywhere near doing so.

93. Where there are competing alternative, rather than cumulative, potential causes of a disease or injury, such as in *Hotson,* I can see no reason in principle why epidemiological evidence should not be used to show that one of the causes was more than twice as likely as all the others put together to have caused the disease or injury.

Can the "doubles the risk" test be applied in mesothelioma cases?

94. This question calls for consideration of the conundrum that I identified when considering the decisions in *Fairchild* and *Barker*. In the course of argument I put the conundrum to Mr Stuart-Smith. Why, if it was possible to equate increasing exposure to increasing risk, could one not postulate that, on balance of probabilities, where one employer had caused over 50% of a victim's exposure, that employer had caused the victim's mesothelioma? Why could one not, by the same token, postulate that where over 50% of the victim's exposure was not attributable to fault at all, on balance of probability, the victim's mesothelioma had not been caused tortiously? In short, why was there any need to apply the *Fairchild/Barker* rule where epidemiological evidence enabled one to use statistics to determine causation on balance of probability?

95. Mr Stuart-Smith replied that this was a question which puzzled him also. He believed that the answer could be found in consideration given in earlier cases to a hypothetical injury caused by either a blue or a red taxi-cab. This led to some inconsequential discussion as to the colours of the cabs involved. The example in question can be traced, via the speech of Lord Mackay in *Hotson* [1987] AC 750, 789 to the dissenting judgment of Brachtenbach J in *Herskovits v Group Health Cooperative of Puget Sound* (1983) 664 P 2d 474, a decision of the Supreme Court of Washington:

> "Brachtenbach J dissented. He warned against the danger of using statistics as a basis on which to prove proximate cause and indicated that it was necessary at the minimum to produce evidence connecting the statistics to the facts of the case. He gave an interesting illustration of a town in which there were only two cab companies, one with three blue cabs and the other with one yellow cab. If a person was knocked down by a cab whose colour had not been observed it would be wrong to suggest that there was a 75 per cent chance that the victim was run down by a blue cab and that accordingly it was more probable than not that the cab that ran him down was blue and therefore that the company running the blue cabs would be responsible for negligence in the running down. He pointed out that before any inference that it was a blue cab would be appropriate further facts would be required as, for example, that a

> blue cab had been seen in the immediate vicinity at the time of the accident or that a blue cab had been found with a large dent in the very part of the cab which had struck the victim."

96. This example is an extreme example of the fact that statistical evidence may be an inadequate basis upon which to found a finding of causation. Keeping to that example, it was not possible to postulate that the risk of being knocked down by a negligent driver of a taxi-cab was proportional to the number of taxi cabs in the town. Much more significant would have been the care taken by the rival taxi firms in employing competent drivers, and the past accident record of the firms in question.

97. Thus the first answer to the conundrum may be that, in the case of mesothelioma, epidemiological evidence alone has not been considered by the courts to be an adequate basis for making findings of causation: that so long as medical science is unable to demonstrate, as a matter of fact, the aetiology of mesothelioma, data relating incidence to exposure is not a satisfactory basis for making findings of causation.

98. Not only is the adequacy of epidemiological evidence relevant to the weight to be attached to it. So is its reliability. A helpful description of the factors that can limit the reliability of epidemiological evidence is to be found in an article by C E Miller on "Causation in personal injury: legal or epidemiological common sense?" in 26 Legal Studies No 4, December 2006, pp 544 - 569. Deducing causation in relation to mesothelioma on the basis of epidemiological evidence requires a comparison between the statistical relationship between exposure and the incidence of the disease and the experience of the victim who has sustained the disease. A number of factors make this exercise particularly problematic. The first is the difficulty in collating sound epidemiological data. The second is the difficulty of obtaining reliable evidence as to the relevant experience of the victim. The third is uncertainty as to the adequacy of the epidemiological evidence that is available as a guide to causation.

99. The epidemiological data that has been collated in relation to mesothelioma relates largely to the exposure of victims to asbestos dust. It must be gathered from the histories of those who, tragically, have succumbed to mesothelioma. Because of the very long latency of the disease and the limited time between the first experience of its symptoms and death, obtaining the necessary data is difficult. Most of the data relates to victims who were subjected to substantial occupational exposure to dust. This data has been extrapolated to cover victims who have had very light exposure, but there is no certainty that this extrapolation is reliable.

100. The same difficulty arises in relation to obtaining details of the relevant experience of the particular victim. That difficulty is illustrated by the two appeals before the Court.

101. The most significant inhibition on the use of epidemiological evidence to determine causation in cases of mesothelioma is uncertainty as to the adequacy of the data. The data is relied on as establishing that the risk of contracting mesothelioma is proportional to exposure to asbestos dust. It used to be thought that mesothelioma was probably triggered by a single asbestos fibre and that the cause of the disease could be attributed exclusively to that one fibre. Were that the case it would be reasonable to postulate that the risk of contracting the disease was proportional to the exposure. In the words of Lord Hoffmann in *Barker* at para 26, referring to the decision of Moses J at first instance:

> "the more you are exposed, the more likely you are to get it, in the same way as the more you spin the roulette wheel, the more is a given number likely to come up."

102. The single fibre theory has, however, been discredited. The amount of exposure does not necessarily tell the whole story as to the likely cause of the disease. There may well be a temporal element. The Peto Report also raised the possibility (but no more) of synergistic interaction between early and later exposures. Causation may involve a cumulative effect with later exposure contributing to causation initiated by an earlier exposure. Applying the conventional test of causation, the relevant question is, on balance of probability, which exposures in an individual case may have contributed to causing the disease? Epidemiology does not enable one to answer that question by considering simply the relative extent of the relevant exposures.

103. The House of Lords was not, in *Fairchild* nor in *Barker* invited to consider the possibility that it might be possible in an appropriate case to demonstrate by epidemiological evidence that, on balance of probabilities, the mesothelioma had been caused by exposure that was not wrongful, or alternatively that such evidence might demonstrate that one particular employer had, on balance of probabilities, caused the disease. Had it been I do not believe that the House would have been persuaded that epidemiological evidence was sufficiently reliable to base findings as to causation upon it. I believe that the cumulative effect of the various factors that I have set out above justifies the adoption of the special rule of causation that the House of Lords applied in *Fairchild* and *Barker*. The justification for that rule may diminish or vanish as the aetiology of the disease is revealed by scientific research. Nor does the rule wholly displace a conventional approach to causation. Epidemiological data and medical science show that exposure once a cell has

become malignant is not causative and thus exposure once that point is probably passed, can be discounted as a potential contributor to the disease.

104. The possibility that mesothelioma may be caused as the result of the cumulative effect of exposure to asbestos dust provides a justification, even if it was not the reason, for restricting the *Fairchild/Barker* rule to cases where the same agent, or an agent acting in the same causative way, has caused the disease, for this possibility will not exist in respect of rival causes that do not act in the same causative way.

105. I would add that even if one could postulate with confidence that the extent of the contribution of a defendant to the victim's exposure to asbestos precisely reflected the likelihood that his breach of duty had caused the victim's disease, there would still be justification for the application of the *Fairchild* rule where all the exposure was wrongful. Imagine four defendants each of whom had contributed 25% to the victim's exposure so that there was a 25% likelihood in the case of each defendant that he had caused the disease. The considerations of fairness that had moved the House in *Fairchild* would justify holding each of the defendants liable, notwithstanding the impossibility of proving causation on balance of probability.

106. Thus the conundrum is answered by saying that there are special features about mesothelioma, and the gaps in our knowledge in relation to it, that render it inappropriate to decide causation on epidemiological data as to exposure. So far as concerns apportionment between tortfeasors jointly liable for causing mesothelioma it is likely to be necessary to use epidemiological evidence faute de mieux.

What constitutes a material increase in risk?

107. Liability for mesothelioma falls on anyone who has materially increased the risk of the victim contracting the disease. What constitutes a *material* increase of risk? The parties were, I think, agreed that the insertion of the word "material" is intended to exclude an increase of risk that is so insignificant that the court will properly disregard it on the de minimis principle. Mr Stuart-Smith submitted that there should be a test of what is de minimis, or immaterial, which can be applied in all cases. Exposure should be held immaterial if it did not at least double the environmental exposure to which the victim was subject. It does not seem to me that there is any justification for adopting the "doubles the risk" test as the bench mark of what constitutes a material increase of risk. Indeed, if one were to accept Mr Stuart-Smith's argument that the "doubles the risk" test establishes causation, his de minimis argument would amount to saying that no exposure is material for

the purpose of the *Fairchild/Barker* test unless on balance of probability it was causative of the mesothelioma. This cannot be right.

108. I doubt whether it is ever possible to define, in quantitative terms, what for the purposes of the application of any principle of law, is de minimis. This must be a question for the judge on the facts of the particular case. In the case of mesothelioma, a stage must be reached at which, even allowing for the possibility that exposure to asbestos can have a cumulative effect, a particular exposure is too insignificant to be taken into account, having regard to the overall exposure that has taken place. The question is whether that is the position in this case.

The result in this case.

109. Despite Judge Main's heroic endeavours, the nature of the exercise on which he embarked must raise doubts over his precise finding that Greif's wrongful exposure to asbestos dust increased the environmental exposure to which Mrs Costello was subject by 18%. Having made that finding, Judge Main wrongly applied the "doubles the risk" test rather than the *Fairchild/Barker* test. He did not expressly consider whether the exposure to which Greif wrongly subjected Mrs Costello was so insignificant that it could be disregarded as de minimis. None the less, had he thought it de minimis, he might well have said so. He did describe the very small quantities of fibres that might have been on furniture in Greif's offices as "of statistically insignificant effect and de minimis": para 50.

110. I do not think that Judge Main would have dismissed the addition that Greif's wrongful exposure made to the risk that Mrs Costello would contract mesothelioma as statistically insignificant or de minimis. If one assumes, as is likely, that Mrs Costello's disease was asbestos induced, it is plain that a very low level of exposure sufficed to cause the disease. This accords with the expert evidence that there is no known lower threshold of the exposure that is capable of causing mesothelioma. No one could reasonably conclude that there was no significant possibility that the incremental exposure to which Greif subjected Mrs Costello was instrumental in causing her to contract the disease. I am in no doubt that the wrongful exposure to which she was subjected materially increased her risk of contracting mesothelioma.

111. The reality is that, in the current state of knowledge about the disease, the only circumstances in which a court will be able to conclude that wrongful exposure of a mesothelioma victim to asbestos dust did not materially increase the victim's risk of contracting the disease will be where that exposure was insignificant compared to the exposure from other sources. I note that in *Rolls Royce Industrial Power (India) Ltd v Cox* [2007] EWCA Civ 1189 counsel for the

employer conceded that exposure to asbestos dust for a period of one week would not be de minimis.

112. For these reasons I would dismiss the appeal in *Greif*.

ANNEX

A. In the *Trigger* litigation Rix LJ set out the following extract from the judgment of Longmore LJ in *Bolton Metropolitan Borough Council v Municipal Mutual Insurance Ltd* [2006 EWCA Civ 50, [2006] 1 WLR 1492:

> "7 There are three forms of asbestos: brown (amosite), blue (crocidolite) and white (chrysotile). Their fibres have different bio-persistence: 20 years after exposure to fibres about half the inhaled amosite fibres remain in the body, a smaller proportion of the crocidolite fibres remains and, relatively, few chrysotile fibres remain.
>
> 8 The human body is composed of cells of various types. Of the fibres which reach the lungs many are engulfed by macrophages (scavenger cells). The macrophages may then be expelled by the mucosiliary process or may die within the lungs. All cells can and do die for various reasons, but cells are in communication with each other and the death of one can cause another to divide so, with some exceptions such as men losing their hair with age, the number of cells remains approximately the same throughout a person's life. When macrophages die in the lungs they release various chemicals, some of which attract neutrophils, another type of cell, which can engulf fibres. A different mechanism which destroys fibres in the lungs is that they are dissolved in tissue fluids. Another mechanism, by which the body protects itself, is that some fibres become coated by proteinaceous material containing iron which, it is believed, renders them less likely to produce fibrosis.
>
> 9 The division of cells in human tissue is important for understanding how mesothelioma occurs. Each cell in the body contains all the genetic information necessary for the construction and functioning of the entire body. This information is contained in the form of DNA, a molecule consisting of two intertwining strands. The different structure and function of the various types of cell in the

body occurs because in each cell only some of the genes contained in the DNA are active and in different cells different genes are active. The coded information in a DNA molecule is in the form of about 3,000,000,000 ‘base pairs’. Each pair consists of two collections of atoms called nucleotides. There is one half of each pair in each of the two intertwining strands. When cell division occurs the strands unravel and two ‘daughter’ double helices are created. Normally the daughters are identical with each other but sometimes they are not. Dr Rudd uses the word ‘mutation’ for an imperfect copy. This word ‘mutation’ thus means a thing - a cell - and not a process, and is not a synonym of ‘change’; for change Dr Rudd uses the term ‘generic alteration’. I shall adopt this usage. The word ‘mutation’ does not have any derogatory connotations. A mutation is different from, but not necessarily worse than, the cell from which it is derived or otherwise undesirable. The body contains what can be described as a ‘repair mechanism’ which sometimes corrects the discrepancy between a daughter and its parent. This repair mechanism is vital to normal health, and people whose repair system lacks some components (a very rare condition) will die early, often of cancer. Sometimes, however, a perfectly normal repair and correction mechanism fails to correct a mutation. Such failure can lead to any of three possibilities. First, the mutation may be unable to survive and die. Secondly it may be better fitted for its purpose than the cell from which it is derived, and this is the cause of evolution. As Dr Moore-Gillon put it ‘Without the normal process of imperfect copying, mankind (and indeed all other species) would not have emerged’.

