



Case No: 7LV30059

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION
LIVERPOOL DISTRICT REGISTRY

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 16/05/2008

Before:

MR JUSTICE MACKAY

Between:

**John Shortell (executor of the estate of John
Joseph Shortell deceased and litigation friend of
Eileen Shortell)**

Claimant

- and -

**BICAL construction Ltd (sued as successor to BIC
Construction Ltd)**

Defendant

**Mr Allan Gore QC and Mr Andrew Macdonald (instructed by Catherine Higgins) for the
Claimant**

Mr Charles Feeny (instructed by Berrymans Lace Mawer) for the Defendant

Hearing dates: 28 April – 1 May 2008

Approved Judgment

I direct that pursuant to CPR PD 39A para 6.1 no official shorthand note shall be taken of this Judgment and that copies of this version as handed down may be treated as authentic.

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MR JUSTICE MACKAY

Mr Justice Mackay:

1. John Joseph Shortell was born on 1 February 1932 and died of lung cancer on 8 July 2006 aged 74. For much of his working life he was exposed to asbestos, but only when employed by the Defendant. This case is about whether the claimant, who acts on behalf of his estate and his widow, can prove that his death was caused by that exposure at that work.
2. It is common ground that the deceased was a fairly heavy smoker, though over what period is something I will have to return to. There was no post mortem carried out and therefore no histopathological examination of his lungs has been possible. There is no radiological evidence that he suffered from the fibrosis of the lungs called asbestosis, though he did have signs of bilateral pleural plaques together with pleural thickening. Because of the combination of the effects on his lungs of asbestos and his smoking habit the issues on causation in this case have been acute.
3. As to breach of duty, the Defendant admits that if it is proved that the deceased was exposed to substantial quantities of asbestos dust for significant periods during his employment by the Defendant, or that after 1966 he was exposed to quantities of asbestos dust which more than minimally increased the risk of mesothelioma, then such exposure was an actionable breach of the duty they owed him.

The Level of Exposure

4. This is a key issue which I have to determine at the outset. Evidence has been come from two witness statements made by the deceased before he died and two consultant engineers.
5. The claimants called Peter Deary, who holds a Bsc in Biochemistry and is a Fellow of the Institution of Occupational Safety and Health. After a spell in industry he became a member of HM Factory Inspectorate for some eleven years where he inspected premises where asbestos products were manufactured, as well as many other diverse sites where asbestos-containing materials were being used or worked on. These included power stations. For three years he was the Nominated Asbestos Inspector in Greater Manchester in which position he advised Inspectors and Environmental Health Officers. From 1989 he has practised in a consultant capacity and has prepared over 3000 forensic reports in cases involving asbestos, in the course of which he has done hundreds of dose calculations. In the course of his work he has visited many power stations in the Manchester area, either when they were operating in normal conditions or shut down for overhaul or when being demolished.
6. To address the important question of the likely level of exposure of the deceased to asbestos in his work for the defendants he started with the deceased's employment history as apparent in his 10 page statement dated 20 September 2005. He then interviewed him on 9 May 2006 for, he thought, 1 - 1¼ hours. The claimant by then was very ill and died within two months. Mr Deary did not claim to have a clear individual recollection of this interview because he does so many, but he said, and I accept, that he was satisfied that Mr Shortell was in a position to give him proper

instructions; he did not have to abandon the interview as he sometimes finds he has to with those who have suffered asbestos-related disease.

