



KINGS
CHAMBERS

ASBESTOS LUNG CANCERS – WHERE ARE WE NOW AFTER BLACKMORE?

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Introduction

1. In *The Department for Communities and Local Government v Blackmore* [2017] EWCA Civ 1136 the Court of Appeal delivered another judgment arising from asbestos-related lung cancer (“ALC”).
2. The question on appeal was very narrowly framed: *where a smoker had developed lung cancer arising from his occupational exposure to asbestos should any contributory negligence for smoking be based solely upon the causative potency of his smoking when compared to that of the inhaled asbestos or should it also factor in a comparison between the moral culpability of the exposurer in exposing him to asbestos dust (usually during employment) as opposed to the (lesser) moral culpability of the smoker in continuing his habit after such time as he ought to have given up?* On the facts in *Blackmore* if the comparison-of-moral-culpability element was included in the assessment that would lead to a much smaller deduction for contributory negligence being made than would a consideration of relative causative potencies alone. In that respect *Blackmore* was a standard ALC case in that most victims were also smokers as well and by its biological effect, smoking is a far more potent promoter of lung cancer than asbestos.
3. Perhaps unsurprisingly, the Court of Appeal answered the question upon appeal ‘yes: relative culpability should be included in the assessment of contributory negligence’ - and to a significant degree: a level of contribution which otherwise would have been in the region of 85% had causative potency alone been considered, was reduced to 30% to reflect the relative culpabilities.
4. The case is of interest for two reasons, one direct and one indirect. The direct interest lies in the submissions made by the Defendant in that case as to why causative potency should be the only factor considered in contributory negligence in ALC cases (and relative fault should remain ignored). The indirect interest lies in using this case as the template against which a more general review of ALC cases can be undertaken; seeing how this case slots in with other asbestos cases and what it tells us about how these cases should be fought – from the perspective of both sides.
5. It seems to me that this indirect interest falls into 3 discrete areas:
 - 5.1. The role of the advocate in seeking to make submissions which consider not just the narrow point being raised but, where relevant, the wider narrative of asbestos litigation generally. Nothing I set out below should be taken as personal criticism of any advocate (or indeed of any Court!) and I accept at once that I have not seen the Defendant’s skeleton in *Blackmore* and it may be that the points of apparent omission addressed later within this paper were indeed covered amply in the skeletons but that the Court of Appeal simply failed to either acknowledge or grapple with them. That said, on its face, *Blackmore* seems, by unintentional consequence to have amounted to an invitation by Defendants to apply *Fairchild* to ALC – a proposition which would appear at best, counter-intuitive, from the perspective of those who would have to meet greatly enhanced numbers of claims arising from asbestos if so.

5.2. When one considers ALC litigation more generally, there remain a number of fundamental unanswered questions – not least of which is that there now appear to be competing and contradictory opinions within the Court of Appeal authorities about the legal effects of the ‘doubling of the risk’ test in ALC cases. Which is correct and how should advisors advise?

(a) As I shall hope to demonstrate below, the ‘doubling of the risk’ test for causation within ALC cases does not seem apposite to ALC where there are two major pollutant risk factors for causing it, namely asbestos and smoking act, *and* those two risk factors act in combination with each other; not additively but, rather, in a multiplicative way. This multiplicative (and not additive) effect means that when asking the question ‘which is the predominant element of the two in any particular case of lung cancer’ it becomes critically important to decide which of the two risks one takes as being the background risk and which is the ‘unnatural’ risk which, when multiplied against the ‘background’ risk suddenly creates a much higher overall risk. Because this is a fundamental problem in consideration of ‘doubling of the risk’ it is worth setting it out at this very early stage by considering a set of assumed facts

- Suppose the smoking history when considered in isolation in any one case increases the true ‘background’ risk (which we all face because we are humans) by (x 10)
- Suppose in the same case, the effect of the asbestos exposure history alone is to increase that background risk by (x 5)
- The multiplicative rule is that the *combined* risk from smoking and asbestos is (x 50).
- If one *chooses* to assume that smoking is the basic enhanced risk against which the effect of the addition of asbestos is tested then we can see that asbestos converts that risk from (x 10) (ie smoking only) to (x 50). Therefore, on that basis, asbestos is responsible for 80% of the *total* risk increase since (x 10) is 20% of (x 50). Equally clearly, on this basis, the asbestos has been the factor which has more than doubled the risk overall as it is responsible for 80% of all risk.
- But, conversely, if one *chooses* to assume that asbestos is the basic enhanced risk already present against which the effect of adding smoking in is to be tested, then the addition of smoking is responsible for increasing the risk from (x5) to (x50) and therefore, on this basis, smoking is responsible for 95% of the total risk (ie since 45 is 90% of 50). On precisely the same basic facts one can now conclude that smoking has been the factor which has more than doubled the overall risk of lung cancer having been caused.

Whether one chooses to treat smoking or asbestos as the basic enhanced risk against which the effect of adding the other is tested *is simply a value judgment made by the Court*¹. Thus any calculation based on combined risk and which agent more than doubled it is always predicated on a pure value judgment which comes close to pre-judging the decision you want to arrive at! In this paper, this is referred to as the ‘Shortell fallacy’ since it arose in the evidence of Dr Rudd in the case of *Shortell v BICAL Construction (Liverpool DR 2008 WL 2148256)* albeit the Court did not accede to it on the facts since the level of asbestos exposure was in any event so high²

- (b) The multiplicative effect means that in absolute terms, very little smoking by the victim can have a dramatic effect on his life-chance of developing lung cancer where a tortfeasor-employer has exposed him to very high levels of asbestos (eg where the exposer has created a (x 50) risk of lung cancer, a smoking history which merely increases the risk (x 2) acts in combination to create a second (x50) risk (since $50 \times 2 = x 100$)

¹ *It is difficult to consider to find a principle which would allow the Judge to answer why one should be considered as the background to the other: both smoking and asbestos inhalation may arise as result of someone’s breach (either the employer or that of the smoking manufacturer); both involve reduced volition – the victim qua employee inhales asbestos because his employer requires him to work in conditions where that exposure occurs whilst the victim qua smoker behaves according to the addiction induced by the very product which is increasing his risk of cancer and both factors are capable of amounting to contributory negligence*

² *Given the importance of the ‘Shortell Fallacy’ to this paper, it is worth setting out the facts for reference: Mr Shortell succumbed to lung cancer on 08.07.06 aged 74. He had been a fairly heavy smoker and during his working career had worked as a joiner fixing sections of large cables inside power stations where he was exposed to asbestos. His exposure was predominantly due to working in the vicinity of ladders, who were carrying out asbestos materials on scaffolds. The Judge found that the level of exposure amounted to approximately 99 fibre/ml years. The medical evidence was agreed that both asbestos and tobacco smoke were established as risk factors for the development of lung cancer and that in each case the risk increases in approximate proportion to the dose of those carcinogens and there was a multiplicative interaction between the 2 risk factors. The evidence also accepted that at 25 fibre/ml years the risk of lung cancer was approximately doubled for blue and brown asbestos. Per Mackay J*

“The traditional test for the proof of causation is that the Claimant should show in 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. 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] 1 AC 32 and *Barker v Corus Limited* [2006] 2 AC 572, the so-called “Fairchild exception” to the above general rule, which has been formulated to meet, largely the 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 he condition but none of whom can be proved to have done so on a traditional basis. This is not a problem in the current case. 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 the 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 a 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”.

- (c) Whatever the conceptual fitness for purpose of the ‘doubling the risk’ test as an analytical tool in ALC cases, its ramifications are not agreed on within the Court of Appeal in fact as we shall see below.
- (d) When one considers the following factors for any test of causation, namely:
- Is it, in its conception, consistent with the wider common law?
 - As between the victim and the exposor is it fair, just and reasonable³ and does it provide reproducible results which can be advised upon accurately prior to trial?
 - Does the test permit both contributory negligence against the victim and contribution between tortfeasors to occur consistent with the relevant statutes (or otherwise does it limit liability overall to the proportion of contribution of each tortfeasor where several exist)?

the ‘doubling of the risk’ test (especially as interpreted) is not sufficiently clear to bear the burden these tests place upon it.

- (e) There are only 2 coherent causation tests available for ALC which are consistent with the common law: the first is the requirement to prove, on a mechanistic level and by reference to cell biology in an individual case, that the contribution of the tortfeasor before the Court contributed *in fact* to the happening of the cancer. If proven then the orthodox rules regarding material contribution to an indivisible condition are triggered (the tortfeasor is liable for all subject to contributory negligence and the right of seeking contribution from coevals) (*Bonnington Castings v Wardlaw* [1956] AC 613; *Rahman v Arearose* [2001] QB 351). The second is to apply *Fairchild* fully to ALC (that is including all subsequent Supreme Court explanation and extrapolation of *Fairchild* and the provisions of s3 Compensation Act 2006). On this basis proof of mere increase in the risk would be sufficient to permit full compensation – again subject to contributory negligence - which is expressly permitted under s3(3)(b) of the Compensation Act 2006 - and the right of contribution).
- (f) Neither of the above solutions is free from trouble: the first solution is evidentially close on impossible to prove and would choke off all ALC claims. The second solution would lead to a tide of cases and in any event is not entirely free of the problem of how to apportion between tortfeasors inter se where the two risk factors are multiplicative. Each however is better than the present jumble based upon the interpretation of doubling of the risk. The stakes could not be higher. The number of ALC cases outnumber that of mesotheliomas and upon the choice of causation test rests the fate of billions of £.

³ *After all, causation is merely a policy tool which permits the Court to determine whether a loss ‘should’ be permitted to be recovered: to this end the Court can apply a number of techniques, whether to consider the scope of the duty of care; remoteness or more general recourse to public policy etc (Kuwait Airway Corp v Iraq Airways (Nos. 4 and 5) [2002] UKHL 19 @ 70-74 per Lord Nicholls)*

- 5.3. In addition to causation, the second major area of enquiry is how contributory negligence should be dealt with in smoking cases since this will also have an enormous effect on damages – since most ALC victims were also smokers.
- 5.4. One of the broad themes of this paper is that these matters interlock to such a degree that it is the duty of any advocate preparing a trial or an appeal in respect of one aspect needs to be fully able to consider and make submissions not just on that aspect but on all the interlocking parts as well.