10 It is the third possibility with which this case is concerned. A mutation which does not die, which is not repaired and which does not perform its purpose better than the cell from which it was derived may itself divide, and the ‘daughter’ cells or (to continue the parental analogy) the grand-daughter or more distant descendants may in turn die, be repaired or be mutations from the cell from which they are derived. Eventually there may be a mutation which is malignant, i e a cell which divides in an uncontrolled manner, as opposed to maintaining the normal balance between cells dying and cells dividing. It normally takes a ‘heredity’ of six or seven genetic alterations before a malignant cell occurs. The body has ‘natural killer’ cells which, as their name indicates, can target and destroy mutations, possibly even after they have become malignant. A tumour is a growth consisting of a number of cells dividing in that uncontrolled manner. Mesothelioma is a tumour in the pleura...

11 Asbestos fibres in the pleura increase the likelihood of genetic mutation. It is now thought likely that, if there is a series of genetic alterations which ends with a malignant cell in the pleura, fibres will have acted in causing several of those genetic alterations, rather than just one genetic alteration. However the final genetic alteration which results in a malignant cell is not necessarily caused by fibres directly. Fibres may also inhibit the activity of natural killer cells. Pre-cancerous genetic alterations in cells do not give rise to any symptoms or signs. They cannot be detected by any routine clinical or radiological examination. It would be possible to detect them by examining in a laboratory tissue taken from a part of the body containing cells which have become genetically modified, but the exercise would be pointless because pre-cancerous genetic alterations do not necessarily or even usually lead to mesothelioma.

12 It is furthermore important to note that there may be a long time lapse not only between exposure and the first formation of a malignant cell but that there may be a similarly lengthy lapse of time between first malignancy and the onset of noticeable symptoms such as breathlessness."

B. Rix LJ then summarised the findings of Burton J in the *Trigger* litigation, which brought the findings of Longmore LJ up to date:

"50. The judge heard evidence from five internationally recognised experts in the field: Dr Rudd and Dr Moore-Gillon, who have between them given evidence in most if not all of the cases involving mesothelioma in recent years including *Fairchild* and *Bolton* itself; Professor Geddes, on whose pioneering work the first two experts have based their own theories (see his crucial 1979 paper concerning the rate of tumour growth, published in volume 73 of the British Journal of Diseases of the Chest, *The Natural History of Lung Cancer: a Review based on Rates of Tumour Growth* (the 'Geddes article')); and Professor Phillips of the Institute of Cancer Research and Professor Heintz of the Vermont Cancer Centre. The last two are biochemists, the first three are respiratory consultants. The judge observed that the evidence of the biochemistry experts is a new feature of such litigation.

51. On the basis of this expert evidence, the judge remarked on two matters which were common ground between the parties. One is that it is the exposure to quantities of fibres which is causative of mesothelioma, and the risk increases with the dosage. This was

recognised already in *Fairchild* (see Lord Bingham at para 7; and Lord Rodger at para 122, where the latter observed: 'the greater the number of asbestos fibres taken into the body, the greater are the chances that one of them will trigger a malignant transformation'). The second matter is that once the mesothelioma tumour is present and assured of growth (ie has passed the stage where a malignant mutation may die off), further asbestos exposure and indeed further asbestos fibres in the body can make no difference and are not causative.

52. Burton J also described 'the unknowability and indescribability of much of the pathogenesis of mesothelioma' as being common ground (at para 30). Subject to that caution, the judge made the following findings about the disease. He described asbestos fibre as a 'complete carcinogen', ie no other agent or co-agent is required to cause the ultimate malignancy (at para 130). Unlike a normal cancer of spherical or similar shape which sooner or later can be seen on a scan or x-ray, the mesothelioma tumour grows along the surface of the lungs rather like a fungus and is thus practically undetectable, and only becomes diagnosable when the symptoms of impaired breathing bring it to the patient's and his doctor's attention. As the details of actions 1-3 illustrate, that is only shortly before death. The average time between manifestation/diagnosis and death is some fourteen months.

53. The judge described the normal process of cell mutations in healthy bodies and lungs. Even in a person who has not been exposed to asbestos as part of his occupation, the lungs will typically contain millions of asbestos fibres, albeit not the hundreds of millions to be found in the occupationally exposed and with far less proportionately of the more dangerous blue and brown asbestos varieties. He said:

> '108…The mesothelial cells, like all cells in the body, are constantly dividing: Dr Rudd told us that there are 10 trillion cells in the body and 50 billion are replicated every day. Cell division, or mitosis, by which the cell divides, duplicates its chromosomes and passes on a complete set to each of its "daughters", is the norm; but there can be mutations – again Dr Rudd told us that incorrect copying can take place in one in a million cell divisions and thus possibly 5,000 times per day in the human body, or every 17 seconds. The body's repair mechanisms are quick to correct and

> abort the mutations, but even if there are mutations there are four possible consequences. The incorrect copy may be unable to survive, and die; the mutation can make no difference; the mutation can positively improve the cell – hence evolution; or the mutated cell can survive and can itself divide, passing on the genetic alterations, eventually after many generations and with further mutations creating a malignant cell.'

54. What then makes the difference between a normal and a diseased process? The judge continued:

> '109. There will or may be thousands of mutations, only one of which may have any deleterious effect on successive mitosis. But, the experts gave evidence that there are six or seven genetic alterations which are required, not necessarily occurring in the same or any particular order, which, when they are all in place, can lead to a malignant cell. The characteristics of a malignant cell are (i) self sufficiency of growth signals (ii) insensitivity to growth-inhibitory signals (iii) evasion of programmed cell death (apoptosis) (iv) limitless replicative potential (v) the ability to invade tissues and to metastasise ie to transfer to other parts of the body (vi) the availability of its own blood supply – obtained by a process which is called *angiogenesis*…
>
> 111. Once a cell has acquired what Dr Rudd calls a '*full house*' of the necessary 6/7 mutations, and has evaded all the bodily defences (described by Dr Rudd as '*full house plus*'), then it can be described as a malignant cell, and can and does begin a period of uncontrolled growth by multiplication. Notwithstanding what Dr Rudd has called evasion of the bodily defences, Professors Phillips and Heintz [the biochemists] conclude that many *full house* cells with malignant potential may fail to grow into tumours. It appears to be common ground, at any rate so far as the biochemists are concerned, that such cell or cells at this stage are still at risk from natural killer cells, although they apparently develop a method of switching off the signals which summon the natural killer cells or put them on notice. There is also, despite the characteristic of limitless replication, the possibility or probability, of periods of dormancy. Professor Phillips points out that the norm of 40 years from exposure to diagnosability

suggests either that the mutation period lasts a long time or that there are periods of tumour dormancy (or both).'

55. The judge then described the growth of a malignant cell towards the status of a mesothelioma tumour, premised on the figures to be derived from the *Geddes* article concerning the more normal type of spherical tumour. Professor Geddes found that the average rate of doubling of cells was 102 days (albeit that was a speculative average, which could vary between 45 and 130 days). It is only at a tumour size of 10^6 cells (1 million cells) that it becomes unlikely for the bodily defences, still until then available, to be able to neutralise it. Angiogenesis then occurs at somewhere between 10^6 and 10^9 (1 billion cells). Symptoms of breathlessness will begin to be experienced when the tumour is between 10^9 and 10^{12} (1 trillion cells). In the biochemists' view, angiogenesis occurred about 5 years or so before death."

C. The Peto and Rake study led the authors to the following conclusions:

"1. Mesothelioma risk is determined largely by asbestos exposure before age 30, and ranges from a lifetime risk of 1 in 17 for ten or more years of carpentry before age 30 to less than 1 in 1,000 in apparently unexposed men and women. Our results suggest that the predicted total of 90,000 mesotheliomas in Britain between 1970 and 2050 will include approximately 15,000 carpenters.

2. The risk of lung cancer caused by asbestos is likely to be of the same order as the mesothelioma risk. This would imply that more than 1 in 10 of British carpenters born in the 1940s with more than 10 years of employment in carpentry before age 30 will die of a cancer caused by asbestos.

3. Asbestos exposure was widespread, with 65% of male and 23% of female controls having worked in occupations that were classified as medium or higher risk.

4. Britain was the largest importer of amosite (brown asbestos), and there is strong although indirect evidence that this was a major cause of the uniquely high mesothelioma rate. The US imported far less amosite than Britain but used similar amounts of chrysotile (white asbestos) and more crocidolite (blue asbestos), and US mesothelioma

death-rates in middle age are now 3 to 5 times less than British rates. British carpenters frequently worked with asbestos insulation board containing amosite.

5. We found no evidence of increased risk associated with non-industrial workplaces or those that were classified as 'low risk', including motor mechanics and workers handling gaskets and mats that may have contained asbestos.

6. The only potential non-occupational exposure associated with increased risk was living with an exposed worker.

7. The increasing trend in female rates in Britain and a comparison between British and US female rates both suggest that a substantial proportion of mesotheliomas with no known occupational or domestic exposure were probably caused by environmental asbestos exposure. The sources of this presumably included construction, building maintenance and industrial activities but may also include release of asbestos from buildings due to normal occupation and weathering."

LORD RODGER

113. Defendants whose breaches of duty expose someone to asbestos and so materially increase the risk that he will develop mesothelioma are liable jointly and severally for the damage which he suffers if he does in fact develop mesothelioma. The fundamental question in these two appeals is whether this special rule – the so-called "*Fairchild* exception", as it applies to mesothelioma – applies in cases where only one defendant is proved to have exposed the victim to asbestos, but she was also at risk of developing the disease from low-level exposure to asbestos in the general atmosphere ("environmental exposure"). I would hold that the special rule does apply in such cases.

Karen Sienkiewicz v Greif (UK) Ltd

114. In these proceedings the claimant, Mrs Karen Sienkiewicz, is the daughter, and administratrix of the estate of, the late Mrs Enid Costello who died of mesothelioma on 21 January 2006.

115. From 1966 until 1984 Mrs Costello worked for the defendants' predecessors in title at their factory premises in Ellesmere Port where they manufactured steel drums. The process involved the release of asbestos dust into the factory atmosphere. Although Mrs Costello worked mostly in an office, she spent time in areas of the factory which were, from time to time, contaminated with asbestos.

116. The trial judge held that Mrs Costello's exposure to asbestos on the defendants' premises was "very light" and that it would have been through the inhalation of the general factory atmosphere, as she moved about. The judge also held that this exposure was in breach of the relevant legal duties owed by the defendants to Mrs Costello.

117. It was common ground that, like anyone else, Mrs Costello would have been subject to environmental exposure to low levels of asbestos in the atmosphere in the areas where she lived.

118. The trial judge found that the defendants' exposure of Mrs Costello to asbestos over her working life at their premises "increased her background risk (of contracting mesothelioma) from 24 cases per million to 28.39 cases per million, an increase of risk of 18%." Putting the point slightly more precisely, the environmental risk of contracting mesothelioma was 24 cases per million; exposure of the level of the occupational exposure in Mrs Costello's case would increase the risk of contracting mesothelioma to 28.39 cases per million – an increase of 18%.