7. He prepared his report of 11 May 2006, which he dictated straight away, and in a second witness statement the deceased affirmed the truth of its contents having read it. He recollected, as an indicator that the deceased's mind was unaffected by the state of his health, that having had it explained to him that from 1970 as a result of a change in the regulatory framework all new work would have been carried out using asbestos free materials, certainly within the premises of the CEGB who had issued an edict to that effect, Mr Shortell was able to appreciate that and the impact of it on his evidence.
8. The deceased worked as a joiner that is to say he fixed together sections of large cables, some twelve inches in diameter which carried 33,000 volts. He worked inside power stations which were new buildings under construction or which were being repaired under shut down conditions. In a narrative form he said he often worked close to ladders who were using asbestos, that when they mixed it the atmosphere was very dusty and sometimes so much so that "you couldn't easily see what you were doing so you had to stop work for a bit". The asbestos was carried up ladders to scaffolding or grating where the ladders were working and bits of asbestos fell on the grating and occasionally landed on him and returned to dust. He said dust was floating down and landing everywhere as he worked and he used the phrase "the dust fell down like snow". There would of course have been dust from a number of sources within these structures.
9. When Mr Deary interviewed him the deceased described some ten or so power stations where he remembered working in this way, some on shut down some under construction, as well as the Shell refinery at Stanlow. He described ladders engaged in the dry stripping of asbestos insulation of the pipe work and plant using hand tools causing clouds of dust, as well as mixing and applying asbestos paste, sawing pre-formed sections to insulate pipes, using asbestos tape to insulate small bore pipe work and sweeping asbestos dust and debris from floors. Often, but not always, he worked in proximity to those carrying out these operations and he said that at times he had the appearance of a snowman by reason of the quantity of dust deposited on him. This, says Mr Deary, was the deceased's own description of his appearance, and he was careful not to lead him to it. All this exposure was inside buildings except for the work he did at Stanlow.
10. Mr Deary has attempted to quantify the deceased's experience in terms of his exposure load by reference to the only published material now available which could indicate the likely level of asbestos fibres generated by the types of activity that he describes.
11. The first source of data is an internal document generated at the request of the medical department of Shell Chemicals UK Ltd at their Carrington works in November 1973. Two men had been engaged in de-lagging asbestos in a small closed room and had samplers fitted to them. The man who did the de-lagging for a 34 minute period experienced a level of fibres per cubic centimetre ("fibres/ml") at presumably a mean level of 100 for the first 20 minutes measured and 360 for the last 14 minutes measured. Over this same period his assistant, who would necessarily have been within a few feet of him throughout, recorded readings of 115 and 113. The impetus

for this survey was new standards which the company was keen to adhere to, as it would seem. Mr Deary said that these were very high levels and were present for more than a few moments, but obviously allowance has to be made for the fact that they were in a closed room, very much smaller than any of the structures in which the deceased worked. However, he said to an extent that was counter-balanced by the fact that in a power station there would be teams of men rather than one individual doing such work and it would likely last for a longer time.

12. His second source of information was guidance from the Health and Safety Executive (Guidance Note EH 35) first issued in 1984 as a guide to the airborne fibres levels that could be expected close to an operator's breathing zone in the course of various processes. The dry stripping of crocidolite asbestos, it said, gave rise to readings ranging from 100 -1000 fibres/ml, whereas the dry stripping of all other kinds was merely described as "greater than 20". Mr Deary explained that crocidolite was not just the most toxic of the three forms of asbestos but also the most prone to becoming airborne in that it produced more dust when worked on in this way. Dr Leigh later supported this notion, saying that amphibole asbestos (i.e.amosite and crocidolite) was 3 to 5 times more likely to become and remain airborne than serpentine (chrysotile).
13. There is very obviously a very wide range of readings here, which Mr Deary suspected could be accounted for by the fact that at the low extreme were probably such cases as one man working alone out of doors or in well ventilated conditions and at the other extreme a number of men working in more confined spaces or with worse ventilation. It is noteworthy that the HSE guide pre-supposes that "these figures are the concentration found when the processes are carefully carried out. Bad handling practices may result in higher concentrations". Mr Shortell's descriptions were not of good handling techniques, in general terms.
14. Though these figures are said to have been "based on measurements taken by HSE" nobody has seen the underlying data. Equally, though it is plain (see Mr Walker's evidence below) that there must have been a mass of material in existence in the hands of relevant employers, similar in kind to the Shell Carrington readings, nobody has sought to challenge the validity or basis of this important guidance note.
15. The third source of material Mr Deary considered was EH41, HSE guidance dated 1985 where the executive was concerned to give a simple guide to employers as to what types of respirator needed to be used for different commonly encountered jobs, and in each case the adequacy of protection provided by different equipment. The dry stripping of crocidolite or amosite asbestos, it said, could give rise to 180 – 500 fibres/ml as could stripping with ineffective damping, and dry stripping in confined areas to a burden of 500 + fibres/ml.
16. Finally he considered two papers by Surgeon-Commander Harries RN who measured dust generated in the removal of pipe lagging in the engine and boiler rooms of aircraft carriers, cruisers and frigates at HM Dockyard Devonport. A large number of breathing zone samples were taken as near as possible to the men's faces and atmosphere samples were taken at "convenient points in the compartment". The mean readings in the breathing zone range from 91-97 fibres/ml and in the general atmosphere from 88-171 fibres/ml. The higher readings in boiler rooms as against engine rooms was said to be likely to be because there was more insulating material in

a boiler room and, because work was carried on at different levels, so that debris fell some distance to the deck creating more dust in the general atmosphere. Mr Deary said, and I accept, that this picture is not dissimilar to a boiler area in a power station where the boilers are tall structures with a series of ladders and gratings allowing access to various stages of their overall height.