(In this document Judgment paragraph numbers are represented by square brackets)

The Facts and Approach of the Courts in Blackmore

6. The facts set out below are taken from both the judgment of the CA and at first instance (HHJ Cotter QC – “First Instance”):
 - 6.1. The Deceased, Cyril Hollow, was born in 1936 and died in 2010 (aged 74) from lung cancer.
 - 6.2. He was exposed heavily (and hence tortiously) to asbestos in the period 1966 to 1986 during the course of his employment as a decorator within the Devonport dockyards.
 - 6.3. In addition, he had smoked from the age of 14 (commencing 1950) and the Judgment implies that he never ceased smoking albeit from 2005 he reduced his daily intake from around 20 cigarettes per day down to 12⁴.
 - 6.4. The Deceased had been warned specifically not to smoke by his GP in 1976 and had tried to give up twice thereafter.
 - 6.5. HHJ Cotter QC made no specific finding either as to when (a) a reasonably well informed member of the public should have known sufficient about the dangers of smoking to render it unreasonable for him to continue to do so or (b) as to when this Claimant should have so concluded. However he appears to have acceded to D’s assertion that smoking the ‘mid 1970s’ was ‘guilty’ (ie sufficiently culpable for the purpose of the Law Reform (Contributory Negligence) Act 1945 (First instance [15])
 - 6.6. On post mortem an asbestos fibre count revealed 2.49 million fibres per g of dry lung tissue. D conceded that at this level was proof that, in life, the Deceased had inhaled sufficient quantities of asbestos fibre as would more than double the risk that he would go on to develop asbestos lung cancer. Based on that concession D also conceded primary liability (First instance [9]). It will be necessary to revisit this test of breach more than once again below but for present purpose all that need be noted was that this *appears* to have been a concession based upon the Helsinki

⁴ Most chest physicians would consider this to equate to a smoking history of 58 pack years

criteria⁵ whereby it was stated that for fibres exceeding 5 micrometers in length more than 2 million per gram will suffice to prove that doubling of the risk.

- 6.7. The Defendants argued that there should be a deduction made from any damages to represent the Deceased's smoking habit insofar as he continued to smoke after the date when he knew or ought to have known of the risks of doing so). They argued that the deduction should reflect closely the relative risks of smoking compared to asbestos and find in the region of 85-90% contributory negligence deduction. This reduction percentage was not to be reduced to reflect competing culpabilities and in particular, any notion that as the Defendant was an employer at the time of the exposure to asbestos, it must have been in breach of an entire web of statutory duties in permitting that exposure to take place.
 - 6.8. HHJ Cotter QC did not accede to this approach: he took both causative potency and relative culpabilities into account and hence limited the deduction for contributory negligence to 30%. The Court of Appeal did not disturb this finding.
7. Before going on to consider more fully the arguments before, and hence the approach of, the Court at first instance, it may be helpful to 'draw back' and consider a little further the context in which Defendant's concession of breach had arisen.
- 7.1. In order for the fibre count referred to above to lead to a concession of breach, it was necessary for the parties and the Court to agree that the fundamental test for causation in respect of ALC was proof that the dose of asbestos received more than doubled the risk of the cancer occurring (when compared to the background risk which would have existed without exposure). What was the basis for this in logic and in law?
 - 7.2. The basis for this in logic was simple and, at first blush, apparently sound. However problems do exist with it.
 - (a) If the act of pulling a ball out of a cloth bag is taken as the metaphor for a person receiving the diagnosis of lung cancer one can envisage the risk of the cancer happening 'naturally' as being represented by 5 white balls contained within the bag.
 - (b) Now assume that for each given unit of exposure to asbestos exposure the victim suffers, a black ball is added to the bag. The question can now be asked – at what point does it become more likely than not that a black ball is pulled out of the bag (representing that the cancer was caused by asbestos) than that a white ball was pulled out (representing that the cancer was caused by 'natural' processes)? The answer is when there are 6 black balls in addition to the 5 white balls. Thus, it is only when the risk originally assessed as 5 white balls is more than doubled to 11 balls (of both colours) does it become more likely than not that a ball pulled out will be black rather than white. Put into a legal formula it is only when the tortious element more than doubles the risk of the event can it be said that once

⁵ *Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution* : Scand J Work Environ Health 1997;23(4):311-316 (http://www.sjweb.fi/show_abstract.php?abstract_id=226)

the event occurs it was caused, on balance, by the tortious cause and not the non-tortious cause. As we shall see at the end of this paper, this test is not without its difficulties and it is right that others should be at least considered.

- (c) But let us return to the multiplicative effect. Assume now that the white balls are the risk of lung cancer whilst the black balls remain representative of the risk of developing lung cancer because of asbestos exposure.
- (d) The reason why the analogy of the balls in the bag as representing the doubling of the risk falls down where the balls act multiplicatively is that in order to make the analogy biologically valid, you have to assume that when the second black ball is added to the bag (in addition to the 5 white balls) one is not increasing the risk from 6 to 7 (ie 5 white + 1 black increases to 5 white + 2 black) but from 5 to 10 (ie 5 white balls x 1 black ball increase to 5 white balls x 2 black balls). But what colour should these 'excess' (ie the excess over the number which would exist if the risks were merely added and not multiplied) be? Are they black balls since they exist because of the addition of the 2nd black ball? Or are they white balls because, without the prior presence of the 5 white balls, the addition of a 2nd black ball would have nothing to be multiplied against?
- (e) Thus the analogy throws us immediately back onto the *Shortell* fallacy: the colour of the excess balls becomes not a matter of logic or science but rather Judicial choice. Now, just because the metaphor fails it does not necessarily follow that the underlying theory is unsound – but it may and, in any event, where it is very difficult to conceptualise the doubling of the risk approach by being able to create working analogies or metaphors, it becomes equally difficult to 'play with' or conceptualise the ramifications of adopting such a test.
- (f) In addition to the problem of the multiplicative effect is the problem that it was never made clear whether it had to be shown that each Defendant sued before the Court, when considered in isolation, had to have more than doubled the risk in order to be liable under this doctrine or whether it was enough that all exposers had, in combination caused the risk to be doubled and that all that was required of each exposers before the Court was that it had been the cause of a material contribution. If the latter then three further issues arose
 - * Was each tortfeasor taken to have materially contributed to the cause of the lung cancer *in fact* such as to render it liable to pay the full amount to the victim (subject to the rules against double recovery and contribution between tortfeasors inter se?). This formulation could lead to a Defendant who had only been responsible for an exposure which was nowhere near sufficient to have doubled the risk being forced to pay for the entire damages.
 - * Was each tortfeasor taken to have merely increased the risk of causing the lung cancer and hence in some way be liable only for its aliquot share of either the proportion of the victim's total dose of asbestos or of the proportion of his dose which was covered by all of the Defendants before the Court (and if so, upon what jurisprudential basis could such an arrangement be sanctioned). This could lead to a victim having large holes in his compensation

if parts of his exposure arose from Defendants who could either not be sued or not be identified.

These issues arose in *Heneghan*.

- 7.3. So much for the logical basis: what about the legal basis? HHJ Cotter QC heard this trial almost 3 years ago (Sept/Oct 2014). This pre-dated the first instance trial in *Heneghan v Manchester Dry Docks [2014] EWHC 4190 (QB)* (subsequently [2016] *EWCA Civ 86*). At that stage the consensus amongst those practicing in this area was that proof of ‘doubling of the risk’ would be accepted to be sufficient to prove causation in an ALC case. The consensus was built on (a) the balance of authority and (b) the tactical stand-off.
- (a) Practitioners could rely upon the weightiest opinions from Judges who had been expert in the field as practitioners, namely Smith LJ (*Sienkiewicz v Greif (UK) Ltd [2009] EWCA Civ 1159 @ [23]*; *Novartis Grimsby Ltd v Cookson [2007] EWCA Civ 1261 @ [74]*; and Swift J (*Jones v Secretary of State for Energy & Climate Change [2012] EWHC 2936 (QB) (@8.61-8.65)*⁶).
 - (b) Neither side wished to upset this apple cart. No Claimant’s representative wished to take a case to the Supreme Court on a CFA to see whether a more relaxed test (ie more closely allied to Fairchild) should be applied to lung cancer because of the risk of failed litigation creating a huge amount of unrecovered fees and heartache to the individual litigant. No Defendant wanted to argue for a stricter test for fear of getting the ‘wrong’ answer from the same Court. Both sides were entirely reasonable in their circumspection.
- 7.4. If, in theory, the law permitted recovery in ALC cases where there had been proof of doubling of the risk then practitioners were entitled to ask – how does one prove the existence of that doubling practically? The Helsinki Criteria have already been mentioned briefly in 6.6. above but deserve slightly fuller treatment here.
- (a) Their basis was the epidemiological evidence that for every 1 fibre/ml year of total asbestos exposure the risk of lung cancer being caused rose by somewhere between 0.5 – 4%. (Of course the cancer risk would have been doubled once the risk rose to exceed 100% overall). At a ‘gradient’ of 0.5% increase per fibre/ml that would take 200 fibre/ml years; at a ‘gradient’ of 4% per fibre/ml it would take 25 fibre/ml years.
 - (b) This apparently objective finding in fact obscured the very important gloss that this depended on an assumption that the exposure had been to mixed asbestos fibre type. But how was the precise fibre combination of the asbestos dose ever to be proven by the individual Claimant? And even if

⁶ Whilst it is true that Lord Phillips in the Supreme Court in *Sienkiewicz* [2011] UKSC 10 @ [78] rejected Smith LJ’s analysis in the CA regarding considering proof of doubling of the risk in respect of lung cancer as being sufficient, it was widely thought (with respect to him) that his views were both obiter and apparently based upon a misapprehension of the action of asbestos within lung cancer. In this respect it will be noted that Swift J made her determination *after* Lord Phillips’ dictum in *Sienkiewicz*. However as will be appreciated from my analysis above, where Lord Phillips denied generally [@90] that ‘doubling of the risk’ as a test could apply because there had been two agents operating cumulatively and simultaneously (in our case smoking and asbestos) then I believe that he was correct

proven, there was no guidance provided as to what proportions constituted ‘mixed’ etc.