119. The trial judge concluded that the claimant had failed to establish that any exposure by the defendants had caused Mrs Costello's mesothelioma because "once there is only one occupational cause for the mesothelioma the claimant has to prove that it is the likely cause." On this basis he held that the special rule of law laid down by the House of Lords in *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 did not apply and that the claimant could therefore not succeed on the basis that, on the balance of probability, Mrs Costello's exposure to asbestos in the course of her employment with the defendants had materially increased the risk that she would contract mesothelioma. She could only succeed by proving, on the balance of probability, that the defendants' breach of duty had caused Mrs Costello's mesothelioma.

120. The Court of Appeal (Lord Clarke of Stone-cum-Ebony, Scott Baker and Smith LJJ) allowed the claimant's appeal: *Sienkiewicz v Greif (UK) Ltd* [2009] EWCA 1159; [2010] QB 370. They held that the decision of the House of Lords in *Fairchild* applied. The defendants' breach of duty had materially increased the risk

of Mrs Costello developing mesothelioma. So they were liable. The defendants appeal against that decision.

121. Although the Court of Appeal ultimately held that the rule in *Fairchild* applied to mesothelioma cases of this kind because of section 3 of the Compensation Act 2006 ("the 2006 Act"), in the course of her judgment, [2010] QB 370, 379, at para 23, Smith LJ made a very general statement about the approach which courts should adopt to issues of causation:

> "In my view, it must now be taken that, saving the expression of a different view by the Supreme Court, in a case of multiple potential causes, a claimant can demonstrate causation by showing that the tortious exposure has at least doubled the risk arising from the non-tortious cause or causes."

An important issue in the present appeals is whether this guidance is sound.

Willmore v Knowsley Metropolitan Borough Council

122. In these proceedings the claimant is Mr Barré Willmore. He is the husband, and administrator of the estate, of the late Mrs Dianne Willmore who died of mesothelioma on 15 October 2009 at the age of 49. Prior to her death, Mrs Willmore had raised proceedings for damages for her illness against Knowsley Metropolitan Borough Council ("the Council").

123. After her condition was diagnosed, Mrs Willmore made a number of different allegations as to her possible exposure to asbestos. Initially she alleged that she had been exposed to asbestos dust in the course of her employment with the Army & Navy Stores in Liverpool between 1979 and 1981. But when she raised her proceedings against the Council in February 2008 she alleged that she had been exposed to asbestos when some prefabricated houses near her childhood home in Huyton were demolished. She also alleged that she had been exposed to asbestos while a pupil at her primary school run by the Council.

124. On 14 February 2008, however, Mrs Willmore read an article in the *Liverpool Echo* referring to a report prepared by the Council which identified the presence of asbestos in a number of secondary schools, including Bowring Comprehensive, where she had been a pupil. On 27 November 2008 Mrs Willmore amended the particulars of claim to allege, in essence, that when she first attended Bowring Comprehensive, the construction of the school had not been completed and she and other pupils had been exposed to asbestos as a result of workmen

using materials containing asbestos. She also alleged that she had been exposed to asbestos as a result of other disturbance of asbestos materials at the school. She subsequently abandoned all her allegations of exposure to asbestos except those relating to Bowring Comprehensive.

125. Following a trial in July 2009, Nicol J found that, while a pupil at Bowring Comprehensive, Mrs Willmore had been exposed to the type of asbestos known as amosite in three separate ways: (1) as a result of work involving the removal, handling and disturbance of ceiling tiles in a corridor along which pupils, including Mrs Willmore, passed; (2) as a result of pupils' misbehaviour, which caused ceiling tiles containing asbestos to be damaged or broken; (3) as a result of asbestos ceiling tiles, including broken tiles, being stored in a girls' lavatory which had been used by Mrs Willmore on many occasions. The judge held that each of these exposures to asbestos fibres had materially increased the risk of Mrs Willmore contracting mesothelioma later in life. In so concluding, he found that none of these exposures was de minimis. He awarded Mrs Willmore the agreed gross sum of £240,000 as damages.

126. The Council appealed to the Court of Appeal. The Court of Appeal held, [2009] EWCA Civ 1211, that the judge had been wrong to hold that she had been exposed to asbestos as a result of pupils' misbehaviour. But they confirmed that the judge had been entitled to find that Mrs Willmore had suffered significant exposure to asbestos from the other two sources. On that basis the Court upheld his judgment and his award of damages. The Council now appeal to this Court. Since the lower courts applied the *Fairchild* exception, obviously the same point as to its application in this type of case arises. But the Council also challenge the judge's findings in fact.

The Defendants' Legal Argument

127. As already indicated, the feature of both the cases under appeal to which the defendants attach importance is that the proceedings are directed against only one defendant. In this respect they are different from the leading authorities, *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 and *Barker v Corus UK Ltd* [2006] 2 AC 572, in both of which the claimants alleged that the victims had been exposed to asbestos as a result of a breach of duty by more than one employer. In *Barker*, however, one of the three material exposures had occurred when Mr Barker was working as a self-employed plasterer.

128. On behalf of the defendants in both of the appeals, Mr Stuart-Smith QC characterised the present cases as "single exposure" cases: the claimants alleged only one possible tortious source for the exposure. In both cases the exposure

could be regarded as slight. In addition, the victims had been exposed to asbestos in the general atmosphere in the areas where they lived. Counsel renewed the argument that in such cases the special rule in *Fairchild* did not apply and that, in order to establish liability, the claimant required to prove, on the balance of probability, that the victim's mesothelioma is to be attributed to her exposure to asbestos as a result of the defendant's breach of duty. The claimant could do this by leading epidemiological evidence to show that the exposure by the defendant had doubled the risk of the victim developing mesothelioma. This was essentially the argument which the trial judge had accepted in *Sienkiewicz*: the claimant failed because the defendants' breach of duty had merely increased the risk of her developing mesothelioma by 18% – far short of doubling the environmental risk.

Section 3 of the 2006 Act

129. In the Court of Appeal in *Sienkiewicz* [2010] QB 370, 379, para 26, Smith LJ saw considerable force in the view that in *Fairchild* and *Barker* the House of Lords had not been considering a single exposure case and that, if they had done so, they would not have included such a case within the scope of the rule. But she held that such speculation was now pointless since Parliament had intervened by enacting section 3 of the 2006 Act, which had the effect that the common law simpliciter no longer governed claims for damages in mesothelioma cases. In this regard Smith LJ observed, [2010] QB 370, 381-382, at paras 34 and 35:

> "34. However, in my view, Parliament used clear words which provide that, in all mesothelioma cases, a claimant can take advantage of section 3(2) provided that he or she can satisfy the four conditions in section 3(1) and the fourth condition can, in my judgment, be satisfied by proof of causation by reference to a material increase in risk.
>
> 35. I conclude therefore that, in a mesothelioma case, it is not open to a defendant to put the claimant to proof of causation by reference to a twofold increase in risk. The judge was therefore wrong to require the claimant in this case to attempt to cross that hurdle. If he had applied the correct test on causation, namely whether or not the tortious exposure had materially increased the risk, the answer was plainly yes. In my view, the claimant should have succeeded and the appeal must be allowed."

Scott Baker LJ agreed with Smith LJ, as did Lord Clarke of Stone-cum-Ebony. Lord Clarke considered, [2010] QB 370, 387, at para 57, that it was plain from the

terms of section 3 and from the analysis of the common law that the respondent was liable for the mesothelioma which caused Mrs Costello's death.

130. Subsection (1) of section 3 of the 2006 Act describes the circumstances in which the section is to apply in actions of damages for mesothelioma. According to subsection (1)(d), it applies where "the responsible person is liable in tort, by virtue of the exposure mentioned in paragraph (a) in connection with damage caused to the victim by the disease (whether by reason of having materially increased a risk or for any other reason)." Smith LJ appears to have considered that, by referring to the defendant being held liable in tort "by reason of having materially increased a risk", Parliament had precluded any argument that, in particular circumstances, a defendant could not be held liable on that basis. I would not read the provision in that way.

131. Section 3 was not concerned with prescribing the basis for defendants being held responsible for claimants' mesothelioma. Rather, its purpose was to reverse the decision of the House of Lords in *Barker v Corus UK Ltd* [2006] 2 AC 572. The House had held that, where more than one defendant had materially increased the risk that an employee would contract mesothelioma, liability was to be attributed, not jointly and severally, but according to each defendant's degree of contribution to the risk. In section 3 Parliament laid down that, on the contrary, where a defendant was held liable in a mesothelioma case, he was to be liable for the whole of the damage caused to the victim and, if anyone else was held responsible, they were to be liable jointly and severally. The reference to the defendant having been held liable "by reason of having materially increased a risk" is simply designed to show that the statutory rule applies in cases where the defendant is held liable (as in *Barker*) on the basis of materially increasing the risk to the claimant. But the concluding words, "or for any other reason", show that Parliament envisages that a defendant might be held liable on some other basis. In that eventuality also he is to be liable for the whole of the damage and, if anyone else is held responsible, they are to be liable jointly and severally.

132. It follows that section 3 of the 2006 Act does not shut out the appellants' argument that in a single exposure case a defendant should not be held liable unless the claimant proves on the balance of probability that his breach of duty caused the victim's mesothelioma. That argument and the more particular argument, that the claimant must show that the defendant more than doubled the risk of the victim developing mesothelioma, have therefore to be addressed on their merits.

The Rock of Uncertainty

133. The discussion and decision in *Fairchild* proceeded on the basis described by Lord Bingham, [2003] 1 AC 32, 43, at para 7: "There is 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." This was what he described as the "rock of uncertainty": [2003] 1 AC 32, 43G-H. On behalf of the appellants, Mr Stuart-Smith accepted that this remains the position in cases where a victim has been exposed to asbestos in the course of his employment with a number of employers. The same would presumably apply if the victim had been exposed to asbestos, say, when visiting a number of cinemas run by different companies. But he submitted that, where the claimant alleges that only one defendant wrongfully exposed her to asbestos and environmental exposure is also a possible source of the asbestos which affected her, the claimant must prove on the balance of probability that her disease was caused by the defendant rather than by environmental exposure.

134. In *Fairchild*, as can be seen from Lord Bingham's speech, at p 40, para 2, it was common ground that "any cause of [the claimant's] mesothelioma other than the inhalation of asbestos dust *at work* can be effectively discounted" (emphasis added). At the time, some commentators indeed found this surprising, since exposure can occur in a variety of ways. Most obviously, perhaps, a factory may pollute the surrounding area and lead to the residents inhaling asbestos fibres in the atmosphere. But fibres are actually widespread in the atmosphere throughout most of the country. One European study suggested that one person in seven shows lung damage of a kind caused by exposure to asbestos. See the examples in Jane Stapleton, "Lords a'leaping evidentiary gaps", (2002) 10 Torts Law Journal 276, 277-279. But, for some reason, only certain people develop mesothelioma as a result of being exposed to asbestos. The issue in the present appeals arises because both parties accept that Mrs Costello and Mrs Willmore, who did develop mesothelioma, might have developed it as a result of being exposed to asbestos in the general atmosphere.

135. At first sight it is somewhat surprising that the defendants should submit that in these cases the claimant must prove, on the balance of probability, that the defendant's breach of duty caused her illness, since *Fairchild* proceeded on the basis that there is no way of identifying, on the balance of probability, the source of the fibre or fibres which initiated the genetic process that culminated in the victim's malignant tumour. Medical science has not advanced significantly in this respect in the intervening eight years. So counsel's argument is – and must be – that, in a case where the only possible source of the fibre or fibres which caused the disease is either environmental exposure to asbestos or exposure by the defendant, a claimant could always have proved, on the balance of probability, that the defendant was the source of the relevant fibre or fibres by leading appropriate

epidemiological evidence to show that the exposure by the defendant more than doubled the background risk of the victim developing mesothelioma. So the *Fairchild* exception would never have applied.

136. Take *Sienkiewicz* as an example. The defendants argue that the claim fails since, on the basis of the expert evidence, the judge found that the exposure due to their breach of duty increased Mrs Costello's risk of developing mesothelioma by only 18%. By contrast, it is said, if the expert evidence had shown that their exposure had doubled the background risk, Mrs Costello would have proved that, on the balance of probability, her mesothelioma had been caused by the defendants' breach of duty rather than by any environmental exposure. In that event the claim would have succeeded. There is no rock of uncertainty and so no room for the *Fairchild* exception. By applying *Fairchild*, the Court of Appeal had erred in law and the appeal should therefore be allowed.