17. Although he accepted that there was a quick turn around for warships in a dock the same would be the case for power stations in, for example, their summer shut down period where there would be pressure to work all trades alongside each other to get the station back on stream in time for winter. Mr Walker accepted that Harries was one of the largest studies of this kind, based on some hundreds of samples, and the finding of widespread dispersion of fibres that he found in his Table 2, for example, would be similar to the pattern of distribution found in a power station. Though he used sampling devices which differed from what he called the “regulated method” nobody had since then challenged or attacked his data in any published article or commentary. I find Harries an impressive piece of work in this field, and highly relevant when considering the likely fibre levels in power stations.
18. This was the raw material that he had, together of course with the claimant’s own account which necessarily was descriptive only. Mr Deary started his calculations with the last six years of exposure that is to say 1970-1976, as it was common ground that asbestos was not used in new insulation work at this time, but old asbestos would still have had to have been stripped. From the wide range in the HSE concentration figure of 100-1000 he took a figure of about 300 fibres/ml, as, he said, a conservative figure representing what was being told to him by the deceased, and allowing for the fact that some proportion of the lagging would have been chrysotile, and he also took into account EH41 with its range of 180-500. The next step in his calculation was to note that these figures were yielded only when the actual work in question was being undertaken, and therefore if, as he understood was the case, it only went on for half the working day the average daily figure should be reduced by 50% to 150 fibres/ml. It was agreed between the engineers that if the deceased was working within twenty feet of the working lagger then his likely burden at that distance would be 10%, or 15 fibres/ml which is the likely average exposure figure he uses for this period. Mr Deary assumed that all the deceased’s exposure was at this distance, no closer and no further from the source.
19. He then considered the earlier period 1953-1970 when there was a mixture of both stripping and fitting of new asbestos. Starting from the post 1970 figure, and in order to account for the fact that the operation of re-lagging would be less dusty than stripping he attributed to it a notional value of 5 fibres/ml and that gave him an average for the two types of operation of 10 fibres/ml.
20. He then accounted for the fact that in the earlier period the claimant spent, he said, about 75% of his time only at power stations and at Stanlow and for 40% of that he was within the twenty foot radius. Factoring in his lengthy working week, which at that time averaged 70 hours, gave approximately 8.9 years of exposure or a load of 89 fibres/ml-years.
21. For the later period of 1970-1976 the claimant said that he was only exposed to asbestos dust for about 10% of the time he spent at the power stations and at Stanlow (an overall period of 4.5 years) that is to say 0.4 years which again applying the 70

hour working week yielded a figure of 10 fibres/ml – years. Therefore by this route he calculated that the claimant total asbestos dose while in the employ of the defendants would have been 99 fibres/ml – years.