- (c) Notwithstanding that area of profound uncertainty, the Consensus report *chose* to assume that 1 fibre/ml caused the highest possible increase in risk available on the evidence – 4%. This was an unwarranted assumption. Having made that assumption they then followed the logic of their position and determined that it followed that the risk would be doubled (ie increased by 100%) at 25 fibre/ml years (ie 4% x 25 fibre/ml years = 100% increase in risk).
- (d) Armed with this statistic they then set out about considering what biological evidence in any individual would constitute such exposure (and in this they set out objective criteria such as the number of asbestos fibres discovered in the lungs on post mortem – the measure used in *Blackmore*). Less acceptably however, the Helsinki Committee also suggested that descriptions of exposure by the victim would suffice. An example would be 1 year of ‘heavy exposure’. Or ‘5-10 years of moderate exposure’. What constituted such exposure was wholly unclear.
- (e) Even the presence of asbestosis in the lungs (the presence of which would be taken under the criteria to be proof of sufficient exposure to asbestos to have caused subsequent lung cancer) was, in essence, a self-validating chimera. From a histological perspective there is no distinction to be made between asbestosis and the large number of other non-specific lung fibroses. Asbestosis is simply fibrosis occurring in the presence of a history of heavy asbestos exposure. Thus as a tool of proof it is wholly circular: one cannot prove the history of heavy exposure to asbestos by reference to the presence of asbestosis because in order to call such fibrosis an asbestosis, there needs to already have been proven a history of past heavy exposure!
- (f) HHJ Cotter QC noted the concession of breach and its foundation in an acceptance of the proof of doubling of the risk but he was very careful not to express an opinion on it (1st instance [29])

7.5. Thus, to summarise so far, the correct test for legal causation was not entirely clear but was at least subject to broad consensus; that test was of doubtful biological validity and in any event the criteria for proving compliance with the test (in the absence of asbestosis) were in some respects wholly impressionistic.

7.6. Finally in this review of the law as it would have appeared to HHJ Cotter QC in *Blackmore*, it was understood that the victim of a proven ALC could be held contributorily negligent for having smoked.

- (a) Whichever agents (or combinations of agents) caused a lung cancer in any particular case, that cancer - once present – would clearly be an ‘all or nothing’ or an “*indivisible*” condition. The law in respect of indivisible conditions is that any cause which is found in law to have materially contributed to that condition *in fact*⁷ will be taken to have been *the* cause of the entire condition for the purpose of compensation. (*Rahman*;

⁷ *ie not merely to have been a material cause to an increase in the risk of the indivisible outcome occurring*

Bonnington). It follows that under this approach, the Court can find that several competing causes were, in fact, each *the* cause of the entire condition provided that each competing cause materially contributed to the outcome in fact (even if one cannot say precisely how each causative agent actually did so) (*Bailey v MOD [2008] EWCA Civ 883; Williams v Bermuda Hospital Board [2016] UKPC 4*)

- (b) The practical effect of this is threefold with the last of the three effects being of importance in the context of contributory negligence
- Where one source of material contribution to the actual happening of the indivisible injury *in fact* is tortious whilst all others are non-tortious, then full compensation is due from the tortfeasor without deduction for the existence of the non-tortious causes (*Rahman v Arearose*)
 - Where there are two or more sources of material contribution to the indivisible injury in fact, each of which are tortious, then each tortfeasor is liable for the whole amount to the victim and the victim can elect whom to sue (subject to the rule against double recovery and the ability of each tortfeasor to seek contribution) (*ibid*)
 - Where one source of contribution to the *indivisible* outcome in fact is tortious and the other is the victims' own conduct and where that conduct arises from a want of reasonable care for herself then by operation of the Law Reform (Contributory Negligence) Act 1945 the tortfeasor remains liable to compensate the victim but there shall fall to be made a reduction 'to such extent as the Court thinks just and equitable having regard to the Claimant's share in the responsibility for the damage' (s1)
- (c) Before *Blackmore* it had not been doubted that contributory negligence in the case of smoking and ALC operated in precisely the same manner as it did in all other areas and that the assessment of contributory negligence required an assessment both of causative potency and relative culpabilities (*Davies v Swan Motor Co [1949] 2 KB 291*). One of the central features in *Blackmore* was that this assumption was refuted by the Defendant, who sought to argue that the only consideration was causative potency as between smoking and asbestos exposure (because as we shall see below, the figures favoured a larger deduction on that basis).
- (d) I emphasise the absolute necessity for the underlying 'damage' to be indivisible before the Law Reform (Contributory Negligence) Act 1945 can apply. If the damage sustained by the individual is cumulative (*'divisible'*) then, as we shall see below, there can be no question of contributory negligence – simply an assessment of the degree to which, when viewed in isolation, the tortfeasor's conduct contributed to the overall condition and limiting the tortfeasor's compensation accordingly). In such circumstances the folly of the victim is not thought of as contributory negligence but rather being a separate and sole cause of some proportion of the overall damage⁸ One of the many questions arising in ALC is whether the

⁸ By way of example, suppose a man was employed by X for 10 years and for the next 10 years he was self employed. In each period he was uniformly exposed to silica dust without protection. He developed silicosis – which is cumulative and hence divisible in nature. The level

dichotomy between the way we assess liability for divisible and indivisible injury is supported by the usually cited authority of *Barker v Corus* [2006] AC 572. (I will return to this later but to spoil the punchline at this early stage, the difficulty is that *Barker* does not apply to lung cancer and even if it did, its analysis of divisibility of risk assumed that each source of risk occurred sequentially and solely, not multiplicatively and simultaneously).

- (e) What is the medical basis for the idea that the potential *causes* of lung cancer in fact are to be treated as indivisible (and hence amenable to contributory negligence)? It is now standard for ALC cases to proceed on the following basis
- Asbestos exposure in isolation is a risk factor for causing lung cancer the size of which increase risk is proportionate to the level of exposure
 - Smoking in isolation is a risk factor for causing lung cancer which rises as smoking continues
 - The gradient at which the rate of risk rises is different for smoking then from asbestos. Smoking is the predominant risk for lung cancer
 - Where there is a history of exposure to both agents then the risk is not *additive* but rather is *multiplicative* (ie an increased risk of lung cancer from smoking alone is multiplied by the risk from asbestos exposure)
 - Since carcinogenesis is essentially random and chaotic ('stochastic') it is simply not possible to say mechanistically how each factor inter-relates to the other and at which stages in the carcinogenic process it does so, in any individual case⁹.

This was the evidence also presented in *Blackmore*.

8. At first instance in *Blackmore*:

8.1. Both sides' experts agreed that it so happened that, on the facts of this case, either agent (ie tobacco or asbestos) would, on its own, have been sufficient to more than double the risk of developing lung cancer.

8.2. D's expert argued that:

- (a) The risk increase from smoking alone was background x 9.1
- (b) The risk increase from asbestos exposure alone was background x 2.1
- (c) The combined risk was background x 9.1 x 2.1 = background x 19.11

8.3. C's expert was more circumspect and emphasised that the Deceased's exposure to asbestos was heavier than his job title alone suggested; D's figures did not take into

of lung damage is directly proportional to the dust inhaled. For the first 10 years X is responsible for his exposure and for the second 10 years the man is responsible for his own exposure. Let it be assumed he suffers a total of £100,000 injury. He obtains £50,000 from X not because X's liability is £100,000 but that thereafter there has been a deduction for contributory negligence down to £50,000, but rather because X is liable for only half of the damage in the first place and the man is responsible for the other half.

⁹ *It follows that if the law were to set the test for causation in ALC as requiring proof of such mechanisms (as is close to being the case in Australia) Amaca Pty v Ellis* [2010] HCA 5 (High Court of Australia) then Claimants would usually fail since the level of medical knowledge is simply insufficient.

account sufficiently the pre mid-1970s ‘innocent’ smoking (which itself would have been acting synergistically with the asbestos exposure occurring at the same time) and the chaos of cancer causation in any particular individual. He therefore considered that the asbestos risk was at the lower range of the band x2 to x5 (1st instance [21]).

- 8.4. As set out above, the Defendant in *Blackmore* argued that contributory negligence should be set according to the causative potencies of the two factors alone (ie ignoring culpability). However it is not at all clear how the Defendant arrived at its figures for relative culpabilities. At [16] (First instance) it was stated:

	Relative Risk	Percentage
Smoking	9.1	88.04%
Asbestos	2.1.	11.95%

The percentages are puzzling.

- (a) If D was suggesting that one *added* the relative risks and saw what each element was in comparison to the other, I arrive at different figures to the ones stated

Smoking	$9.1 / (9.1 + 2.1) = 81\%$
Asbestos	$2.1 / (9.1 + 2.1) = 19\%$

- (b) If D was suggesting that one, more conventionally, *multiplied* the relative risks and saw what each element’s contribution was in comparison to the other, I arrive at different figures again.

Smoking	$9.1 / (9.1 \times 2.1) = 47\%$
Asbestos	$2.1 / (9.1 \times 2.1) = 11\%$

- (c) We know what HHJ Cotter QC did *not* do was to assume that the presence of asbestos as a background risk was to be taken as a given against which the effect of the addition of smoking was to be tested i.e. he specifically referred to and hence avoided the Shortell Fallacy¹⁰. On the facts of the case the Shortell Fallacy would have worked as follows

Asbestos alone risk	= 2.1
Smoking alone risk	= 9.1
Asbestos and smoking risk	= $9.1 \times 2.1 = 19.11$

Assume asbestos is the background factor against which the addition of smoking is tested then smoking is responsible for increasing the risk from 2.1. to 19.11 ie smoking was responsible for 17.01/19.11 of the total risk = 89% of the total risk and asbestos for 11% of the total risk.