Unpacking the Defendants' Legal Argument

137. The defendants' argument appears simple, but it would actually involve a major change in the law.

138. Usually, in English or Scots law, a court awards a claimant or pursuer damages for his injuries only if the judge is satisfied, on the balance of probability, that the wrongful act of the defendant or defender actually caused, or materially contributed to, his injury. Unless he proves this, his claim will fail.

139. In the case of a disease like mesothelioma the claimant will be able to prove on the balance of probability that he is suffering from mesothelioma and that he has suffered loss as a result. He may also be able to prove, on the balance of probability, that a defendant or a number of defendants negligently exposed him to asbestos in the course of his employment with them, or while – as in Mrs Willmore's case – she was a pupil in a school run by the Council. What, however, the claimant will be quite unable to prove, on the balance of probability, in the present state of medical knowledge, is that he developed mesothelioma as a result of inhaling any particular fibre or fibres and that, therefore, a particular defendant was responsible for exposing him to the fibre or fibres that caused his illness. Moreover, medical experts are no more able to tell whether the fibre or fibres which triggered the claimant's mesothelioma came from the general atmosphere than they can tell whether they came from exposure during the claimant's work with one or other of a number of employers.

140. Faced with the problem that, in the present state of medical science, a claimant can never prove his case to the standard that the law usually requires, a legal system may react in a variety of ways. It may simply adhere to its usual stance and say that, since the claimant has not proved on the balance of probability that the defendant actually caused his disease, the claim must fail. That was, in effect, what the Court of Appeal decided in *Fairchild v Glenhaven Funeral Services Ltd* [2002] 1 WLR 1052. Alternatively, if that approach seems to be unduly harsh on victims, a system may hold that, if the claimant proves on the balance of probability that the defendant's breach of duty has exposed him to asbestos, an evidential burden falls on the defendant to show that this exposure did not play any part in the claimant's illness. *Menne v Celotex Corp* 861 F 2d 1453 (10 Cir 1988) is a case in point. Another possibility would be that a system would choose to hold a defendant liable because his breach of duty doubled the risk that his employee would develop mesothelioma. The decision of the Supreme Court of Texas in *Merrell Dow Pharmaceuticals Inc v Havner* (1997) 953 SW 2d 706 is an example of that approach being carefully applied in relation to proof that a mother's consumption of a drug caused a birth defect in her baby. As I point out at para 154 below, the court was conscious that it was deliberately applying a special rule to deal with the particular evidential difficulties facing plaintiffs in that kind of case. Or else a system may adopt a (different) rule to the effect that, if the claimant proves, on the balance of probability, that the defendant materially increased the risk that he would develop mesothelioma, then the defendant is to be held to have contributed materially to the development of the claimant's illness. That is what the House of Lords appeared to do in *Fairchild*. In *Barker v Corus UK Ltd* [2006] 2 AC 572, however, the approach in *Fairchild* was refined: it was now said that a defendant was liable simply on the basis that his breach of duty had materially increased the risk that his employee would contract mesothelioma and the employee had done so.

141. The response of English law to the problem posed by the rock of uncertainty in mesothelioma cases is therefore to be found in the combination of the common law, as laid down in *Fairchild* and *Barker*, and section 3 of the 2006 Act. Defendants whose breaches of duty materially increase the risk that the victim will develop mesothelioma are liable jointly and severally for the damage which the victim suffers if he does in fact develop mesothelioma. This is the current version of the *Fairchild* exception, as it applies in cases of mesothelioma.

142. Of course, the *Fairchild* exception was created only because of the present state of medical knowledge. If the day ever dawns when medical science can identify which fibre or fibres led to the malignant mutation and the source from which that fibre or those fibres came, then the problem which gave rise to the exception will have ceased to exist. At that point, by leading the appropriate medical evidence, claimants will be able to prove, on the balance of probability, that a particular defendant or particular defendants were responsible. So the

Fairchild exception will no longer be needed. But, unless and until that time comes, the rock of uncertainty which prompted the creation of the *Fairchild* exception will remain.

Proof of a Fact and Proof of a Probability

143. Although a claimant cannot prove what happened, in any given case his illness has a determinate cause. In other words, his mesothelioma was actually caused by a particular fibre or fibres and so a particular defendant either did or did not materially contribute to his contraction of the disease. Whether a defendant did so is a matter of fact, but one which, in the present state of medical science, we can never know.

144. In *Hotson v East Berkshire Area Health Authority* [1987] AC 750 the plaintiff fell from a tree and sustained an acute traumatic fracture of the left femoral epiphysis. He was taken to hospital, but his injury was not correctly diagnosed or treated for five days. In the event, he suffered avascular necrosis of the epiphysis, involving disability of the hip joint and the virtual certainty that he would later develop osteoarthritis. The health authority admitted negligence. The trial judge, Simon Brown J, found that, even if the hospital had diagnosed the injury and treated the plaintiff promptly, there was a 75% chance that the necrosis would still have developed. He held that the plaintiff was entitled to damages for the loss of the 25% chance that he would have made a full recovery if treated promptly: [1985] 1 WLR 1036. The Court of Appeal upheld the trial judge: [1987] AC 750. The House of Lords allowed the health authority's appeal.

145. The House of Lords emphasised that what had happened to the plaintiff by the time he reached hospital was a matter of fact – albeit one as to which there was no direct evidence and as to which the medical experts who gave evidence were divided. As a matter of fact, by the time he reached hospital, the plaintiff either did or did not have sufficient intact blood vessels to keep the affected epiphysis alive. In the words of Lord Mackay of Clashfern, [1987] AC 750, 785A-B, on that matter, having regard to all the evidence, including the conflicting medical evidence, the trial judge took the view "that it was more probable than not that insufficient vessels had been left intact by the fall to maintain an adequate blood supply to the epiphysis …". Lord Mackay went on to say, at p 785C-E:

> "It is not, in my opinion, correct to say that on arrival at the hospital he had a 25 per cent chance of recovery. If insufficient blood vessels were left intact by the fall he had no prospect of avoiding complete avascular necrosis whereas if sufficient blood vessels were left intact on the judge's findings no further damage to the blood supply would

> have resulted if he had been given immediate treatment, and he would not have suffered the avascular necrosis."

146. In *Hotson* therefore not only was the plaintiff's condition by the time he reached hospital a matter of fact, but it was one which, the House held, the trial judge had been able to determine, on the balance of probability: insufficient vessels were left intact to maintain an adequate blood supply to maintain the epiphysis. Here, by contrast, although as a matter of fact, for instance, the defendants' exposure of Mrs Costello to asbestos dust either did or did not materially contribute to her contraction of the disease, in the present state of medical science we can never know – and the claimant can never prove – whether it did or did not.

147. Lord Hoffmann made the same point in *Gregg v Scott* [2005] 2 AC 176, 196, at para 79, when he said that, for the law

> "Everything has a determinate cause, even if we do not know what it is. The blood-starved hip joint in *Hotson's* case, the blindness in *Wilsher*'s case, the mesothelioma in *Fairchild*'s case; each had its cause and it was for the plaintiff to prove that it was an act or omission for which the defendant was responsible. The narrow terms of the exception made to this principle in *Fairchild's* case only serves to emphasise the strength of the rule. The fact that proof is rendered difficult or impossible because no examination was made at the time, as in *Hotson's* case, or because medical science cannot provide the answer, as in *Wilsher's* case, makes no difference. There is no inherent uncertainty about what caused something to happen in the past or about whether something which happened in the past will cause something to happen in the future. Everything is determined by causality. What we lack is knowledge and the law deals with lack of knowledge by the concept of the burden of proof."

148. It appears that in the House of Lords in *Hotson* there was some argument about the use of statistical evidence, but most members of the appellate committee did not find it necessary to deal with it. Lord Mackay did address the issue, however – while making it clear that his comments were obiter. At the hearing of the present appeals counsel made some reference to Lord Mackay's comments and Lord Phillips has referred to them in his judgment. It may therefore be worthwhile to look a little more closely at what Lord Mackay said in order to see whether it has any application in the present case.

149. Lord Mackay put forward a hypothetical example loosely based on *McGhee v National Coal Board* [1973] 1 WLR 1. He supposed a case in which an employer had negligently failed to provide washing facilities at the end of their shift for men who had been exposed to brick dust in the course of their work. One of the men developed dermatitis and sued his employer. He led epidemiological evidence which showed that of 100 men working in the same conditions 30 would develop dermatitis even though they had showered after their shift. But the evidence also indicated that, if the men did not shower, 70 would develop dermatitis. Lord Mackay observed, [1987] AC 750, 786D-E:

> "Assuming nothing more were known about the matter than that, the decision of this House [in the *McGhee* case] may be taken as holding that in the circumstances of that case it was reasonable to infer that there was a relationship between contraction of dermatitis in these conditions and the absence of washing facilities and therefore it was reasonable to hold that absence of washing facilities was likely to have made a material contribution to the causation of the dermatitis."

150. Two comments are appropriate. First, the decision of the House of Lords in *McGhee* actually goes much further than holding that, in such circumstances, it is reasonable to infer that the absence of washing facilities was likely to have made a material contribution to the causation of the dermatitis. As Lord Mackay himself pointed out, in *McGhee* there were no statistics. The House had to deal with the appeal on the basis of the evidence of Dr Hannay, a dermatologist led by the pursuer, which the Lord Ordinary had accepted. Dr Hannay, who was not cross-examined on the point, said that the provision of showers would have "materially" reduced the risk of the pursuer contracting dermatitis: 1973 SC (HL) 37, 42. So the lack of showers "materially" increased the risk of the pursuer contracting dermatitis. In these circumstances, "from a broad and practical viewpoint," Lord Reid could 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": *McGhee v National Coal Board* [1973] 1 WLR 1, 5B-C. From his previous reference, at p 4D-F, to *Bonnington Castings Ltd v Wardlaw* [1956] AC 613 it is evident that Lord Reid was thinking of any increase in the risk that could not be regarded as de minimis. There would, for example, have been a material (20%) increase in the risk in a case like *McGhee*, if 30 out of the population of 100 workmen would have been expected to develop dermatitis even after showering, but 36 would have been expected to develop it if no showers were provided. On that basis the House would have held the defenders liable.

151. Secondly, as Lord Phillips points out, Lord Mackay must be supposed to have chosen the figures in his hypothetical example because, among the population of 100 workmen exposed to brick dust, more than twice as many (70) would be

expected to develop dermatitis if no showers were provided, as would be expected to develop it even if showers were provided (30). In terms of the defendants' argument in the present appeals, failure to provide showers would more than double the risk. In that situation, assuming that nothing more were known, Lord Mackay thought that the House might be taken as holding that it was reasonable to infer that there was a relationship between contraction of dermatitis in these conditions and the absence of washing facilities and therefore it was reasonable to hold that absence of washing facilities was likely to have made a material contribution to the causation of the claimant's dermatitis.

152. Lord Mackay's introductory words ("assuming nothing more were known") show that he was conscious that, if the House did indeed reason in that way, it would be reasoning, from statistics about the situation in a population of 100 workmen in the same conditions, to the case of the individual claimant. Obviously, care has to be taken in doing so. For example, if the claimant had some underlying condition which made him particularly sensitive to brick dust, that would affect any reliance that could be placed on the statistics in his case.

153. More fundamentally, however, it is necessary to see what the epidemiological evidence would actually show in Lord Mackay's hypothetical case. Suppose the claimant, who had not been able to shower, developed dermatitis. As a matter of fact, he either developed the dermatitis because of the lack of a shower or he developed it simply because of his exposure to the dust. In other words, either he was one of 30 who would have developed dermatitis anyway, or he was one of the additional 40 who, the epidemiological evidence suggested, would have developed it only because there were no showers. Ex hypothesi, however, general medical science is incapable of saying into which category the claimant falls. And epidemiological science is equally incapable of determining that particular question – indeed it is no part of its function to do so.

154. In that situation a court could simply say that the claimant's case failed since he had not proved that he was among the 40 who would have developed dermatitis only because there were no showers, rather than among the 30 who would have developed it even if they had showered. Alternatively, a court might say that it was more likely that the claimant's dermatitis was caused by the lack of showers. And, in fact, various courts have adopted an approach based on doubling the risk as their way of dealing with the problems of proof in toxic tort cases. As already mentioned at para 140 above, an example is the decision of the Supreme Court of Texas in *Merrell Dow Pharmaceuticals Inc v Havner* (1997) 953 SW 2d 706 which Lord Phillips discusses at paras 85-89. It should be noticed, however, that the starting-point for the court's discussion was that "epidemiological studies cannot establish the actual cause of an individual's injury or condition." The court explained the basis of its approach in this way:

> "In the absence of direct, scientifically reliable proof of causation, claimants may attempt to demonstrate that exposure to the substance at issue increases the risk of their particular injury. The finder of fact is asked to infer that because the risk is demonstrably greater in the general population due to exposure to the substance, the claimant's injury was more likely than not caused by that substance. Such a theory concedes that science cannot tell us what caused a particular plaintiff's injury. It is based on a policy determination that when the incidence of a disease or injury is sufficiently elevated due to exposure to a substance, someone who was exposed to that substance and exhibits the disease or injury can raise a fact question on causation."