22. Mr Deary says that he has taken conservative figures throughout this calculation, which of course depends heavily on not just the veracity but the accuracy of the claimant's recollection on such matters as for example percentage of his working time he spent within the proximity of those working on asbestos. It also makes assumptions or estimates (the 50%, 40% and 20 foot radius) which if wrong would radically alter the final figure. Beyond an agreement between him and Mr Walker that the relevant lagging contained all types of fibre there is no way of identifying the proportion of amphibole fibres present on these occasions. But giving reasonable credence to the claimant's account, which I feel should be the approach I too should take, he has he says used such objective evidence as there is of a contemporary nature as to the likely fibre load encountered by some one in the deceased position. From his great experience of these cases he says there is no other information on this topic. He says he has, for example, assumed careful working methods were used throughout, he has included nothing for exposure working outside the 20 foot radius, or for working closer than 20 feet away (whereas the deceased's statement talked of working at times within 5-6 feet.)
23. Mr Walker for his part responds that the HSE data in guidance note EH35 gives too wide a range of figures (100-1000) to enable any expert opinion to be based upon it. He points out that there is no clear indication as to how these figures were arrived at and he would want to see the original data to test the validity of the estimates produced. It is as it states guidance, not a statutory instrument and he adds that its findings have "never been challenged or vindicated".
24. As to the Harries data he says the work in power stations is not comparable, as the re-fitting of military vessels was extremely highly pressured work, the work places were necessarily poorly ventilated and contamination spread from remote areas without significant dilution. He thinks that those conditions were not likely to have been replicated in commercial power stations. Mr Deary recognises some force in this and does not seek to draw direct comparisons but he sees similarities, particularly in terms of the pressure of work and the large number of contractors who would be present at the same time on power station insulation contracts. He points out that the ladders and the jointers would naturally tend to be working at the same final stages of the re-fitting or new building of such structures, only being able to ply their respective trades after the major construction and installation of machinery had taken place.
25. Mr Walker argues that a jointer by virtue of the work he was doing would most likely be found at the electrical generating end of a power station or in the transformer or switch gear units rather than in the boiler house. A plan of one particular power station was put in which Mr Deary accepted showed a not unfamiliar lay out. He points out that high voltage cables ran through the turbine area into the boiler areas and switch rooms since there was a high power requirement within all those areas. While the 12" cables the deceased referred to would all run downstream from the heating area of the power station, the alternators on the plan put to him, and from which the generated power would be led, are shown as being in the turbine area, where there was much lagging, and the deceased would have worked on large cables there. He also said from his experience of these structures, which was not

inconsiderable, that there was a lot of heavy machinery (coal grinding mills and fans) within the boiler area which required high voltage supply and jointing would be needed, albeit on rather smaller cables than the 12” type.

26. In reaching his own assessment of the long term exposure at 1 fibres/ml on a continuous time weighted average Mr Walker has heavily relied on what he called “my own occupational hygiene experience”, by which he meant data acquired or seen by him in the context of consultancy work when he has been instructed by users of asbestos. This remains the commercially confidential property of the commissioning employers and cannot be quoted by him in detail, or indeed at all, as those who commissioned it have never chosen to put it into the public domain. His summary of such data is that starting from his inspections at about 1970 the likely exposure levels in power stations never achieved the concentrations indicated by Mr Deary of 10 or 15, certainly not for periods of time to enable weekly monthly or annual fibre doses to be calculated as Mr Deary has done. His own figure was that a reasonable average exposure of 0.1 – 1.0 fibres/ml would be likely. His corrected lifetime figure is 9.55 fibres/ml –years, based on what he called the worst case.
27. Mr Walker is a chemist by qualification and was one in his 5 years in industry. In the Factory Inspectorate he was a Chemical Inspector of Factories and not primarily concerned with asbestos, though when he was so concerned, and in his private practice after 1982, the tighter regime imposed following the 1969 Regulations was in force. He was a candid and intelligent witness but I have no hesitation in preferring the evidence of Mr Deary on this issue. Mr Walker was not just less qualified and experienced in this field; he made one fairly egregious error in his calculations of a “working year”, which was pointed out to him by the Defendants’ statistical expert Professor Berry, and which he recanted, calling his initial approach “silly or misleading”. He also was hampered, with one hand behind his back as he accepted, by being initially instructed with only the first three pages of Mr Deary’s report supplied, and he challenged him without seeing his full report, which he agreed was less than a satisfactory state of affairs.
28. But more importantly he was critical of Mr Deary’s approach for its perceived lack of scientific rigour. He said for his part he would look for data found by means of a properly structured academic exercise so as to ensure it was statistically acceptable, so as to achieve statistical confidence, with a 95% confidence interval. Not only was he imposing an unduly high burden of scientific proof, at least for the purposes of a trial such as this, he was also failing to practise what he preached. His own data, which informed his lifetime burden figure, failed any test one could apply; they were entirely invisible, non-specific, a meta-analysis (presumably) of a number of disparate findings, and incapable therefore of being tested in any way.
29. By contrast Mr Deary had the benefit of directly questioning the deceased, which I find he did carefully and without putting words in his mouth, and a number of sets of data which I have found of assistance. The Shell letter is presumably much like the sort of material Mr Walker’s former clients will have in their files, and relates to a very different environment. Harries is by any standards a large and impressive piece of research, scientifically carried out, and of an environment with as many similarities to as differences from the deceased’s workplaces for much of the time. The HSE guidance is plainly evidence based and has never been challenged by anyone, as it could have been. I am sure if the large industrial users of asbestos in the 1970s and

1980s were sitting on material which showed that the executive had exaggerated the dangers of this material by several orders of magnitude they would have deployed it and at least initiated some sort of debate or dialogue about the subject.