¹⁰ HHJ Cotter QC (1st instance [33]) refused to approach the matter that way because as he correctly pointed out such an approach put the cart before the horse: why should one assume that the asbestos inhalation was the background risk against which the additional effect of smoking was applied?

Assume smoking is the background factor against which the addition of asbestos is tested then asbestos is responsible for increasing the risk from 9.1. to 19.11 ie it is responsible for 10.01/19.11 or 52% of the total risk and hence smoking would be responsible for 48% of the total risk.

(These figures assume that the Defendant's risk increase figures were used by the Court – they weren't – but even then I cannot get the figures to match the Defendant's assessment of the risk contributions set out at the beginning of 8.4. above)

8.5. Pausing there fore a moment, what we should take from this is as follows:

- (a) It is not clear from the Judgment precisely how the overall contribution to risk was handled mathematically by the Defendants in *Blackmore*;
- (b) We should be very wary of spurious accuracy in formulae in this area since they all nearly rely upon hidden assumptions – whether as to the nature of the exposure in the first place (precisely how many cigarettes/precisely which asbestos fibre types and in what amount was the victim exposed to?) or as to the basis upon which the 'background' risk is to be assessed – ie the Shortell Fallacy.
- (c) This latter point becomes of critical importance when we come onto consider alternative models of proving causation as exemplified in *Heneghan v Manchester Dry Dock [2016] EWCA Civ 86* – where the Court appears to have acceded to the Shortell Fallacy.

8.6. However the Defendant arrived at its figures, HHJ Cotter QC commented on them in this way “9...*These figures were relied upon by Mr. Fortt on behalf of the defendant, as directly setting the appropriate level of contributory negligence. This would result in a very large deduction for contributory negligence in the region of 85% to 90*”. HHJ Cotter QC did not, of course, accede to the detail of D's approach. In rejecting it he made a number of specific (and in some cases subtle points)

- (a) He quoted with apparent approval Mackay J's dictum in *Shortell* that contributory negligence should always be approached as a broad 'jury' question.
- (b) That since the Deceased had commenced smoking prior to being exposed to asbestos the exposor had to take the victim as he found him and thus to a degree at least, the smoking should be seen as part of the background.
- (c) Whilst the Deceased always faced an increased risk of lung cancer from his smoking from the 'innocent years' and whilst this increased risk remained elevated for ever, it had to be held out of account when comparing risks arising from asbestos (First instance [42-43])¹¹

¹¹ whilst the points in (b) and (c) are entirely valid, it would render the area something of a chronological lottery if the entire basis of the appreciation of increased risk depended on the happenstance of whether asbestos or smoking exposure occurred first).

- (d) The biological harm done by the period of time when the Deceased smoked ‘innocently’ during which period he was necessarily younger, may not have been the same as the biological harm wrought by the smoking undertaken once the Deceased knew he ought to have stopped (and hence was older). (First instance [41]). The Judge could have also added that presumably the smoking in the years immediately prior to the clinical onset of the lung cancer were a complete irrelevance because the tumour was already in existence (albeit undetectable).
- (d) He decided the case on the assumption that the relative risk from the asbestos exposure was towards the bottom of the range $x_2 - x_5$ and from smoking in the range $x_{5.5} - x_{9.1}$. He did not apparently thereafter do anything than simply take a comparison between those two ranges and take as his starting point that the risk from smoking was 2 or 3 times higher than from the asbestos.

8.7. In seeking to make the argument that causative potency should be the only parameter for contributory negligence, the Defendant raised the following points, namely:

- (a) Where causative potency is based upon mathematical modelling it can be known precisely. This places it in contrast with seatbelt cases and hence renders it inappropriate to follow cases determined in such a context which hitherto have indicated the need to consider causative potency too

*This point was answered swiftly by the Judge in three ways: first, he had already taken pains to show that the mathematical model of risk assessment was spuriously precise and may in reality have been wholly inaccurate owing to the specific facts not following the assumed basis of the risk models; second, the Judge held that the submission missed the point that the requirement to consider both causative potency and culpability was too deeply fused into the common law to be ignored; third, to ignore causative potency would also involve another serious departure from all earlier authorities which suggested that where the employer’s breach consisted of breaches of statutory duty then the level of contributory negligence would always be relatively low however great the employee’s contribution to his own injury was in fact because the very existence of the statutory duties was to protect against folly or ignorance etc of it (i.e. *Toole v Bolton MBC* [2002] EWCA Civ 588 @ [13]; *Ashbridge v Christian Salvesen* [2006] CSOH 79) (see First instance [57-59]). The Judge stated:*

“.....that the defendant should bear the lion’s share of responsibility in a case of prolonged breaches of statutory duty such as this is a proposition which does not give me pause”

- (b) By analogy with s2 Civil Liability (Contribution) Act 1978 concurrent tortfeasors determining their mutual liability for contribution would consider only causative potency and not relative culpability.

The Judge dealt with this shortly by observing that was cast contribution in terms of ‘responsibility’ and this was wider than mere causative potency. Since the argument was returned to in the Court of Appeal, I shall deal further with it below –

but I comment at the moment at this stage that I cannot see what justification there ever was for that submission.

- (c) The Court in *Barker* looked at mesothelioma causation and considered it to be an issue based on risk which was divisible. In the light of that finding apportionment between Defendants (and hence also between a Defendant and a victim) would be based upon actual contribution and not culpability as set out in my section 7.6.(d) above

Again the Judge rejected this shortly by pointing out that the situation in Barker was simply not germane. Again, this point was raised again in the Court of Appeal and, again, I will deal further with it below.

- 8.8. The Judge then considered the facts in *Badger* (in particular) and *Shortell* and noted that the predominant causative factor had been smoking and yet there was had only been a small percentage deductions. Notwithstanding that the risk from smoking was ‘double or treble’ that from asbestos, the disparity in the relative culpabilities between the parties meant it right to limit the deduction for contributory negligence to 30% (although it should be noted that this was higher than the previous cases and this was notwithstanding that – unlike the earlier cases - there was no extensive history of the victim having ignored numerous medical warnings regarding his smoking habit.

Blackmore in the Court of Appeal.

9. On appeal the Defendant did not challenge the Judge’s handling of the Relative Risk calculations but instead limited its argument to stating baldly that it must clearly be wrong to conclude only 30% be deducted for contributory negligence when the finding on causation had been that smoking was clearly the predominant risk (Lloyd Jones LJ @ [18]). From this single launching point, the Defendant both reprised and expanded upon its reasoning as to why, in the case of lung cancer, causative potency (and not culpability) should be the sole determinant of the level of contributory negligence [21].
10. In response to this submission, the Court of Appeal, echoing the approach of HHJ Cotter QC below, first emphasised the general point that caselaw under s1 of the 1945 Act was unequivocal: both causative potency *and* culpability must be considered (Lloyd Jones LJ @ [22-24]). In particular the CA placed reliance on a long citation of Lord Hoffmann from *Reeves v Commissioner of Police of the Metropolis* [2000] 1 AC 360 @ 371 in which Lord Hoffman had emphasised that the touchstone test under s1 (namely mutual ‘responsibility’) not only incorporated the notion of culpability generally but, in deciding how to weigh that culpability, permitted the Court to consider the policy of the statute which had imposed the duty on the Defendant in the first place i.e. if the statute was intended to protect a worker from his own folly then folly by the worker should be considered leniently when assessing the level of contributory negligence [23]
11. The Court of Appeal gave short shrift to the novel arguments that contributory negligence by the victim which either arose from activities lying outside the scope of the duty of care owed by the tortfeasor to him or even lying outwith any workplace activity at all should somehow be treated differently to ‘ordinary’ want of care by the victim. The Court simply

recorded that the language of s1 of the 1945 Act was sufficiently wide to cover both situations in the ordinary way (ie including an assessment of culpability) [24-25]

12. Next, there was a slightly strange argument put to the effect that where public policy did not require the Defendant to accept the ‘lion’s share’ of liability, then by some analogy with Section 2 Civil Liability (Contribution) Act 1978, the only issue to be considered should be that of causative potency¹². The Court of Appeal rightly rejected this argument on the basis that the jurisprudence under s1 of the 1945 Act was to be kept separate from that of s2 of the 1978 Act – notwithstanding the similarity of language. [27] This rejection was clearly right since the legal basis of apportionment between Defendants inter se may be very different from that of contributory negligence. Take, for example, a victim who is injured by one person (partly through that person’s fault and partly through the fault of the victim himself) but where the tortious person stands in some form of contractual relationship with a third party requiring the third party to meet the tortious party’s liability. The tortious party pays so much of the victim’s damages as the 1945 Act demands but thereafter may receive a full indemnity from the contracting third party (say under a denied contract of indemnity) even though the third party is in no way factually responsible for the infliction of the injury: the calculation of the contributory negligence and then the apportionment between the defendants are wholly separate processes (see *Fitzgerald v Lane* [1989] 1 AC 328). What the Court of Appeal could have gone to observe (but did not) was that the Defendant’s basic averment was wrong in any event: where two tortfeasors seek contribution from each other under the 1978 Act then (just as in the 1945 Act) the Court is required to consider both causative potency and culpability (*Downs v Chappell* [1997] 1 WLR 426). I will return to the operation of the 1978 Act one last time when considering the many unanswered questions in ALC below
13. There then followed the most significant line of attack from the Defendant and the one which the Court of Appeal recorded had taken the ‘greater part of the hearing’.
 - 13.1. I have not seen the skeleton arguments, still less a transcript of the proceedings or the evidence and so to reconstruct that argument, I am reliant on the published judgment. However, it appears that the argument proposed by the Defendant was as follows:
 - (a) It is fair to draw an analogy between assessing the level of contributory negligence in ALC cases and assessing the apportioned level of contribution which one tortfeasor has made to the overall level of risk in a mesothelioma case.
 - (b) *Barker v Corus* [2006] 2 AC 572, in explaining the rationale of *Fairchild v Glenhaven* [2003] 1 AC 32, held that in mesothelioma cases, the tortfeasor’s liability was limited to the precise degree by which it had increased the risk of the victim developing that disease when compared with the total lifetime exposure experienced by the victim i.e. the assessment of the tortfeasor’s liability was based solely on the degree of culpability without reference to culpability.