The court acknowledged that it was adopting a particular policy on what counted as raising a question on causation in such circumstances. On the basis of *McGhee* Lord Mackay envisaged that in an appropriate case the House of Lords would take a somewhat similar approach.

155. Lord Mackay first suggests that in his hypothetical case the House could be taken as holding that, on the basis of the statistics, it would be reasonable to infer that there was a relationship between contraction of dermatitis in these conditions and the absence of washing facilities. Assuming that the epidemiological evidence is reliable, that is plainly so. He goes on to suggest that, on the basis of that inference, it might be reasonable to hold that the absence of washing facilities was likely to have made a material contribution to the causation of the dermatitis – by which he means the claimant's dermatitis. This is the critical step.

156. It is important to recognize that in such a case the claimant would not have proved, on the balance of probability, that his exposure to the brick dust by the defendant actually caused his dermatitis. Indeed the starting point of the entire hypothetical example is that, in the present state of medical knowledge, the claimant could not prove this. Assuming that the epidemiological study is reliable, the statistics in Lord Mackay's example would simply indicate that, if you took 100 workmen who developed dermatitis after working in the same conditions, you would expect to find that 30 developed it after having showered and 70 developed it when they had not been able to shower. So, by leading the epidemiological evidence, the only "fact" that the claimant can prove and offers to prove, on the balance of probability, is that *in most cases* the dermatitis *would have been* related to the lack of showers. So, if the judge accepts the evidence, it may legitimately satisfy him, on the balance of probability, not that the claimant's dermatitis was caused by the lack of showers, but that, in the absence of any evidence that the claimant is atypical, it is more probable than not that his dermatitis was caused by the lack of showers. In short, the chances are that it was. Whether, in any particular case, the claimant's dermatitis was actually caused by the lack of showers is a

matter of fact – and one that remains unknown, if the only available evidence is statistical. See Steve Gold, "Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence" (1986) 96 Yale LJ 376, 382-384.

157. Where the claimant led only statistical evidence, a court could simply say that his case failed. Alternatively, as Lord Mackay envisaged, the court might have held, exceptionally, that, where no other proof was possible, the defendant should be held liable on the basis of Lord Mackay's rule.

158. Of course, it is possible to conceive of a legal system which chose, as a matter of policy, to make defendants liable for all the damage which a court was satisfied, on the balance of probability, they had probably caused. But only the legislature could alter English or Scots law so as to introduce a general rule to that effect, which would change the very nature of the system and completely alter its balance, in favour of claimants and against defendants and their insurers. In *Hotson* Lord Mackay was not suggesting that English law operated, or should operate, generally on that basis. On the contrary, he had just been at pains, along with the other members of the appellate committee, to emphasise that in civil proceedings for damages the role of the judge is to decide, on the balance of probability, what actually happened. He introduced his discussion of the hypothetical case by saying, [1987] AC 750, 786A-B, that he considered that it would be unwise, however, "to lay it down as a rule that a plaintiff could never succeed by proving loss of a chance in a medical negligence case." He then referred to *McGhee*. So he seems to have envisaged that the court might adopt such an approach in an exceptional case like *McGhee* where, because of the state of medical knowledge, the claimant could not prove his case on the usual approach.

159. There is now no room, however, for Lord Mackay's rule in cases of that kind in English or Scots law since, in *Fairchild*, the House dealt with the problem of proof which they present by adopting a different and – for claimants – much less stringent rule. With Lord Mackay's rule, the claimant would succeed if he showed, on the balance of probability, that it was more likely than not that the defendant's breach of duty had materially contributed to the causation of his dermatitis; under the rule in *Fairchild*, the claimant succeeds if he shows, on the balance of probability, that the defendant's breach of duty materially increased the risk that he would contract dermatitis. Indeed, the rule in *Fairchild* is more generous to claimants precisely because it is modelled on the rule which the House had adopted in *McGhee* and which was itself more generous to pursuers than the rule described by Lord Mackay. Put shortly, if the House had applied Lord Mackay's rule, the claimants in *Fairchild* would unquestionably have failed since there was no evidence, whether epidemiological or of any other kind, to show that, on the balance of probability, it was more probable than not that the breach of duty of any of the individual defendants had materially contributed to the causation of

the victims' disease. All that the claimants could show was that, on the balance of probability, each of the defendants had materially increased the risk that the victims would develop mesothelioma. For the policy reasons which it gave, the House of Lords held that this was enough.

Single Exposure Mesothelioma Cases

160. Similarly, in my view, there is now no room for introducing the doubling of the risk approach in single exposure mesothelioma cases. As already explained, in these cases, because of the state of medical knowledge, it is impossible to prove whether the victim's mesothelioma was actually caused by the defendant's breach of duty or by asbestos fibres in the general atmosphere. The claimant comes up against the same rock of uncertainty. In that respect single exposure cases are no different from multiple defendant cases and the same approach should be applied. The point is covered by what Lord Hoffmann said in *Barker v Corus UK Ltd* [2006] 2 AC 572, 584H-585B, at para 17, in a short passage with which all the members of the appellate committee agreed:

> "The purpose of the *Fairchild* exception is to provide a cause of action against a defendant who has materially increased the risk that the claimant will suffer damage and may have caused that damage, but cannot be proved to have done so because it is impossible to show, on a balance of probability, that some other exposure to the same risk may not have caused it instead. For this purpose, it should be irrelevant whether the other exposure was tortious or non-tortious, by natural causes or human agency or by the claimant himself. These distinctions may be relevant to whether and to whom responsibility can also be attributed, but from the point of view of satisfying the requirement of a sufficient causal link between the defendant's conduct and the claimant's injury, they should not matter."

The position accordingly is that in single exposure cases the *Fairchild* exception applies and a claimant succeeds if he proves, on the balance of probability, that the defendant's breach of duty materially increased the risk that he would develop mesothelioma.

161. Since that is the rule which applies in cases where the state of medical knowledge makes it impossible for a claimant to prove whether a defendant's breach of duty actually caused his disease, there is no reason why a claimant needs to prove anything more than that the defendant's breach of duty materially increased the risk that he would develop the disease. So in such cases the doubling of the risk approach is irrelevant. And there is no room for Mr Stuart-Smith's fall-

back suggestion that, in single exposure cases, a material increase in risk should be equated with doubling the risk. That would be utterly inconsistent with the established law that, for these purposes, a risk is material if it is more than de minimis. See the discussion of the hypothetical use of statistics in *McGhee* at para 150 above.

162. It also follows that there is no room in such cases for applying the approach laid down by Smith LJ in the Court of Appeal in the passage quoted at para 121 above. The purported guidance to courts in that passage should not be followed.

163. Finally, nothing which I have said is intended to discourage the use of epidemiological evidence or to depreciate its value in cases where a claimant has to prove his case on the balance of probabilities. Far from it. Obviously, for example, epidemiology is likely to lie behind much of the evidence on which a court determines whether an exposure has materially increased the risk of the claimant developing a disease. Epidemiological evidence may also be relevant when deciding whether it would have been reasonable for a defendant to take precautions to avoid the risk of the claimant suffering a particular injury – say, the side-effect of a drug. And, of course – it must be emphasised once more – epidemiological and statistical evidence may form an important element in proof of causation. I have simply emphasised the point made by *Phipson on Evidence*,17th ed (2010), para 34-27, that, unless a special rule applies, "Where there is epidemiological evidence of association, the court should not proceed to find a causal relationship without further, non-statistical evidence." In other words, since, by its very nature, the statistical evidence does not deal with the individual case, something more will be required before the court will be able to reach a conclusion, on the balance of probability, as to what happened in that case. For example, where there is a strong epidemiological association between a drug and some condition which could have been caused in some other way, that evidence along with evidence that the claimant developed the condition immediately after taking the drug may well be enough to allow the judge to conclude, on the balance of probability, that it was the drug that caused the claimant's condition.

164. Of course, in any actual dispute, the epidemiological evidence may be contested. The judge will then have to decide which expert view he accepts and how reliable the evidence is – whether, for example, the study has been properly constructed and, in particular, what the confidence intervals are. In that respect epidemiological evidence is no different from other evidence.

Disposal

165. Since the *Fairchild* exception applies in single exposure cases, the claimants in the present appeals were entitled to succeed if they proved that the defendants' breach of duty materially increased the risk that Mrs Costello and Mrs Willmore would develop mesothelioma. There was therefore no error of law on the part of the Court of Appeal. The defendants' appeal in *Sienkiewicz* must accordingly be dismissed.

166. So far as the law is concerned, the same applies to *Willmore*. In that case the Council also appealed on the facts. The Court of Appeal reviewed the evidence and the judge's reasoning. Having rejected his finding on one point, they accepted that he had been entitled to find that she had been exposed to asbestos in two other ways and that those exposures had been material. It is important that judges should bear in mind that the *Fairchild* exception itself represents what the House of Lords considered to be the proper balance between the interests of claimants and defendants in these cases. Especially having regard to the harrowing nature of the illness, judges, both at first instance and on appeal, must resist any temptation to give the claimant's case an additional boost by taking a lax approach to the proof of the essential elements. That could only result in the balance struck by the *Fairchild* exception being distorted. Mr Feeny made a number of plausible criticisms of the findings of Nicol J and of the approach of the Court of Appeal and suggested that they had been unduly favourable to Mrs Willmore. Some of the inferences which Nicol J drew in Mrs Willmore's favour from the evidence relating to her exposure at Bowring Comprehensive can properly be regarded as very generous. With considerable hesitation, however, I have concluded that the criticisms would not justify this Court in taking the exceptional step of disturbing the concurrent findings of fact of the courts below. I would accordingly dismiss the Council's appeal on the facts. In the result, the appeal in *Willmore* must also be dismissed.

LADY HALE

167. I pity the practitioners as well as the academics who have to make sense of our judgments in difficult cases. But these cases are hard rather than difficult. We are here concerned with one case of relatively light but long term exposure and one case of very slight and short term exposure, both set against a lifetime of environmental and other possible exposures about which nothing much is known. As Lord Brown implies, *Fairchild* kicked open the hornets' nest. The House of Lords were confronted with several employers, each of which had wrongly exposed their employees to asbestos, but none of which exposure could be shown to have caused the disease. I find it hard to believe that their Lordships there

foresaw the logical consequence of abandoning the "but for" test: that an employer or occupier whose wrongful exposure might or might not have led to the disease would be liable in full for the consequences even if it was more likely than not that some other cause was to blame (let alone that it was not more likely than not that he was to blame). But, as Lord Rodger has explained, that is the logical consequence and there is nothing we can do about it without reversing *Fairchild*. Even if we thought it right to do this, Parliament would soon reverse us, and it is easy to understand why. Asbestos has long been known to be a dangerous (as well as a useful) substance, employers and occupiers turned a blind eye to those dangers long after they knew or should have known about them, and mesothelioma is a dreadful disease.

168. In *Barker*, Mr Stuart-Smith tried very hard to persuade the House of Lords that the *Fairchild* exception applied only where all the exposure was in breach of duty. He failed in that, although he succeeded in persuading the majority that the price to be paid for abandoning conventional rules of causation was aliquot liability. Parliament swiftly disagreed. The Compensation Act 2006 restored the principle that any tortfeasor is liable in full for an indivisible injury. But that leaves us with the result that a defendant who may very well not have caused the claimant's disease – indeed probably did not do so - is fully responsible for its consequences. I do not see any answer to that. It is the inevitable result of *Barker*, made even more severe through the intervention of Parliament, but inevitable none the less.