30. Mr Feeny relies on an entry in some medical notes taken when the deceased was first admitted to hospital as an in patient and was spitting blood. The note records, in the context of his work exposure “lead + + 0 asbestos”. Dr Rudd said it is not uncommon for people at this stage of an investigation not to recall asbestos exposure when in fact they have been exposed, though he said that is more understandable in mesothelioma cases where the exposures are often very slight. Mr Gore QC made the valid point that much depended on the question the House Officer taking the history asked – was it on the lines of “Did you work with/use asbestos?” to which the answer would have been no. Would a junior doctor have thought to ask a question designed to elicit neighbourhood exposure? I cannot attach significance to this entry in the face of the deceased’s later evidence to his solicitors and to Mr Deary.
31. I am satisfied on a balance of probabilities that Mr Deary’s assessment of the lifetime burden of asbestos suffered by Mr Shortell was as he stated, namely 99 fibres/ml-years, and that I shall therefore proceed on the basis that this level is proved as an established fact. As Lord Diplock said in Mallett v McMonagle [1970] AC 166:

“In deciding what did happen in the past a court decides on the balance of probabilities. Anything that is more probable than not it treats as certain”

What that lifetime burden comprised, in terms of fibre types, is another matter, to which I will have to return.

The Medical Evidence

32. The principal medical evidence in the case came from Dr R.M.Rudd called by the claimant. He is extremely well known as a pre-eminent authority in this field. The defendant originally commissioned a report from Dr John Moore-Gillon, an expert of equal standing, who in all essential respects agreed with the propositions that Dr Rudd was putting forward. At a late stage the defendant instructed a pathologist Dr Richard Attanoos but he was not called to give evidence nor was his report relied on by Mr Feeny. The propositions that I set out below are therefore effectively agreed by all other experts, or were not challenged when Dr Rudd gave his evidence.
33. The propositions are:-
- i) Both asbestos and tobacco smoke are established as risk factors for the development of lung cancer.
 - ii) In each case the risk increases in approximate proportion to the dose of asbestos received or the amount of tobacco smoked.
 - iii) There is a multiplicative interaction between the two risk factors, with the result that when both are present the overall risk is much greater than the sum of the two (Hammond 1979; where a leading study of North American insulation workers found a risk factor from smoking of approximately ten fold,

from asbestos exposure approximately five fold and in the cases where both hazards were present together the risk factor was more than fifty fold).

- iv) Current mainstream medical opinion does not hold to the view that the risk of lung cancer from asbestos is confined to those who have developed established asbestosis (Henderson 1997 and 2004). This so called “precursor theory” is not advanced in this case, and indeed is expressly disavowed by Mr Feeny.
 - v) The 1997 Helsinki criteria list as sufficient for the attribution of a causal role to asbestos in the development of lung cancer in an individual case a cumulative exposure of 25 fibre/ml years. To the extent that there is controversy about this figure it is as to whether a higher figure is appropriate where the asbestos type is chrysotile.
 - vi) At the level of 25 fibre/ml years the risk of lung cancer is approximately doubled.
 - vii) If Mr Deary’s estimate of 99 fibre/ml years is right that exposure on its own increased his risk of developing lung cancer by a factor of approximately five.
34. Dr Rudd said that if he had smoked until the age of 49 that would have left him with a risk of about 5% for the development of lung cancer, which is approximately ten times the risk to a person who has never smoked and has not been exposed to any other causative agent.
35. Expanding on the Helsinki criteria, these presuppose a mixture of fibre types including a substantial proportion of amphiboles, and say that the relative risk is doubled at the figure of 25 fibre/ml years. If Mr Deary’s 99 fibre/ml years figure is right the relative risk is probably three or more in Dr Rudd’s view. Dealing with the Helsinki figure, he accepted that it was too low for a doubling of risk if a substantial part of the mixture was chrysotile – if for example the chrysotile /amphibole mix was 50-50 Dr Rudd would want a figure of 40 fibre/ml years to satisfy himself that there was a doubling of the risk.
36. In his report of 12 December 2007 which he explained in his evidence Dr Rudd considered the implications of the multiplicative interaction between asbestos and smoking in this area. He quoted the work of the distinguished epidemiologists Sir Richard Doll and Professor Julian Peto in 1985 to the Health and Safety Commission when they said:
- “Asbestos, moreover, seems to exert its effect synergistically with tobacco smoke, increasing the incidence rate among people of given age by the same proportion in smokers and non smokers alike. Whether the two agents act to multiply each others effects exactly is uncertain; but the interaction is so strong and so nearly multiplicative that, on present knowledge, we must assume that the chance that the lung cancer in a particular man or woman who has been exposed to asbestos is attributable in part to that exposure is unaffected by his or her past smoking habit. This is convenient from a legal view point because it means that evidence about tobacco use is not needed”