¹² s2(1) of the 1978 Act states ‘...in any proceedings for contribution under Section 1 above the amount of the contribution recoverable from any person shall be such as may be found by the court to be just and equitable having regard to the extent of that person’s responsibility for the damage in question’

- (c) In *Heneghan v Manchester Dry Dock* [2016] 1 WLR 2036 the Court of Appeal had held that *Fairchild* approach had been extended to ALC cases *once an anterior question had been answered, namely could it be shown on balance that the cause of the lung cancer in that particular case had been asbestos and not smoking.*
- (d) It followed that therefore *Heneghan* must necessarily have imported the *Barker* ‘explanation’ of *Fairchild* into ALC and hence when assessing liability as between the tortfeasor and the victim, it was fair to simply limit the assessment of the respective parties solely by reference to an assessment of their relative levels of contribution in fact – the contribution made by the victim via smoking and the contribution made by the tortfeasor by the exposure to asbestos.
- (e) The Court of Appeal had amplified the submission they had received on this by reference to the dictum of Lord Hoffmann in *Barker* where he had said @ [41]

“If the Defendant is deemed to have caused the mesothelioma but the claimant, like Mr Barker, was himself responsible for a significant period of exposure, the court may find that he did not take adequate care for his own safety or was in breach of safety regulations and, as Moses J did in the Barker case, reduce the damages for contributory negligence. On the other hand, if liability is several, there is no question of contributory negligence any more than of contribution. A defendant is liable for the risk of disease which he himself has created and not for the risks created by others, whether they are defendants, persons not before the Court or the Claimant himself” (Underlining added)

- (f) Thus via this analogy, it was said, it was right and proper to limit this Claimant’s recovery via the 1945 Act by the degree to which he had contributed to his ALC.

13.2. There are so many issues arising out of this submission (not all of which were adverted to by the Court of Appeal) that it is difficult to know where to start

- (a) First, and foremost, there is nothing to suggest within the Judgment that D ever grappled with the reality that its submission was in reality not one involving contributory negligence at all. Lord Hoffmann (in the underlined passage above) had made it abundantly clear that he drew a sharp distinction between those cases involving indivisible injury and those cases involving divisible or ‘several’ injury (See 7.6(d) above). He did so by holding that the gist of liability for mesothelioma was the creation of risk and by declaring that risk was infinitely divisible: thus mesothelioma was, to all intents and purposes a divisible condition. It was in that context that he held that a tortfeasor was liable only for his own proportion of the risk. Equally, it was for that reason I emphasised in setting out the background to ALC that contributory negligence required the presence of indivisible injury. In the case of divisible injury there was simply no basis upon which contributory negligence could operate for the reasons given above. Thus in the appeal it would appear that the Defendant accepted that the Judge was right to find that contributory negligence was the technique by which the Court would reduce the damages otherwise recovered by the victim in

order to reflect his ‘guilty smoking’. It must therefore have accepted that lung cancer was an indivisible injury such that the 1945 Act could apply. However, in order to criticise the level of contribution found by the trial Judge, the Defendant chose to use an analogy which, in order to operate at all, had to assume that lung cancer was divisible. Thus to limit apportionment to the consideration of contribution-only actually had nothing to do with contributory negligence at all but rather the apportionment of a divisible condition.

- (b) Lloyd Jones LJ dealt with this aspect by pointing out @ [34] that the Defendant had expressly chosen to concede breach and primary liability by accepting the model of doubling of the risk. As I set out above that necessarily means that it also accepted having made a material contribution to a (plainly) indivisible condition. At that stage it could not argue for the Hoffmann approach since (as we have already seen) that was to treat mesothelioma as if it were divisible). A necessary concomitant to liability for making a contribution in fact to indivisible injury which rendered the tortfeasor, prima facie, wholly liable to the victim (again as set out above) was the balancing availability of contributory negligence. Thus the availability of contributory negligence and, by extension, the rules governing the level of such contributory negligence, were ‘hard wired’ into the basis upon which this Defendant chose to concede breach.
- (c) Thus, the Court of Appeal held there was nothing in the analogy being drawn and overall the approach by the trial Judge was impeccable. The appeal failed.
- (d) There are two intriguing factors in respect of the Defendant’s *apparent* argument to the Court of Appeal (again – I have not seen the skeletons and so I do not know whether these issues were in fact raised but simply ignored by the Court of Appeal or whether they were not raised at all). Either way they are of some moment.
 - (i) It is difficult to see what relevance *Barker* could ever have had to any aspect of any ALC case. Lord Hoffmann expressly excluded the operation of his approach to lung cancer cases because they were multi-factorial (as opposed to mesothelioma which can only be caused by asbestos exposure and where the risk of developing mesothelioma is directly proportionate to the level of exposure)

“24 If the distinction between [Fairchild](#) and [Wilsber](#) does not lie in the fact that in the latter case a number of very different causative agents were in play, I think it would be hard to tell from my [Fairchild](#) opinion what I thought the distinction was. 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”

The Defendant might seek to argue that where the trial Judge had determined which was the more likely to have been the causative agent (smoking) then the injunction contained in the last sentence of Lord Hoffmann’s dictum above (against using *Fairchild* in a lung cancer case) was lifted. But that would be to miss the point that Lord Hoffman was referring to proving which agent was more likely to have been the cause of the injury in fact and not merely proving which agent had been the greater contributor to the *risk* that the cancer occurred. Once this is understood, a moment’s further thought would show why *Barker’s* inapplicability to causation in a multi-factorial case such as ALC rendered it equally inapplicable as an analogy for limiting contributory negligence assessment to one of causative potency only.

- * One can see how, in a mesothelioma case, multiple employers acting sequentially can each add an identifiable amount to overall risk of mesothelioma such that one can limit their liability to the degree of exposure involved. This was the factual underpinning of Lord Hoffmann’s approach in *Barker* and hence was the basis of his above dictum.
- * However, Lord Hoffmann’s approach would be impossible where two or more agents are acting synergistically (as occurs in lung cancer) where the two agents of asbestos and smoking co-exist. First each risk interacts with the other – they do not exist within the body as separate free standing cumulative risks as the separate exposures to asbestos were considered to do within a mesothelioma case; second, a necessary concomitant of the fact that their mutual effect is multiplicative is that the two agents must be acting simultaneously *not sequentially*. Put another way, unlike sequential separate exposures to a single agent, the effect of each employment tested against the smoking background cannot be severed either one employment from another or as against the smoking as a whole
- * Third, the presence of the Shortell Fallacy means that one cannot decide empirically which risk to take as the background risk. This is another concomitant of the fact that the risks are multiplicative.

- (ii) The second intriguing *apparent* omission from the Defendant’s case was any reference to the fact that, *Barker* was in fact reversed by statute, namely the Compensation Act 2006; *Section 3(3)(b) of that statute expressly reserved the right of the exposers to plead contributory negligence*. The technique of the Act permitting

the continued availability of contributory negligence was to reverse *Barker's* holding that mesothelioma cases (being based on the creation of risk) constituted a divisible condition. The Act effectively declared instead that mesothelioma was an indivisible condition (s3(2)(b)). As we have seen above, once it was an indivisible condition then contributory negligence was available to be alleged by the tortfeasor

- What was/would have been the Defendant's case on this? That the operation of S3 did not extend to lung cancer cases? It is true that s3(1) limits the operation of the Act to mesothelioma and so, prima facie, it would not apply to Lung Cancer claims and hence might not be thought to be relevant. Further, it has been held (*Zurich v IEG [2016] AC 509*) that where Section 3 does not apply, *Barker* remains good law.
- But the basis of Section 3 technique is what is important. It emphasised that the mischief of *Barker* was the treatment of mesothelioma as an divisible injury. But Section 3 limits itself to mesothelioma because *Barker* limited itself to mesothelioma. Thus the *existence* of Section 3 highlights the limitation of *Barker* to the very special circumstances of mesothelioma and helps to highlight the importance of Lord Hoffmann's denial that anything said in *Fairchild* and hence *Barker* could have relevance to multi-factorial lung cancer cases.
- Whilst therefore, with respect, the Court of Appeal in *Blackmore* were entirely correct to reject the analogy with *Barker*, overall, its reasoning as set out in [33], namely "*Barker v Corus establishes that liability under this principle is several as opposed to joint and several. Accordingly each Defendant is liable only to the extent that he has caused an increase in the risk of the damage occurring. As a result, questions of contributory negligence never arise*" does not necessarily set out the full story. The full story was that *Barker* never applied to ALC cases in the first place; and (in the alternative) insofar as it did *Barker's* use of aliquot-share-liability-only analysis was reversed by s3(3)(b) of the 2006 Act. That reversal emphasised that *Barker* could have no relevance in any condition which was beyond any shadow of contention, indivisible in nature.
- This should be borne in mind when *Heneghan* comes to be considered.

13.3. Before leaving the part of this paper dealing with an analysis of what was being argued by the Defendant in *Blackmore* and moving on to consider *Heneghan* and the nature of the major questions remaining in the area of litigating ALC, it is perhaps useful to note that this is apparently the second time that a Defendant has sought to import notions relevant to *Fairchild* to ALC (the first was in *Heneghan* as we shall see below). From the Defendant's perspective this is far from risk free. There are two principal risks created.