169. That means that in cases where the *Fairchild* exception applies, there is no room for the "more than doubles the risk" approach to causation: it is not necessary in order to establish causation and it is not an appropriate test of what is a more than de minimis increase in risk. So we do not need to go into the relevance of statistical probabilities to the finding of causation for the purpose of deciding these cases. Nor, in the event, did the Court of Appeal need to do so. The reason why Lord Phillips and Lord Rodger have discussed the subject at such length is the obiter observation of Smith LJ, at para 23 of her judgment in *Sienkiewicz*, that "in a case of multiple potential causes, a claimant can demonstrate causation by showing that the tortious exposure has at least doubled the risk arising from the non-tortious cause or causes." Anything we say on the subject, therefore, is also obiter.

170. However, I do agree with Lord Rodger that doubling the risk is not an appropriate test of causation in cases to which the *Fairchild* exception does not apply. Risk is a forward looking concept – what are the chances that I will get a particular disease in the future? Causation usually looks backwards – what is the probable cause of the disease which I now have? Epidemiology studies the incidence and prevalence of particular diseases and the associations between both of these and particular variables in the diseased population. From these it is

possible to predict that a particular percentage of the population, for example of women aged between 60 and 70, will contract a particular disease, for example, breast cancer. It is also possible to say that certain variables, such as life-style or age of first child-bearing, are associated with a greater chance of developing the disease. So a doctor will sensibly advise her patient to behave in a way which will reduce the risks. But if the disease materialises, the existence of a statistically significant association between factor X and disease Y does not prove that in the individual case it is more likely than not that factor X caused disease Y.

171. The same applies to less sophisticated calculations. The fact that there are twice as many blue as yellow taxis about on the roads may double the risk that, if I am run over by a taxi, it will be by a blue rather than a yellow one. It may make it easier to predict that, if I am run over by a taxi, it will be by a blue rather than a yellow one. But when I am actually run over it does not prove that it was a blue taxi rather than a yellow taxi which was responsible. Likewise, if I actually develop breast cancer, the fact that there is a statistically significant relationship between, say, age at first child-bearing and developing the disease does not mean that that is what caused me to do so.

172. But as a fact finder, how can one ignore these statistical associations? Fact-finding judges are told that they must judge a conflict of oral evidence against "the overall probabilities" coupled with the objective facts and contemporaneous documentation: see, for example, Robert Goff LJ in *Armagas Ltd v Mundogas SA (The "Ocean Frost")* [1985] 1 Lloyd's Rep 1, 57. Millions of pounds may depend upon their decision. Yet judges do not define what they mean by "the overall probabilities" other than their own particular hunches about human behaviour. Surely statistical associations are at least as valuable as hunches about human behaviour, especially when the judges are so unrepresentative of the population that their hunches may well be unreliable? Why should what a (always middle-aged and usually middle class and male) judge thinks probable in any given situation be thought more helpful than well-researched statistical associations in deciding where the overall probabilities lie? As it seems to me, both have a place. Finding facts is a difficult and under-studied exercise. But I would guess that it is not conducted on wholly scientific lines. Most judges will put everything into the mix before deciding which account is more likely than not. As long as they correctly direct themselves that statistical probabilities do not prove a case, any more than their own views about the overall probabilities will do so, their findings will be safe.

173. So in my view it would be wrong for judges to change their fact-finding behaviour because of anything said in this case. On the issues of law, the *Fairchild* exception has to apply to these single tortious exposure cases, no matter how unjust it may seem to the defendants. Even if I were convinced of the merits of the "more than doubling the risk" approach to causation in other contexts, which I am

not, it does not apply in these cases. That is enough to dispose of the appeal in the case of the late Mrs Costello. In the case of Mrs Willmore, the judge's findings of fact were truly heroic, and I would endorse what Lord Rodger says about this, but I do not think that it is open to us to disturb them. I would dismiss both appeals.

LORD BROWN

174. Mesothelioma claims are in a category all their own, so special indeed that Parliament in 2006 chose to legislate specifically for them: section 3 of the Compensation Act 2006. Whilst entertaining no doubt that the position now reached in respect of such claims is precisely as Lord Phillips and Lord Rodger have explained and that these appeals must accordingly fail, I think it only right to indicate just how unsatisfactory I for my part regard this position to be and how quixotic the path by which it has been arrived at.

175. The present position, exemplified by the facts of these very appeals, can be simply stated as follows: any person who negligently or in breach of duty exposes another more than minimally to the inhalation of asbestos fibres will be liable to make full compensation if that other develops mesothelioma more than five years later (five years being now thought to be the minimum period between the development of the first malignant cell and the diagnosis of the disease – see Lord Phillips' judgment at para 19(v)). That statement of the position holds true irrespective of whether the victim was exposed by others to even longer and more intensive inhalation (and indeed inhalation of more noxious fibres), whether negligently or not, and irrespective too of any environmental or other exposure (again, however intensive). It requires qualification only if and to the extent that the victim negligently exposed himself to the inhalation of asbestos fibres (when there may be a finding of contributory fault).

176. One need hardly stress how radically different such an approach to compensation represents from that followed in all other cases of physical injury. All other cases require that the claimant satisfies the "but for" test of causation. True, in the case of cumulative injuries, the law holds a negligent employer liable even if his negligence is responsible for part only of the victim's condition (provided only that it made a material, ie more than de minimis, contribution to the development of the condition). I have difficulty, however, in seeing this as a true exception to the "but for" test: although the claimant in *Bonnington Castings Ltd v Wardlaw* [1956] AC 613, the case which first established the principle, recovered full damages for his condition (pneumoconiosis from the inhalation of silica), that appears to have been because the defendants took no point on apportionment; in a series of subsequent such cases damages have been apportioned, however broadly – for example, as between negligent and non-negligent exposure respectively in

dust inhalation cases, in noise cases and in cases of vibration white finger, and, in respiratory disease cases, between the damage caused by the inhalation of fumes or other noxious agents on the one hand and the claimant's habit of cigarette smoking on the other.

177. It therefore seems to me that there is just one single authority that needs to be noticed before one turns to the three stage process by which the present approach to compensation in mesothelioma cases came to be dictated, namely, of course, *McGhee v National Coal Board* [1973] 1 WLR 1. *McGhee* is undoubtedly a problematic case. The House of Lords was later in *Wilsher v Essex Area Health Authority* [1988] AC 1074 to regard it as not having laid down any principle of law at all; rather it was described by Lord Bridge of Harwich, at p 1090, as merely "a robust and pragmatic approach to the undisputed primary facts of the case" – on the basis that, as in *Bonnington Castings,* the negligent prolongation of the claimant's contact with (in *McGhee*) brick dust had materially contributed to his development (in *McGhee*) of dermatitis.

178. Rightly or wrongly, however (and whether rightly or wrongly now matters nothing), the House of Lords in *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 found altogether greater force in *McGhee.* As was pointed out, for example by Lord Nicholls, it had really not been open to the House in *McGhee* to infer from the established facts that the employer's negligence had caused or materially contributed to the onset of his condition. In short, the House in *Fairchild* regarded *McGhee* as authority for the application to certain cases of a less stringent test than the usual "but for" test for establishing the necessary causal connection between the employer's negligence and the claimant's condition. That said, however, the judgments in *Fairchild* provided no support whatever for a general principle of compensation in mesothelioma cases remotely as wide as I have described the present position to be today. Quite the contrary. The circumstances in which the more relaxed approach to causation said to have been adopted in *McGhee* were held to apply to mesothelioma cases were narrowly circumscribed. One should note particularly Lord Bingham's six relevant factors (conveniently set out at para 39 of Lord Phillips' judgment), all of which had to be present before the special rule of causation was to apply. Note too the agreement between the parties in *Fairchild* that "any cause of [the claimant's] mesothelioma other than the inhalation of asbestos dust at work can be effectively discounted" (Lord Bingham's speech at para 2). Consider also the rationale identified by Lord Bingham as justifying this special rule: the "strong policy argument in favour of compensating those who have suffered grave harm, at the expense of their employers who owed them a duty to protect them against that very harm and failed to do so, when the harm can only have been caused by breach of that duty and when science does not permit the victim accurately to attribute, as between several employers, the precise responsibility for the harm he has suffered" (Lord Bingham at para 33). Lord Bingham was there positing a situation where, for example, a

mesothelioma victim had worked for three successive employers each, say, for fifteen years, all of whom had negligently exposed him to the inhalation of asbestos fibres. Faced with the "rock of uncertainty" – Lord Bingham's graphic characterisation of science's inability to establish on a balance of probabilities which particular source(s) of asbestos fibre exposure had caused mesothelioma to develop – one can readily see how the House came unanimously to endorse this new principle.

179. I am not, of course, suggesting that their Lordships in *Fairchild* were intent on confining the application of this new principle quite so narrowly as that. Lord Rodger, for example, expressly recognised (at para 170 of his speech) that "it can also apply where, as in *McGhee*, the other possible source of the injury is a similar, but lawful, act or omission of the same defendant". But he immediately then "reserve[d] [his] opinion as to whether the principle applies where the other possible source of injury is a similar but lawful act or omission of someone else or a natural occurrence". The point I make is that it is hardly to be thought that had the House, on the occasion of the *Fairchild* hearing, been considering not the facts of those three appeals but instead the facts of the present appeals the claimants would have succeeded and the law have developed as it has.

180. Before parting from *Fairchild* it is, I think, worth noting that, just as in *Bonnington Castings* half a century before, the respondent defendants in *Fairchild* similarly took no point on apportionment: their stance now as then was one of all or nothing - doubtless in the hope (and perhaps even the expectation) of defeating the claims in their entirety.

181. Coming then to stage two of the three stage process, by which the present position with regard to mesothelioma cases came to be established, *Barker v Corus UK Ltd* [2006] 2 AC 572, one finds the House having to face up to some of the problems it had left open with *Fairchild* and, as it seems to me, beginning to have second thoughts both as to the juristic basis for this special rule of causation which *Fairchild* held to apply in certain toxic tort cases and as to where the abandonment of the "but for" principle was taking the law. In the result, the *Fairchild* approach was (as Lord Rodger now puts it at para 140 of his judgment) "refined"; Lord Hoffmann explained that *Fairchild* had recognised a new tort, that of negligently increasing the *risk* of personal injury (although, of course, the injury had to eventuate before any tort was committed), and logically it followed that any liable defendant should be liable only for his aliquot share of the victim's loss, not for its entirety. The damage was no longer to be treated as the indivisible mesothelioma but rather as the readily divisible creation of the risk of developing mesothelioma. Damages, therefore, were to be apportioned according to the contribution made by any particular defendant to the overall risk. On that basis, of course, the special rule whereby the "but for" test of causation is relaxed applies equally whether or not other exposures are partly tortious and partly non-tortious, or indeed wholly

non-tortious, and whether they result from natural causes or indeed, from the employee's own negligence.

182. It is to my mind quite clear that the preparedness of the majority of the court in *Barker* to extend the reach of the *Fairchild* principle this far was specifically dependent upon there being aliquot liability only. Lord Rodger alone thought that liability under the *Fairchild* exception to the "but for" rule should be for full compensation ("in solidum"). But he made clear that had that been the view of the majority, then in a case where the victim had himself been solely responsible for a material exposure – especially where, as in one of the three appeals before the court in *Barker*, the victim had himself been at fault – he would have applied the normal "but for" rule for proof of causation.

183. The third and final stage of the process by which the law with regard to compensation in mesothelioma cases came to reach its present position was, of course, Parliament's enactment of section 3 of the Compensation Act 2006. I have no doubt that Lord Rodger is right (at paras 131 and 132 of his judgment) in saying that the sole effect of section 3 is to reverse the House's decision in *Barker* on the issue of quantum; in no way does it pre-empt or dictate the proper approach of the common law to questions of causation and liability. On the other hand it would be a remarkable thing for this Court now in effect to reverse the decision in *Fairchild* and revert, in mesothelioma cases as in all others, to the normal, "but for", rule of causation – the principle, vindicated periodically down the years in cases of indivisible no less than of cumulative injury (*Gregg v Scott* [2005] 2 AC 176 being the latest such decision in point), that to establish liability the claimant must show that but for the defendant's negligence he would probably not have suffered his injury (or at least not have suffered it to the full extent that he has).

184. In my judgment it could only be by reversing *Fairchild* and allowing no exception whatever to the normal rule of causation that this Court could now avoid what Lord Phillips (at para 58 of his judgment) rightly describes as the "draconian consequences" of coupling section 3 to the *Fairchild/Barker* principle: the liability in full even of someone "responsible for only a small proportion of the overall exposure of a claimant to asbestos dust". There is in my opinion simply no logical stopping place between the case of successive negligent employers dealt with in *Fairchild* itself (apparently circumscribed though that decision was) and the extreme ("draconian") position now arrived at, well exemplified as it seems to me by the facts of these very appeals. If, because of the "rock of uncertainty", the law is to compensate by reference to negligence which merely increases the *risk* of such injury as then develops, why should not that relaxation of the normal rule of causation apply equally when, as here, there is but one negligent employer (or negligent occupier) as when there are several? As *Barker* recognised, there can be no rational basis for confining the special rule within narrow bounds, whatever may have been contemplated by the House in *Fairchild*.