37. Perhaps the authors of this report spoke too soon, at least from a legal perspective, since it may become relevant to undertake an exercise seeking to identify or partition the contribution of each of these two agents. For his part Dr Rudd approached the matter this way. He estimated the deceased's risk factor from asbestos exposure at five fold, acting synergistically upon a 5% risk of lung cancer from smoking, giving a risk from both hazards of 25%. The synergistic effect of the asbestos has therefore increased his total risk of lung cancer from 5% to 25% which is an increase of 20%. Asbestos has therefore accounted for 20%/25% i.e. 80% of his total risk. If the deceased had in fact smoked until a later date namely 1987 then his smoking would have conferred a risk factor of 8% for the development of lung cancer. This would have been increased five fold by the effect of asbestos increasing his overall risk from 8% to 40%. Asbestos would therefore have accounted for 32%/40%, i.e. still 80% of the total risk that he would develop lung cancer.
38. Therefore it is the view of Dr Rudd that the deceased's asbestos burden in itself, if it stood alone, would have satisfied the "but for" test of causation. In combination with his risk from his smoking the smoking attributable risk is likely to have been more than doubled (as Dr Moore-Gillon agreed) by the asbestos attributable risk. It is therefore the view of both doctors that the deceased would not have developed his lung cancer had he not had his asbestos exposure. That exposure itself, if its effect is to be isolated for forensic purposes, can by the mechanism proposed by Dr Rudd, and on which he was not cross-examined, be described as contributing 80% of the total risk of contracting the disease.

The Epidemiological Evidence

39. This came from two distinguished sources. Dr James Leigh is a medically qualified physician practising as a consultant occupational physician and has spent much of his life researching and publishing the occupational health implications of asbestos. He has published 85 peer reviewed papers in this field and has graduate qualifications in medicine, mathematics computer science and law. His opposite number retained by the defendants was Professor Geoffrey Berry, a consultant biostatistician and epidemiologist of great distinction, but who has no medical qualification. Perhaps he lacked a little of Dr Leigh's direct experience in investigating asbestos related cases in power stations, which enabled Dr Leigh to express the opinion that in those environments to say that 50% of the asbestos bodies in the air would be amphibole would be quite a conservative estimate. The mix in the lagging is not the whole story; amphibole is more readily volatile and less inclined to settle than chrysotile, so it is the mix in air that he was referring to.
40. There was very little between these two distinguished experts. They agreed that the most contentious part of the Helsinki criteria was their alleged failure to take sufficient account of the differences between different types of asbestos fibre. It is to be remembered that the engineers in this case agreed in their joint statement that the fibres encountered by the deceased would have been comprised of all three types, but they were not able to identify the respective proportions. Professor Berry cited Doll and Peto in the paper quoted above, and their estimate that, assuming Mr Deary's 99 fibre/ml years lifetime figure was comprised of nothing but chrysotile, there would still be a relative risk of lung cancer in the region of two i.e. a doubling of risk.