- (a) The first is that one immediately imports jurisprudential uncertainty. Having seen which way the wind was blowing in the House of Lords in *Fairchild*, we submitted to the House on behalf of the Defendants that they were at grave risk of opening Pandora's Box since the ramifications of what they were about to do simply could not be foreseen. This proved accurate

– since the Courts have struggled thereafter to even define what the ratio of *Fairchild* was, it can hardly be a surprise that they then struggled to see how the ratio fitted in to the pre-existing weave of the Common Law thereafter (whether by reference to divisibility (*Barker*) or its relationship to the ‘background’ risks (*Sienkiewicz*) or the triggering of insurance contract liability (*Durham v BAI*) or liability for periods of non-insurance (*IEG v Zurich*). Each declaration has led onto the next logical problem further down the line in its application. As we shall see in a short while, when *Fairchild* was given limited application in *Heneghan* the species of uncertainty created there was how contributory negligence and contribution could ever be held to exist at all.

- (b) The second principal risk in invoking *Fairchild* into ALC from the Defendants’ perspective is that for all the advantages this may give in respect of divisibility of risk – and hence diminution of damages – in any individual case, the Court is eventually likely to impose primary liability for ALC based upon mere increase in risk. This eventuality would lead to an increase in insurers’ IBNR¹³ which would at least equal that of mesothelioma.

13.4. How, then, did the Defendants invoke *Fairchild* in *Blackmore*? The short answer is that they did not do so overtly at all: not least because it is not clear that they appreciated that their reliance on *Barker* actually had that effect – but, in fact, it did.

- (a) Suppose a victim is responsible for creating the same level of risk (through smoking) that employer A did via exposure of that victim to asbestos. Now, further let it be supposed that employers B and C subsequently and sequentially also exposed the victim to precisely the same degree as A but B and C cannot be sued by the victim for whatever reason.
- (b) On the Defendant’s argument in *Blackmore* it is not at all clear whether they were suggesting that the victim obtains 50% of his damages for ALC (ie because as between him and Employer A – the only employer sued - there was an equal contribution to the risk) or 75% of his damages (ie since throughout his life his smoking created the same risk of ALC as Employer A and Employer B and Employer C and which therefore meant his own contribution was ¼ of the total risk). However only the latter result would be consistent with the result in *Barker* and so it has to be the solution which the Defendant would have urged upon the Court.
- (c) This latter solution would actually leave the victim worse off because he would only be receiving 75% of the proportion of his damages against A:

Victim is responsible for 25% of his total risk
 Employer A is responsible for 25% of his total risk
 Employer B is responsible for 25% of his total risk
 Employer C is responsible for 25% of his total risk

¹³ “*Incurred But Not Reported*”

But B and C have not been sued and, as we can see from the dictum of Lord Hoffmann above, on the Defendant's theory this is simply the victim's misfortune: A does not pick up the liability of B and C and pay it to the victim. (Again, I emphasise as I did above that actually none of this works because it is impossible to see how contributory negligence exists in the same situation as divisible injury – but all I am doing here is following through the apparent logic of the submissions made in *Blackmore*).

- (d) Thus, following the logic of the submission, where there are multiple expositors some of whom are not before the Court, the victim loses out.
- (e) This is, of course, consistent with the result in *Barker*
- (f) The Court, had it acceded to the Defendant's submission, would have been forced to hold as the necessary quid pro quo that the basis of liability was increase of risk i.e. the Defendant's *Barker* analogy, if successful, would have necessarily imposed Fairchild into ALC cases (albeit without the applicability of s3 to convert apportionment-only level of damages to full verdict damages). Not only would this create a wholly new category of victims it would massively increase the transaction cost of each case thus brought as apportionment was worked out on the facts.

The wider picture

- 14. A series of wider issues arise when the authority of *Blackmore* is compared to the equally interesting case of *Heneghan*. I admit at once that the questions are more easily posed than answered but it will be the job of practitioners in this area to advise on them.
- 15. First we need to consider the facts in *Heneghan* insofar as they related to exposure:
 - 15.1. This was also a lung cancer case where the victim had both been a smoker and exposed to asbestos.
 - 15.2. His asbestos exposure history spanned a number of companies and is set out in tabular form below. The following features are of importance to note:
 - (a) Only a minority of his exposure was constituted by Defendants actually before the Court
 - (b) The majority of his exposure was caused by X Co Ltd – which was not one of the Defendants before the Court
 - (c) Some of the Defendants before the Court had, themselves, been responsible for very little of his overall exposure to asbestos. None of the Defendants before the Court could, themselves, have more than doubled the risk of lung cancer developing.
 - (d) The total exposure history c133 fibre/ml years

- (e) Based on that figure the Court accepted that the risk of lung cancer arising from the asbestos exposure alone was (x5) of the background¹⁴.
- (f) There appears to be a fundamental difference in its facts than those of Blackmore namely that in Heneghan there were multiple asbestos exposers (both sued and unsued) whereas in Blackmore it does not appear from the Judgment that there was ever any more than one exposers.

Heneghan: the exposures

SOURCE (All)	SOURCE (D's)	FIBRE/ML YEARS	%
Unknown		11.6	8.8%
X Co Ltd (Not Sued)		74.5	56.0%
6 D's before the Court		46.9	<u>35.2%</u>
			100%
	D3		10.1%
	D4		2.5%
	D1-D2 & D5-D6		<u>22.6%</u>
			35.2%

- 15.3. The chest physicians (Drs Rudd and Moore-Gillon – the two pre-eminent medico-legal experts in this area) considered that the victim's smoking history when considered in isolation increased the risk of lung cancer by (x 4). It is unusual to see a case where the smoking history risk is less than the asbestos risk but this case was one such example. They also agreed that the total risk in risk in combination was said to be $4 \times 5 = (x 20)$.
- 15.4. At first instance in *Heneghan [2014] EWHC 4190 (QB)* the Claimant suggested an elegant solution to the approach to causation in lung cancer cases. They posited a two stage test in order to prove causation in such cases:
- (a) First, the Court would identify what had caused the lung cancer (the 'what' question). If the Court did not conclude that the cause was asbestos then the Claimant accepted that it failed at this first hurdle [41] (First Instance)

¹⁴ The calculation was relatively simple. As we have seen the level of exposure at which risk is doubled is said (pursuant to the Helsinki criteria) to be 25 fibre/ml years. 133 is c. x5 higher than 25.

(b) Second, the Court should go on to consider the ‘who question’: that is which of the Defendants before the Court had materially contributed to the overall dose. It was at this stage that the nimble nature of the Claimant’s case became clear. They expressly eschewed reliance on *Fairchild* in this respect. They did so because they would have known at that stage that the Supreme Court were about to hear *IEG v Zurich* and would there be invited to hold that where *Fairchild* was invoked and, for whatever reason, s3 of the Compensation Act 2006 was held not to operate, *Barker* remained good law and hence any damages award would be made subject to apportionment. Thus, for the Claimants to have invoked *Fairchild* in the present case would have been to court a large deduction. The Claimant went about their submission by the following progression

- Whilst Dr Rudd considered that every fibre should be taken to be a material contribution in fact to the cancer sufficient to render each party who made a material contribution to the dose liable as the cause of the entire injury (subject to double recovery and contribution interest) (*Bonnington*)
- But in fact it was entirely possible to infer that the creation of a material increase in risk was a contribution to the disease in fact (*This is still a submission relying on Bonnington and not Fairchild*)
- The Claimant could claim against each Defendant in full even though each Defendant before the Court had only caused a minority of the exposure (See [38-39] of the Judgment in *Heneghan* at First Instance).

(c) The Defendant’s response was not to argue that the Court could simply ask whether each Defendant could be shown to have more than doubled the risk of causing lung cancer. Nor, indeed did the Defendant in *Heneghan* argue against the need for the existence of the First Question. Instead the Defendant argued that once Stage 2 was reached the Court had no choice but to either find that the Claimant failed against each Defendant since none had individually more than doubled the risk (an orthodox approach) or to apply *Fairchild* and be clear that simple increase in the material risk was sufficient to found breach but that the recovery should be limited to the level of the increase of risk for which each Defendant was liable. In this respect the difference between the Defendant and the Claimant’s respective approaches was that the Defendant did not require the Court to make the intermediate inference of fact that material increase in risk was itself sufficient to make a finding that the exposure had materially contributed to the disease *in fact* so that *Bonnington* could be invoked. This was an interesting submission since it was another invocation by a Defendant of *Fairchild* into a new class of disease

15.5. Jay J accepted the two stage approach where there were multiple Defendants before the Court [57] First Instance.

15.6. Stage 1: the ‘what’ question

(a) Jay J recorded [15] the conclusion of the experts, namely that:

“3. On the balance of probabilities he would not have developed the lung cancer if he had not been exposed to asbestos....58 Given paragraph 3 of the experts' Joint Statement....Medical causation was not, therefore, an issue at this trial. The reason why the experts have attained common ground on this issue is because analysing the epidemiological evidence and the deceased's personal circumstances clearly establishes that it is more likely than not that his asbestos exposure was causative rather than his smoking. ... a holistic view of the evidence has led to the robust conclusion that the relative risk of asbestos being the culprit in the deceased's case is more than 2:1; or, as the common law would express the same point, the case has been proved on the balance of probabilities”

- (b) *It would appear that Jay J's answer to causation was being led directly into the Shortell Fallacy – in this case the form of Fallacy was that he chose to accept smoking was the background risk against which the effect of adding asbestos was tested without explaining why. Asbestos was 'clearly' only the cause of the asbestos, on balance of probabilities, if one assumes that the smoking risk (x4) was the background risk against which the overall rise in risk caused by the addition of asbestos was tested (ie by raising the risk from (x5) to (x 20)). On these facts if one assumed that the asbestos exposure was the background risk then it was the smoking which was responsible (raising the risk from (x5) to (x20)). (See [58: 67] First Instance*