185. In short, the die was inexorably cast in *Fairchild* – although, as already suggested, it is doubtful if that was then recognised and it is noteworthy too that, even when in *Barker* it came to be recognised, it was then thought palatable only assuming that compensation was going to be assessed on an aliquot basis. Parliament, however, then chose – although, of course, only in mesothelioma cases – to go the whole hog.

186. The result must surely be this. As I began by saying, mesothelioma cases are in a category all their own. Whether, however, this special treatment is justified may be doubted. True, as Lord Phillips observes at the outset of his judgment, mesothelioma is indeed a hideous disease. (And it is perhaps also the case, as Lord Phillips suggests at para 104, that mesothelioma, after all, may result from the cumulative effect of exposures to asbestos dust.) The unfortunate fact is, however, that the courts are faced with comparable rocks of uncertainty in a wide variety of other situations too and that to circumvent these rocks on a routine basis - let alone if to do so would open the way, as here, to compensation on a full liability basis – would turn our law upside down and dramatically increase the scope for what hitherto have been rejected as purely speculative compensation claims. Although, therefore, mesothelioma claims must now be considered from the defendant's standpoint a lost cause, there is to my mind a lesson to be learned from losing it: the law tampers with the "but for" test of causation at its peril.

187. There is a rough justice about the law of personal injury liability as a whole. To compensate a claimant in full for a lost finger because there was a 60:40 chance that he would have worn protective gloves had they been made available to him may be regarded as rough justice for defendants. But it is balanced by the denial of compensation to a claimant who cannot establish that he would probably have worn the gloves – or whose finger the judge concludes was probably already doomed because of frostbite. Save only for mesothelioma cases, claimants should henceforth expect little flexibility from the courts in their approach to causation. Since *Fairchild* and *Barker* there has been much academic focus on a supposedly critical distinction between so-called "single agent" and "multiple agent" cases, the suggestion being that the former more readily lend themselves to special rules of causation than the latter. For my part I have difficulty even in recognising the distinction between these categories, at any rate in some cases. But I have greater difficulty still in accepting that the courts should now, whether on this or any other basis, be thinking of creating any further special rules regarding the principles governing compensation for personal injury. The same logic which requires that the claims of these respondents succeed to my mind requires also that the courts should in future be wary indeed before adding yet further anomalies in an area of law which benefits perhaps above all from clarity, consistency and certainty in its application.

LORD MANCE

188. Cases of mesothelioma are subject to the special rule of causation established in *Fairchild v Glenhaven Funeral Services Ltd* [2002] UKHL 22; [2003] 1 AC 32 and developed in *Barker v Corus UK Ltd* [2006] UKHL 20; [2006] 2 AC 572, but significantly amended by the Compensation Act 2006. I agree that this special rule is applicable to both the appeals before this court, although in each (a) only one person (an employer in one case, a school in the other) is shown to have exposed the victim of mesothelioma to asbestos, the only other such exposure being the general low-level atmospheric exposure incurred by members of the public at large, and (b) the exposure by that person did no more than increase the sufferer's general low-level atmospheric exposure to asbestos "materially" (or, more specifically, in the case of Mrs Costello represented by Mrs Sienkiewicz, by some 18%). The submission that causation can be shown by proving a doubling of the ambient risk, or can be negatived by disproving this, is inconsistent with, or would make a radical and uncertain inroad into, the special rule.

189. I reach this conclusion in agreement with the reasoning on this aspect of Lord Phillips, Lord Rodger, Lady Hale and Lord Dyson, on the basis that our understanding of the aetiology of mesothelioma remains as incomplete and inadequate as ever. I also concur with the further remarks of Lady Hale in her first paragraph and of Lord Brown in his judgment about the impossibility of going back on *Fairchild*, as well as on the lesson of caution that the history may teach in relation to future invitations to depart from conventional principles of causation. I too would therefore dismiss the appeal in Mrs Costello's case.

190. An interesting debate has, somewhat unexpectedly, developed about the significance or value of epidemiological or statistical evidence relating to a population or group in the context of decision-making in particular cases. I share a reluctance to place too much weight on such evidence. This is not because statistics are lies, or because truth can be stranger than fiction. It is because the law is concerned with the rights or wrongs of an individual situation, and should not treat people and even companies as statistics. Despite the intense sympathy which can arise in particular cases like the present, an attribution of liability based substantially on statistical evidence, that, viewing the relevant population or group as a whole, it is more likely than not that the particular defendant was negligent or causatively responsible, appears to me most undesirable.

191. That epidemiological evidence used with proper caution, can be admissible and relevant in conjunction with specific evidence related to the individual circumstances and parties is, however, common ground and clearly right. What

significance a court may attach to it must depend on the nature of the epidemiological evidence, and of the particular factual issues before the court.

192. Whether and if so when epidemiological evidence can by itself prove a case is a question best considered not in the abstract but in a particular case, when and if that question arises. If it can, then, I would hope and expect that this would only occur in the rarest of cases.

193. In other cases, there will be continuing good sense in the House of Lords' reminder to fact-finders in *Rhesa Shipping Co SA v Edmunds (the "Popi M")* [1985] 1 WLR 948 that it is not their duty to reach conclusions of fact, one way or the other, in every case. There are cases where, as a matter of justice and policy, a court should say that the evidence adduced (whatever its type) is too weak to prove anything to an appropriate standard, so that the claim should fail.

194. The American material which we have seen, particularly *Smith v Rapid Transit Inc* (1945) 58 NE 754, *Merrell Dow Pharmaceuticals Inc v Havner* (1997) 953 SW 2d 706 and "Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence" by Steve Gold (1986) 96 Yale LJ 376, demonstrates, with innumerable further references, the detailed and extensive thought which has been given across the Atlantic to the significance and use of epidemiological or statistical evidence. In that light and without hearing fuller argument, as well as because it raises fact-specific issues and is unnecessary for the resolution of these appeals, I think it inappropriate to say more about the use of epidemiological evidence.

195. On the material before us, I would myself see *Willmore v Knowsley Metropolitan Borough Council* as a case where there was no sufficient proof that the defendant exposed the claimant to asbestos. The judge found exposure on a slender and speculative basis which Lady Hale describes as heroic. But, the concurrent findings below on two of the three bases of exposure found by the judge are entitled to some weight, and on that basis I do not dissent from the general view that the appeal on fact in *Willmore* should also be dismissed.

LORD KERR

196. What has been called "the *Fairchild* exception" was described in a variety of ways in *Barker v Corus UK Ltd* [2006] 2 AC 572 but common to all the various formulations is the proposition that where employers through breach of duty expose their employee to asbestos and thereby materially increase the risk to the employee of developing mesothelioma, they will be jointly and severally liable if

he or she develops that condition. This involved a modification of the previously applicable legal rules in relation to the causation element in employers' liability claims. That alteration was thought necessary in order to cater for the particular difficulties that asbestos related disease presents.

197. Implicit in the modification of the normal rule is the acceptance that an employer thus found liable may, in truth and in fact, not have been responsible for the damage at all. This is the price that it was deemed necessary to pay in order to hold the balance of justice between the parties. Because of the limitations of medical and scientific knowledge, it was recognised that it would be unjust to enforce a rigorous requirement of proof that a particular employment had actually caused or contributed to the damage.

198. A potent factor in this equation was that the insidious nature of asbestos and the calamitous consequences that exposure to it can cause, allied to the current lack of scientific knowledge about the aetiology of mesothelioma, warrant a different approach to the conventional burden of proof. To insist on its stringent application would set what would in many instances prove an impossible practical difficulty in the way of a claimant. These considerations – *viz* the constraints that arise from the unavailability of scientific proof and the dreadful illnesses that can result from asbestos exposure – are just as relevant in the approach to so-called "single exposure" cases as they are in cases of multiple employment exposure cases.

199. The use of the expression "single exposure" may be misleading in this context. In *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 the defendants had argued that the claims should be dismissed because there were various exposures each of which could have caused the mesothelioma and each of which might not have done so. In the present cases the appellants' argument resolves to essentially the same proposition. They suggest that there were two possible sources of exposure in each case – in Mrs Costello's case exposure while employed by the defendant and environmental exposure and in Mrs Willmore's case exposure while at school and environmental exposure. It is argued that each of these exposures might have caused the mesothelioma but each of them might not have done so. In effect, therefore, the appellants submit that there is more than one possible source for the mesothelioma that both women suffered. The difference in these cases is not that they involved a single exposure but that each had a tortious and a non-tortious source of exposure. But the same difficulties as to proof as arose in *Fairchild* and *Barker* afflict the present cases. And it was those difficulties that prompted the modification of the causation rules.

200. It might be suggested that it is easier to accept that several employers, none of whom could be positively identified as having caused or contributed to the condition, should have to participate in the compensation package, on the basis

that one of them (at least) had actually caused the mesothelioma and because each of the employers had, in any event, been prepared to have their employee run the risk of contracting the disease. But that is not the basis on which the adjustment to the requirements of proof was made. That adjustment was made precisely because, as a matter of policy, it was considered that it would be unfair to impose on a claimant a requirement of proof which in most cases, because of the limitations of scientific knowledge, was quite incapable of fulfilment. In so far as such considerations might be considered relevant, however, the fact remains that both defendants in the present appeals were prepared to countenance a material increase in the risk to Mrs Costello and Mrs Willmore. The circumstance that the other possible source of mesothelioma in these cases was non-tortious should make no difference.

201. Nor did it in *Barker*. In that case it was expressly accepted by Lord Hoffmann, Lord Scott of Foscote and Lord Rodger of Earlsferry that the *Fairchild* exception did apply to a "non-tortious source of risk". At para 17 Lord Hoffmann said:

> "The purpose of the *Fairchild* exception is to provide a cause of action against a defendant who has materially increased the risk that the claimant will suffer damage and may have caused that damage, but cannot be proved to have done so because it is impossible to show, on a balance of probability, that some other exposure to the same risk may not have caused it instead. For this purpose, it should be irrelevant whether the other exposure was tortious or non-tortious, by natural causes or human agency or by the claimant himself. These distinctions may be relevant to whether and to whom responsibility can also be attributed, but from the point of view of satisfying the requirement of a sufficient causal link between the defendant's conduct and the claimant's injury, they should not matter."

202. Lord Scott stated that he was in "complete agreement" not only with Lord Hoffmann's conclusions but also with his reasons for reaching them (para 50) and at para 97 Lord Rodger said:

> "Starting from 'the *McGhee* extension', counsel considered whether *Fairchild* would apply where one or more of the sources of exposure to asbestos dust had been lawful but unconnected with any wrongdoer. For instance, the victim had been employed for a period before the dangers of exposure to asbestos dust should have been known in the industry and there had been no fault on the part of the employer. Having reserved my opinion on the point in *Fairchild*, I would now hold that the rule should apply in that situation."

203. For the reasons given by Lord Phillips and Lord Rodger in the present appeals, therefore, I agree that there is no basis on which the *Fairchild* exception should not be applied in these cases and, on that account, that the appeals should be dismissed. The policy reason for introducing the modified rule in that case applies with equal force here and it would be anomalous and arbitrary to require these respondents to establish that it was twice as likely that the indicted exposure was the cause of the mesothelioma, while not imposing such a requirement on a claimant in a multiple employer exposure case. In all relevant respects the appellants are in an exactly similar position to a defendant in such a case. In both instances none of the defendants can be proved to have caused the mesothelioma but all have materially increased the risk by wrongfully exposing Mrs Costello and Mrs Willmore to asbestos.

204. In these circumstances the interesting debate that has been had between Lord Phillips and Lord Rodger as to the use to which epidemiological evidence might be put is, at this stage certainly, academic. But I wish to say that I share the misgivings that have been expressed about the capacity of this type of evidence to prove that mesothelioma is more likely to have been caused by a particular exposure, even if advances in medical and scientific knowledge erode the "rock of uncertainty".