41. The main task carried out by these two experts was another attempt at isolating or partitioning the respective contributions of asbestos and smoking in this case. To an extent this is a slightly curious exercise given that this is accepted by Mr Feeny to have been, in the legal sense, an indivisible injury. In a biological sense there is an almost complete absence of understanding as to how asbestos fibres and smoke work on the epithelial cells so as to cause cancer, let alone how the synergistic effect identified by epidemiology works. But the parties agreed concurred in it, and I accept it had to be done.
42. Professor Leigh worked with a doubling of the risk at 20 fibre/ml years for amphibole and 1000 for chrysotile. He then assumed that the mix to which the deceased was exposed was 50-50, on the basis of which he estimated the doubling of risk at 40 fibre/ml years. The relative risks for cumulative exposures of 10 and 99 fibres/ml years were then respectively 1.25 and 3.5.
43. For his part Professor Berry worked with a doubling of the risk at 25 fibres/ml years for amphibole and 200 for chrysotile. The doubling of risk at 50 fibre/ml years corresponded therefore to a mix of 43:57 amphibole to chrysotile and the relative risks for cumulative exposures of 10 and 99 fibres/ml years were then respectively 1.2 and 3.0.
44. Their assessments, therefore, of Mr Deary's lifetime burden figure and its effect on the risk is not far apart and sensibly counsel did not seek to investigate which of the two was right, namely 3.0 or 3.5, since both are plainly more than enough, on a stand-alone basis, to meet the test for causation.
45. In order to attempt to apportion the total risk into the components due respectively to asbestos and smoking they then carried out an entirely mathematical exercise using what they both described as the preferred method namely the so-called "Chase model". They used a range of figures for the relative risk due to former smoking. This ranged between a relative risk of 4 assuming he had smoked 15 cigarettes a day from 1961-1981 at the one end, and at the other end 6 assuming he had smoked 10-15 cigarettes a day from 1952-1987. Table B in their joint report sets out the results of this exercise applied to cumulative exposure of 99 fibre/ml years. The range of the contribution attributable to asbestos exposure, depending on whether the shorter or longer smoking history is adopted, lies between 27% and 42% and from smoking between 51% and 67%.
46. Doctor Leigh described this mathematical model as an artificial construct, something which can be done as he put it. It is a construct used for administrative or legal reasons and is somewhat artificially doing what biologically you cannot really do, as he put it. He was asked by Mr Gore to do another calculation, again using the Chase model, to isolate the contribution of smoking alone. Assuming a relative risk for smoking of 3.5 (his own translation of the Deary estimate) and of 4 for smoking he reached a figure of 21% as the contribution of smoking alone, which became 28% if Prof. Berry's relative risks of 3 and 6 respectively were adopted.

The Legal Test for Causation in this case.

47. The traditional test for the proof of causation is that the claimant should show on the balance of probability that the defendant's breach of duty made a material contribution to the occurrence of the event in question or, put another way, that but for the defendant's breach it would not have occurred.
48. The House of Lords has considered an exception to this rule, and the limits of that exception, in the cases of Fairchild v Glenhaven Funeral Services Ltd [2003]1AC32 and Barker v Corus Ltd [2006]2AC572, the so called "Fairchild exception" to the above general rule, which has been formulated to meet, largely for policy reasons, the problems posed by conditions such as mesothelioma where the risk factors are randomly spread over a succession of tortfeasors, any one of whom may have caused the condition but none of whom can be proved to have done so on the traditional basis.
49. This is not a problem in the current case. The causation of lung cancer as opposed to mesothelioma is dependant on an aggregate dose either of asbestos fibre or smoke. Mr Feeny for the defendant's 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.
50. That this concession is correctly made (and Mr Gore agrees with it) is reinforced by the speech of Lord Hoffmann in Barker between paragraphs 12 and 24, in which he stressed that 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.
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.
52. Lord Hoffmann concludes this passage of his speech in these words

“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”
53. While therefore the kind of exercise that Dr Rudd carried out and that the two epidemiologists gave evidence about may be highly material and necessary in a

Fairchild type of case the sole role of such calculations in this case is, at most, limited to the next decision which I have to make.

Contributory Negligence

54. On this issue the burden is carried by the defendants as Mr Feeny accepts. Section (1) of the Law Reform (Contributory Negligence) Act 1945 reads:-

“Where any person suffers damage as a result partly of his own fault and partly of the fault of any other person ... the damage is recoverable in respect thereof shall be reduced to such extent as the court thinks just and equitable and having regard to the claimants share in the responsibility for the damage”

In this case it is clear that the deceased was a smoker but had given up smoking more than twenty years before his death. Precisely when he gave up is unclear. Certain medical records refer to him as being a smoker into 1985. There is no reliable record I can see which has him smoking as late as 1987, as I interpret the only note which is said to be suggestive of such a date as reading 1981. This issue is unlikely to turn to any significant extent on a difference of two years or so. I take 1985 therefore as the time he gave up smoking and that he had smoked fifteen or twenty cigarettes a day through his adult life up to that stage.