15.7. The Second Question: 'the who' question

- (a) *Jay J rejected the evidence of Dr Rudd that each fibre inhaled could be taken to have been a material contribution to the lung cancer in fact. Thus he found that the fact that an asbestos fibre (or any collection of them) could cause cancer, it did not mean that they necessarily did cause it. [63]*
- (b) *On that basis he was able to dismiss the argument that Bonnington applied (since Bonnington requires proof of contribution to injury in fact and not merely to risk) and hence the notion that each Defendant making a material contribution to the dose should be held liable for the full amount (see 7.2(f) above).*
- (c) *Jay J found (quite correctly) that none of the Defendants could prove 'the who' question by reference to having been shown individually to have been responsible for a dose which more than doubled the risk [61 and 62] First Instance.*
- (d) *Having rejected doubling the risk on the facts and Bonnington on the inability of the epidemiology to prove causation in an individual case Jay J felt himself to be in the dilemma of having either to find wholly against the Claimant or a frank invocation of Fairchild albeit*
** Only at the second stage; and*
** Without s3 Compensation Act operating to render each tortfeasor liable for the whole Thus he applied (effectively) Fairchild as limited by Barker to lung cancer cases and permitted C to recover a proportion of his damages against the Defendants before the Court but not permitting his damages to exceed the total percentage of his exposure to asbestos as stood before the Court.*

But, of course, he could only go on to carry out the apportionment exercise between the tortfeasor by wholly ignoring the effect of smoking. What is not clear are the bases for the unspoken assumptions that either the smoking would have

interacted with each period of asbestos exposure identically (thus rendering it possible perhaps to ignore the presumed-uniform effect of smoking) *or* for why one did not make a prior deduction for the effect of smoking before one then made the apportionment for the effect of each tortfeasor's dose on the overall risk. The job of the First Question in deciding that the cause of the lung cancer swept all these issues aside by determining that the cause of the lung cancer was asbestos and not smoking. But, as we have seen, where the underpinning for answering that question in the way it appears uncertain, this exercise of apportion carried out in the Second Question which was based upon the First can hardly be more jurisprudentially certain.

- 15.8. The Court of Appeal did not disturb these findings but rather than approach the points set out in that Judgment seriatim, it is probably of greater help to consider the problems arising in ALC cases through the prism of that Court of Appeal judgment and its relationship to the Court of Appeal in *Blackmore*.

16. Problems

- 16.1. When considering the problems of the interaction between the two Court of Appeal judgments, it is necessary to bear in mind the chronology
- (a) *Blackmore* (First Instance) was heard before *Heneghan* (First Instance)
 - (b) *Heneghan* (First Instance judgment) does not refer to *Blackmore* (First Instance)
 - (c) *Heneghan* (Court of Appeal) was heard before *Blackmore* (Court of Appeal). The Court of Appeal in *Heneghan* did not refer to *Blackmore* (First Instance)
 - (d) *Blackmore* (Court of Appeal) does refer to *Heneghan* (Court of Appeal) but only en passant [33] (*Blackmore CA*)
- 16.2. The first problem is the treatment of smoking as between the two Court of Appeal decisions. It is simply not clear from the judgments in *Heneghan* how smoking was dealt with – whether any deduction was made for contributory negligence and if so, on what basis? Indeed it is possible to read *Heneghan* as prohibiting any deduction being made for smoking history. The presence of the Stage 1 question: what caused the lung cancer precludes it. If one answers Stage 1 ‘On balance the lung cancer was caused by the asbestos’ or ‘on balance but for the asbestos it would not have occurred’ then it follows as a matter of ineluctable logic that smoking was not the cause. If smoking was not the cause then, again, as a matter of logic it cannot have had any causative potency (however blameworthy it was on the part of the smoker). Without causative potency one of the two limbs of s1 Law Reform (Contributory Negligence) Act 1945 is hacked away and there can be no deduction for contributory negligence for smoking. This issue was not considered in the judgment of either the High Court or Court of Appeal in *Heneghan*. Given that contributory negligence was the gist of the appeal in *Blackmore* it is interesting that the Defendant did not require some consideration by the Court of Appeal of this aspect of *Heneghan*.

16.3. Equally, it might be added, what happens if there is on the facts of an ALC case a single predominant exposor who on its own and (in contradistinction to any other exposor in the case) was responsible for more than doubling the risk? Surely, following the logic of the first question, it would then be possible to say not only that asbestos, on balance, the cause of the lung cancer, but that this single dominant exposor was the cause. If that is so, then how does that exposor seek contribution from any other exposor under s2 Civil Liability (Contribution) Act 1978 since, as we have seen, that Act also requires there to have been causative potency in the default of the party against whom the Defendant seeks contribution. The first question in such circumstances requires the other Defendants to get off ‘scot-free’. Indeed, it is not entirely clear why in *Heneghan* where there was such a predominant exposor, the logic of that exposor not being before the Court did not mean that the Claimant failed completely. It seems that the only answer to that question was the perceived unfairness of such a situation in the eye of Jay J and the Court of Appeal in *Heneghan*. Whilst this is entirely understandable, it is not necessarily the firmest basis for an entire branch of jurisprudence.

16.4. What is the soundness of the First Question?

- (a) Although the existence of the two question approach was endorsed by the Court of Appeal in *Heneghan* it was not asked at first instance in *Blackmore* (although that case pre-dated *Heneghan* of course, it seems that the application of the question had not arisen independently to the Court in that case). Of greater moment is that its necessity was not addressed at all in *Blackmore* in the Court of Appeal. Is the two stage question therefore approved or not?
- (b) The requirement of a two stage test is entirely novel in any event and certainly found no echo in *Fairchild* or indeed anywhere else that I can find.
- (c) As a gatekeeper test its shortcomings have already been pointed out but here is another: it renders it entirely random circumstance whether my 1 fibre/ml exposure of an employee is actionable or not: it is actionable if the employee has the misfortune to be heavily exposed elsewhere such that his total life dose of asbestos renders the subsequent lung cancer deemed to have been caused by asbestos: it is not actionable if the same victim is never exposed elsewhere to asbestos yet still has developed lung cancer. This lottery aspect probably also tells against the validity of the First Question

16.5. The soundness of the ‘Second Question’

In *Heneghan*, as we have seen, the Court sought to get around the problem of contribution by rendering each Defendant before the Court liable only for its own aliquot share (following *Barker*). But there are four major problems with this approach: the first three of which set out below are repeats from the analysis above but I refer to them briefly again here to identify and gather them as ‘Second Question points’.

- (a) First, the approach of breaking down each Defendant’s aliquot share into the proportion of the total asbestos exposure ignores wholly the ‘elephant in the room’ namely how did each asbestos exposure period interacted with the smoking history.
 - (b) Second, how can the single predominant exposor considered to be the cause of the lung cancer ever obtain apportionment under the 1978 Act?
 - (d) Third, it is not at all clear to what extent *Barker* can ever apply to lung cancer cases since in *Barker* itself given Lord Hoffmann’s dictum at [24] –
 - (e) Fourth, how does the *Heneghan* basis of liability (which is emphatically not based on proof of showing material contribution to the disease) lead to any policy of insurance triggering by any proper construction of the contract? It would therefore appear likely that the trigger litigation would have to be fought again in the context of ALC if the *Heneghan* two stage approach were to be adopted
- 16.5. Whilst it is true that in both *Blackmore* and *Heneghan* ‘doubling of the risk’ was accepted to be basis of liability in ALC cases, there were differences in conclusion as to the ramifications of that test

- (a) In *Blackmore* (as we have seen), in order to underpin the overall conclusion that contributory negligence was available and to be implemented in an orthodox manner the Court found that proof of doubling of the risk was proof of actual contribution to injury and thus rendered the tortfeasor liable for the whole damage

“34...liability on the basis of doubling the risk is founded on orthodox principles of causation. It proceeds by drawing an inference from the increase in risk of contracting the disease that the agent in question was a cause of the disease....Heneghan [8]..Where liability is established in this way, a Defendant who has made a material contribution to the damage is, prima facie liable for the full extent of the damage suffered (Bonnington Castings Ltd v Wardlaw...It is at this point that questions of contributory negligence may arise”

- (b) Somewhat surprisingly, given both the recitation of *Heneghan* and *Bonnington*, the Court of Appeal in *Heneghan* had come to a diametrically opposed view

“(Citing Jay J at first instance with approval, Lord Dyson MR

“34...Questions of material contribution arise only if a connection between [the victim] inhaling asbestos and his developing cancer was established. Knowing that inhaling asbestos can cause cancer does not entail that in this case it probably did. For the reasons given earlier that inference cannot be drawn in this case. Questions of material contribution do not arise...”

40. *Swift J (in Jones) therefore decided that she should not approach a case of lung cancer by applying the Bonnington Castings principle...*

41 *Mr Allan does not submit that the decision in the [Jones] case is wrong In my view he is right not to do so...I agree ...that the Bonnington Castings principle could not be invoked [in lung cancer cases]”*

- (c) If the Court could not find that doubling of the risk equated to contribution to the injury in fact, then special rules were required on causation according to the Court of Appeal in *Heneghan*. There was nothing ‘orthodox’ about them. It was entire inability of the Court to draw the inference that doubling of the risk actually meant contribution to injury in fact which had led Jay J to adopt his two question approach (which was then approbated by the Court of Appeal)
- (d) So, to crystallise the difference, the question arises – what is the effect of proving a doubling of the risk of ALC from asbestos exposure? Is it to prove that the party alone responsible for doubling the risk by itself is the sole cause of the ALC or that any employer who has contributed to the overall asbestos fibre burden is to be taken to have materially contributed to the ALC in fact (*Blackmore?*) or is it simply to act as a ‘gatekeeper’ provision which then permits recovery against each employer before the Court only to the level of the overall proportion of the contribution?