205. Epidemiology is the branch of medical science which normally deals with the incidence and prevalence of disease in large populations and with the detection of the sources and causes of disease. It involves the collection of data, usually over significant periods. Unless these coincide with periods of relevant exposure or replicate conditions of exposure experienced by individual claimants, the use of such data to seek to establish any specific proposition in an individual case requires to be treated with great caution, in my opinion. It is an essential and minimum requirement, as Brachtenbach J said in *Herskovits v Group Health Cooperative of Puget Sound* (1983) 664 P 2d 474, that there be evidence connecting avowedly relevant statistical information produced by the epidemiological studies to the facts of the case. In my view, no such connection was made in the present appeals. The "epidemiological evidence" which was adduced consisted of a series of assumptions and speculations rather than actual data which could be related to the experience of those who developed mesothelioma. What the testimony amounted to was the promotion of a theory rather than the establishment of facts and it did not constitute evidence on which reliable conclusions could be reached.

206. There is a real danger that so-called "epidemiological evidence" will carry a false air of authority. It is necessary to guard against treating a theory based on assumptions as a workable benchmark against which an estimate of the increase in risk could be measured. Whether and in what circumstances epidemiological evidence can assist in the determination of whether a particular case of

mesothelioma is likely to have been caused by a particular exposure will have to be decided according to the particular circumstances of an individual case. In my view, the epidemiological material adduced in evidence in the present case could not have assisted in the determination of that issue.

LORD DYSON

207. The central question that arises in these appeals is whether the so-called *Fairchild* exception applies in a "single exposure" case, that is to say a case where a victim has been exposed to asbestos by a single defendant in breach of duty and has also been exposed to asbestos in the general atmosphere. In *Fairchild* itself, the victims had been exposed to asbestos by a number of defendants in breach of their duty of care. The limitations of medical knowledge prevented them from being able to prove on the balance of probability which exposure had caused their mesothelioma. In order to avoid injustice, the House of Lords held that proof on the balance of probability that each defendant's wrongdoing had materially increased the risk of contracting the disease was sufficient to satisfy the causal requirements for liability. For understandable reasons, the Court of Appeal had applied a conventional approach and had dismissed the claims because the claimants had been unable to prove on the balance of probability that their wrongful exposure to asbestos by any particular defendant had caused their disease. Each defendant was able to say that the offending asbestos might have been the result of exposure caused during the claimants' employment by a different defendant. Thus it was that the claims were rejected by the Court of Appeal on what Lord Bingham called "this rock of uncertainty".

208. The *Fairchild* exception was created to circumvent the rock of uncertainty. It is implicit in the reasoning in *Fairchild* (repeated in *Barker*) that, if the rock of uncertainty were to disappear in the light of increased medical knowledge, then the rationale for the *Fairchild* exception would disappear and claimants would be required to prove their cases on the balance of probability in the usual way. It is common ground that medical knowledge about the aetiology of mesothelioma has not materially advanced since *Fairchild.* Mr Stuart-Smith QC accepts that, if this were a multiple exposure case, the claimants would not be required to prove on the balance of probability (whether by the doubling of the risk test or otherwise) that their mesothelioma had been caused by wrongful exposure to asbestos. All that they would have to prove was that the defendant or defendants had materially contributed to the risk of mesothelioma.

209. There has been no previous decision on a single exposure case. In *Barker,* the House of Lords held that the *Fairchild* exception applied even where not all the exposures to asbestos which could have caused the claimant employee's

mesothelioma involved breaches of duty by his employers (in that case, the employee was also exposed to asbestos during a period when he was self-employed). At para 17, Lord Hoffmann said that the purpose of the *Fairchild* exception was:

> "to provide a cause of action against a defendant who has materially increased the risk that the claimant will suffer damage and may have caused that damage, but cannot be proved to have done so because it is impossible to show, on a balance of probability, that some other exposure to the same risk may not have caused it instead. For this purpose, it should be irrelevant whether the other exposure was tortious or non-tortious, by natural causes or human agency or by the claimant himself. These distinctions may be relevant to whether and to whom responsibility can also be attributed, but from the point of view of satisfying the requirement of a sufficient causal link between the defendant's conduct and the claimant's injury, they should not matter."

210. Lord Scott expressed the same view at para 59. But *Barker* was not a single exposure case.

211. So why should the *Fairchild* exception not be applied in a single exposure case? Mr Stuart-Smith advances a number of reasons. He submits that there is no suggestion in any previous case that exposure to asbestos in the general atmosphere should be taken into account as a relevant exposure for the purposes of the *Fairchild* exception. The breathing of ambient air, which should merely be regarded as part of the ordinary vicissitudes of life, is not under the control of any single person or group of persons and should not be treated in the same way as exposures to a carcinogen controlled and caused by an identifiable individual.

212. In my view, these are not good reasons for disapplying the *Fairchild* exception in a single exposure case. In view of the present state of medical knowledge, a single exposure claim would founder on the same rock of uncertainty as a multiple exposure claim. The exception was devised as a matter of policy to overcome the injustice that claimants would suffer if they were prevented by the rock of uncertainty from establishing causation in mesothelioma cases. This policy justification for the exception is articulated in a number of the speeches in both *Fairchild* and *Barker*: see, for example, per Lord Bingham at para 33 and Lord Nicholls at paras 41 and 42 in *Fairchild.* There is no reason in policy or principle why the exception should not apply to a single exposure claim just as it does to a multiple exposure claim. It is true that none of the previous decisions involves a single exposure claim. But that is not a good reason for refusing to apply the *Fairchild* exception if there is no material difference between single and multiple

exposure claims. It is also true that the breathing of ambient air is a vicissitude of life. But that is not a good reason for distinguishing *Fairchild* either. On the present state of medical knowledge, the rock of uncertainty is as much of a problem for victims of single exposure as for victims of multiple exposure.

213. It is implicit in *Fairchild* and *Barker* that, if it were possible for a victim of mesothelioma to establish causation on the balance of probability in the conventional way, then the rationale for the *Fairchild* exception would disappear. Mr Stuart-Smith submits that causation can be established in the conventional way in a single exposure case (but, he accepts, not yet in a multiple exposure case). He says that a claimant can prove causation on a balance of probability by proving that the tortious exposure has at least doubled the risk arising from the non-tortious cause. This was the approach adopted by Judge Main in *Sienkiewicz* and adopted as a correct statement of the law by Smith LJ at para 23 of her judgment. In fact, Smith LJ seems to have considered that it was a legitimate approach even in multi-exposure cases, since she referred to a doubling of the risk arising from "the non-tortious cause *or causes"* (emphasis added).

214. Lord Phillips and Lord Rodger are in agreement that there is no scope in single exposure mesothelioma cases for the application of a doubling of the risk test based *entirely* on epidemiological evidence. But their reasoning differs to some extent. Lord Phillips considers that it is not possible to prove causation on the basis of epidemiological evidence alone because first it is not sufficiently reliable (paras 97 to 101), and secondly there continue to be gaps in our understanding of the aetiology of mesothelioma (paras 102 to 105). If these shortcomings in our understanding were made good, then it is implicit in the first reason that, if epidemiological data were to become sufficiently reliable, victims of mesothelioma would be able (and therefore required) to prove causation on the balance of probability on the basis of epidemiological evidence alone.

215. Lord Rodger agrees with Lord Phillips's second reason. But his objection to proof on the basis of epidemiological evidence alone is not based on the unreliability of epidemiological data. It is more fundamental than that.

216. Lord Rodger draws a distinction between claimant A, who proves on the balance of probability that a defendant *probably* injured him, and claimant B, who proves on the balance of probability that a defendant *actually* injured him. He says that, as a matter of law, claimant B will succeed but claimant A will fail. A claimant who seeks to prove his case on the balance of probability in reliance entirely on statistical evidence will inevitably fail, since he is able to do no more than prove on the balance of probability that the defendant probably injured him.

217. I am grateful to Lord Rodger for drawing attention to the article by Steve Gold, "Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence" (1986) 96 Yale LJ 376. The article distinguishes between "fact probability" and "belief probability". The former is a more than 50% statistical probability of an event having occurred. An illustration of this is the 75% probability that the victim was run down by a blue cab in the example given by Brachtenbach J in *Herskovits v Group Health Cooperative of Puget Sound* (1983) 664 P 2d 474 (see para 95 of Lord Phillips's judgment). The latter is a more than 50% *belief* in the decision-maker that a knowable fact has been established. Mr Gold points out that, particularly in toxic tort cases, US courts have often "collapsed" the distinction between fact probability and belief probability and simply asked the question whether the fact that the claimant seeks to prove has been established as "more likely than not".

218. In my view, this is an important distinction and it is of particular relevance in relation to causation in toxic torts. It is often the basic impossibility of proving individual causation which distinguishes toxic tort cases from ordinary personal injury cases. As Mr Gold points out, epidemiology is based on the study of populations, not individuals. It seeks to establish associations between alleged causes and effects. With proper scientific interpretation, these correlations lend great weight to an inference of causation. However, in an individual case, epidemiology alone cannot *conclusively* prove causation. At best, it can establish only a certain probability that a randomly selected case of disease was one that would not have occurred absent exposure.

219. Ultimately, questions of burden and standard of proof are policy matters for any system of law. It is trite law that our system requires a civil claim to be proved by a claimant on the balance of probability. It is a matter of policy choice whether and, if so, in what circumstances the courts are willing to find causation proved on the balance of probability on the basis of epidemiological evidence alone. In the United States, some courts have been willing to find causation established on the balance of probability on the basis of epidemiological evidence alone. They have been criticised by Mr Gold for collapsing the distinction to which I have referred.

220. As I have said, the House of Lords produced in the *Fairchild* exception a particular policy response to the causation problems created by the lack of scientific knowledge about the aetiology of mesothelioma. This response has been confirmed by the 2006 Act. In these circumstances, I agree with Lord Phillips and Lord Rodger that there is no room for the application of a different test which would require a claimant to prove (whether on the basis of doubling of the risk or otherwise) that on the balance of probability the defendant caused or materially contributed to the mesothelioma.

221. It follows that I do not find it necessary to decide whether there are any circumstances in which, as a matter of English law, causation can be proved on the basis of epidemiological evidence alone. I am unaware of any English authority in which the question whether causation can be proved in a straightforward personal injury case on the basis of epidemiological evidence alone has been the subject of decision. Toxic torts, such as mesothelioma, give rise to particular causation problems. That is why special rules sometimes have been devised so as to avoid injustice. Such problems are not inherent in straightforward personal injury cases where it must be rare for a claimant to rely exclusively on epidemiological evidence to prove his or her claim. The claimant will almost always also be able to point to some specific evidence relating to the particular circumstances of the case. I note that in *Smith v Rapid Transit Inc* (1945) 317 Mass 469, 58 NE 2d 754 it was held on the facts of that case that statistical likelihood alone was insufficient to support a finding that the bus that injured the plaintiff was the defendant's.

222. But ultimately, as I have said, it is not necessary for the resolution of the present appeal to decide whether epidemiological evidence alone suffices, since Lord Phillips and Lord Rodger are agreed that there has been no material change in the understanding of the aetiology of mesothelioma and there is no basis for distinguishing single exposure cases from multiple exposure cases. It seems to me, however, that there is no *a priori* reason why, if the epidemiological evidence is cogent enough, it should not be sufficient to enable a claimant to prove his case without more. Our civil law does not deal in scientific or logical certainties. The statistical evidence may be so compelling that, to use the terminology of Steve Gold, the court may be able to infer belief probability from fact probability. To permit the drawing of such an inference is not to collapse the distinction between fact probability and belief probability. It merely recognises that, in a particular case, the fact probability may be so strong that the court is satisfied as to belief probability. Whether an inference of belief probability should be drawn in any given case is not a matter of logic. The law does not demand absolute certainty in this context or indeed in any context. Judges are frequently called upon to decide difficult and finely balanced questions on the balance of probability and sometimes say that they have reached their conclusions after much anxious consideration of the facts. It is true that, once the facts have been determined, they are treated as having been established and, subject to any appeal, they cannot be challenged. But the judge may even acknowledge in his judgment that he cannot be *certain* that the facts are as he found them to be. He cannot exclude another *possibility*. But he is satisfied on the balance of probability as to the facts and that is all that the law requires.

223. I would in any event endorse what Lord Phillips has said about the limits of epidemiological evidence at paras 97 to 101 and also what he has said about what constitutes a "material increase in risk" at paras 107 and 108. I also agree with

what Lord Rodger has said at paras 130 to 132 about the observations by Smith LJ about the effect of section 3 of the 2006 Act.

224. For these reasons, I would dismiss these appeals.