55. I have been shown the decision of Mr Justice Stanley Burton in Badger v Ministry of Defence [2005] EWHC 2941 QB and I gratefully adopt his analysis of the law as set out by him in paragraphs 6-16 of his judgment. In broad terms the exercise a court has to apply when considering this issue is to determine it objectively that is to say asking as the first stage was there fault on the part of the claimant, in the sense that the lack of care that he took for his own health fell below the standard to be expected of a person in his position at that time. If that was so I have to consider whether the damage or injury that he suffered resulted partly from that fault and, if so, what was the extent of his responsibility for that damage or injury. The final question then is what the reduction in damages should be applying the “just and equitable” test.
56. Though it is helpful to have regard to the kind of detailed calculations that I have set out earlier in this judgment, the final stage of any judgment of contributory negligence is a jury-like exercise decided on a broad and common sense basis. As to fault on the deceased’s part the defence confine themselves to the assertion that from 1971, when public warnings were given to smokers by their being placed on cigarette packets, the claimant was failing to take reasonable care for his own safety by continuing to smoke for another 14 years or so. Unlike in the Badger case, where the judge was given a complete history of the development of the awareness of the link between smoking and cancer, and the publicity given to that awareness, I have been given no evidence. I assume from the fact that they both rely on the Badger case that they are content for me to inform myself from that material.
57. Given those warnings argues Mr Feeny that there is to put it brutally no need for a court to be merciful to smokers who disregard their own health after that date, though he accepted that one can detect in what might be termed gross cases of contributory negligence in the employer/employee field (e.g. the removal of a safety guard from a dangerous machine done by a workman who knew why it was there and that he was

therefore taking a risk) a more forgiving approach to the careless worker than to an employer in breach of his duty of care. There are no such considerations in play in this sort in this case, he argues. Dr Rudd in his report of 12 December 2007 agreed with the contention that the deceased should have known of the risks from at least 1971 and that his continuing to smoke there after was an action on his part which he carried on in the knowledge that he was risking his own health. That he was not irredeemably addicted is shown by the fact that he did in the end abandon the habit in the mid 1980's. Mr Gore says I should allow some period of grace for the realisation of the dangers to percolate through to the deceased, and for him to act on it.

58. I am satisfied that I ought to approach this issue on the basis that the deceased was himself at fault in smoking after the mid 1970's.
59. As to whether that fault of his caused his fatal illness, and if so what the extent of his responsibility for that should be, I have already indicated how Dr Rudd for his part and the epidemiologists for their parts tackled that question from what might be called a statistical viewpoint. An exact application of their results is it seems to me quite impossible since their approach was to factor in a risk factor elevated by reason of a lifetime's smoking up to the mid 1980's. For my present purposes I am confined to that part of that smoking career which can properly be described as the product of the deceased's own fault which is certainly a lesser period. Mr Feeny points to the joint statement of the epidemiologists which shows that the Chase model (looking as I say at a lifetime smoking history) shows that the deceased's smoking, on whatever approach one takes, is responsible for a greater contribution than the asbestos. He asked rhetorically why in those circumstances he should be regarded other than as more blameworthy than the defendant.
60. Mr Gore's approach is to argue that the defendant is to be treated for these purposes as being responsible both for the damage which he caused either individually or synergistically with the other risk factor. If that is the case then the relevant causal contribution of smoking alone is the 21% figure that emerged from Dr Leigh's evidence assuming his preferred relative risks for the two factors.
61. I find myself unable, indeed disinclined to resolve this question on any such mathematical basis. It is certainly not the way the Court of Appeal resolved the important case of Froom v Butcher [1976] QB 286, one result of which was that the reduction for injuries which would have been prevented altogether by the taking of appropriate care by a claimant is no more than 25%. One can understand a jury reaching that decision more readily than one can understand a mathematician or statistician doing so.
62. That the defendant should bear the lion's share of responsibility in a case such as this is a proposition which does not give me pause. Throughout this period it was under a duty first at common law and latterly under statutory instrument to take steps for the care of the deceased which it did not take. This primary liability should not, says Mr Gore, be emasculated by a high finding of contributory negligence, and I agree with him. The actual decision of the court in Badger is of course one which was formed on its own facts and is not in any way binding on me, but it has to be of interest to me in my tasks. In that case the late Mr Badger had a much more extensive smoking history, which he maintained until his death in the face of four specific warnings from doctors to give up because of his health problems.

63. In the final analysis I have to decide what is just and equitable as between the defendant and the claimant in this case. I am satisfied that a reduction should be made for the deceased's contributory negligence and I assess it at 15%.