- 16.6. It seems to me that the *Heneghan* two stage approach is neither ‘fish nor foul’. The only two legally valid approaches to causation are either the need to prove mechanistically (ie by reference to biological evidence pertinent to the individual case and not epidemiology) material contribution to the ALC from asbestos as a matter of fact (in which case *Bonnington* will apply) or, at the other extreme, to apply *Fairchild* completely to ALC cases (in which *Fairchild* will, of course, apply). Obviously the financial implications of this are to be measured in the billions of £.
- 17. The second great area to be determined in the future in respect of ALC is the issue of knowledge of smoking by asbestos victims when assessing contributory negligence.
 - 17.1. In *Badger* the Court helpfully set out in an Annexe something of the history of knowledge of smoking and I recommend you read them.
 - 17.2. The date when smoking warnings were first placed on packets was 1971. Should this be the date? I think perhaps not.
 - 17.3. Consider an analogy. In *Bussey v East Anglian Heating* my instructions are to invite the Court of Appeal to consider whether *Williams v University of Birmingham* sets out correctly the test at which the level of exposure to asbestos mandated action by an exposer. The Court will be invited to conclude that there is no distinction to be made between the level at which a foreseeable risk of injury occurs and the level at

which a duty to take remedying action arises. In commenting on *Bussey*, David Pugh of Keoghs said, with characteristic perception

“What was the true state of reasonable knowledge of SME employers in, say, 1968? How would a small plumbing business even know about TDN 13? Claimants still carry the burden of proof, even in mesothelioma claims. Setting aside TDN 13 might not necessarily turn out to be the claimants’ advantage”¹⁵

By that he is adverting to the fact that there were, until the 1970s, documents which appeared to give equivocal or conflicting advice. Thus, it is said (the merits of which lie outwith the scope of this paper), it may be that the level of exposure at which foreseeable risk was created was higher as a result of this equivocation. This argument has ramifications in respect of contributory negligence for smoking of course. It could equally be argued by a victim that their smoking knowledge was also rendered less culpable because of the existence of contradictory information. By way of example the smoker may be able to consider what was said by representatives of the Tobacco Manufacturers Association as late as 1998, namely that whilst there may have been a statistical association between smoking and lung cancer, there was not proof of actual causation in any particular case. Also the paradigm ‘healthy’ nonagenarian who had smoked all their life etc. (See <http://www.who.int/tobacco/media/en/TobaccoExplained.pdf>)

18. The third area in respect of ALC development which will require attention and, in particular, with respect to contributory negligence is the addict’s dilemma. The more they smoke (and hence the higher the causative potency) then (one presumes) the more addicted (physically and psychologically) they may be presumed to be and hence the less they can control their habit and the less morally culpable they are. How is that to be reflected in deduction for contributory negligence.
19. The fourth and final area for any future litigation development in ALC cases, is the need to master the epidemiology and microbiology permitting us to assess what the effect of ‘innocent’ smoking. This is complex. This is demonstrated by the following statistics cited in *Badger*

“18 In their supplemental joint statement of 9 November 2005, the doctors set out the following points of agreement:

- *(1) Lung cancer is an indivisible injury. We understand this as an all-or-none injury. It is not a cumulative injury for which incremental exposure to a causative agent, for instance asbestos fibres or cigarette smoke, results in increasing severity of the injury itself.*
- *(2) Had it not been for his exposure to asbestos, sufficient to cause asbestosis, it is unlikely that Mr Badger would have developed lung cancer at the age of 63. Had it not been for his cigarette smoking it is unlikely that he would have developed lung cancer at the age of 63. Had it not been for his asbestos*

¹⁵https://keoghs.co.uk/keoghs-insight/client-alerts/bussey-v-anglia-heating-mesothelioma-and-minimal-exposure-williams-upheld-for-now?page=2&type_of_insight=client-alerts

exposure, sufficient to cause asbestosis, and his cigarette smoking, it is unlikely that he would have developed lung cancer at the age of 63.

- (3) *The study of Peto et al indicates that, if Mr Badger had stopped smoking at an earlier age, his cumulative risk of developing lung cancer would have been less than his actual risk, given that he had continued to smoke until the time that his lung cancer was diagnosed. Extrapolation from figure 3 in the publication indicates the following cumulative risks of developing lung cancer at age 63 at various ages of stopping smoking:*
 - 0.7% at age 30
 - 1.3% at age 40
 - 2.2% at age 50 years.

How that fits into the model of multiplicative effect perhaps awaits further definition.

Summary

20. The conclusions I draw from this are:
 - 20.1. No satisfactory test for lung cancer causation where asbestos and cigarette smoke are factors is, as yet, being applied by the Courts.
 - 20.2. ‘Doubling of the risk’ is a perfectly adequate test where there is only ever a single agent which could cause the outcome and where *either* some part of the exposure is tortious and some part is not *or* the total tortious dose is spread across multiple expositors.
 - 20.3. ‘Doubling of the risk’ can never properly deal with a situation where, added to the background risk of an outcome
 - (a) Two or more agents are each causatively active; and
 - (b) Those two or more agents are acting with multiplicative effect in combination.
 - 20.4. ‘Doubling of the risk’ fails as an analytical tool in the above circumstances because
 - (a) the ‘Shortell Fallacy’ means that any scientific rigour present in assessing the risk created by each factor is lost when one seeks to use that information to prove causation because the Court is immediately faced with a pure value/policy judgment, namely ‘which of the two factors do I *choose* to treat as the background risk against which I shall test the effect of the increase created by the introduction of the other factor’;
 - (b) Even if the ‘Shortell Fallacy’ is overcome the Court is then faced with the further value/policy judgment ‘do I *choose* (or am I free) to deem that the agent which more than doubled the risk has materially contributed to the injury in fact?’
 - (i) If ‘yes’ then the result is that under application of the normal rules of causation for an indivisible condition, any tortfeasor is liable to

the victim for the full amount provided that the tortfeasor's contribution was greater than *de minimis*: this seems unfair to the Defendant;

- (ii) If 'no' then how, can the Court justify any system of apportionment (both between the tortfeasor and the victim and between tortfeasors) which is both consistent with the common law and the operation of the Law Reform (Contributory Negligence) Act 1945, Civil Liability (Contribution) Act 1978 and s3 Compensation Act 2006?
- 20.5. That the technique adopted in *Heneghan* of asking the First Question in order to set up the operation of a *Fairchild* approach as the result of the Second Question does not appear to stand up to scrutiny. Alternatively its technique seems at odds with parts of the subsequent authority of *Blackmore* in the Court of Appeal.
- 20.6. As the common law stands at present (both in isolation and in combination with the above Acts) if lung cancer is to be permitted to be the subject of compensation at all then there are only 2 coherent tests available for causation in lung cancer cases:
- (a) Mechanistic/scientific proof at the cellular level that asbestos inhalation permitted by the tortfeasor before the Court made a material contribution to the appearance of the lung cancer present in any particular individual. A Claimant proving this is then entitled by operation of law (*Bonnington/Rahman*) to obtain full compensation against that tortfeasor.
 - (b) Adoption of *Fairchild* (including s3 Compensation Act 2006) into lung cancer. To maintain coherence s3 needs also to be adopted because the Shortell Fallacy prevents any rational method of apportionment either between Defendants or between the Defendant and the Claimant for the reasons given repeatedly above (ie the absence of knowing how differing periods in the smoking history interact with varying periods of the asbestos exposure history). Thus success by the Claimant as to 1 percent will lead to 100% recovery.
- Each solution is extreme. (a) will effectively bar recover at all; (b) will mean that anyone proving material increase in the risk of a cancer which is widespread and multi-factorial would allow full recovery following mere proven assertion of tortious exposure. When viewed on the level of the UK economy such an outcome would visit economic disaster upon insurers which could only be avoided by central subvention or price hikes to their policyholders etc.
- 20.7. Development of this area by caselaw is not proving to be a process which clarifies. This is no criticism of anybody. It is next-to-impossible in any case in which individual points are taken in respect of individual facts to see the whole warp and weft of the common law which could be affected by the determination of the individual point against its factual background and, indeed, it is only the Supreme Court which can truly attempt to do so.

- 20.8. So much money rides upon these questions that the time has come to consider a different approach to asbestos litigation. We should recognise that the manner in which this lethal industrial poison operates renders it as damaging to the body of the common law as to the bodies of its victims. To mix my metaphors somewhat, each novel solution reached by the Courts tends to have been both the child of the last novel solution which the Courts found necessary to adopt and thereafter, in its turn, becomes the father to the next.
- 20.9. In reality the driving issues here are the balance of the legitimate interest of the insurance market (which at present simply cannot predict how the behaviour of insured clients undertaken many decades ago in respect of asbestos will be visited upon the policy premium payers of today)¹⁶ and those of the victims who, to their stunned yet (usually) stoical amazement find themselves told that an innocuous dust with which they came into contact those same decades ago will now debilitate and then kill them.
- 20.10. Perhaps, at the risk of seeming unrealistic, the solution is an independent Commission mandated to consider arguments from both sides of the debate (with the Government firmly understood to be a tortfeasor and not a neutral regulator) to make recommendations about the operation of asbestos claims both as to breach and as to causation with the intention of suggesting a single comprehensive legislative code applicable to asbestos only and being seen to have no wider effect within the common law.
- 20.11. In the interim, any solicitor embarking on litigation in this area will need to satisfy herself that Counsel instructed is in a position to make submissions which show how the arguments being advanced fit in with wider strategic considerations and can present effectively, in addition, the medical and epidemiological evidence.

MICHAEL RAWLINSON QC¹⁷
07.08.17

APPENDIX 1:

EXTRACT FROM STANLEY BURNTON J IN *BADGER* ON SMOKING KNOWLEDGE

44 *The evidence summarised in the Appendix shows, in my judgment that by 1971, when the first health warnings were put on cigarette packets, it was reasonably foreseeable by a reasonably prudent man that if he smoked he risked damaging his health....*

Appendix

¹⁶ *We must not forget the position of the general taxpayer: government is a major tortfeasor and damages paid are funded by general taxation*

¹⁷ *Who asserts his moral right to be identified as the author of this document*

1. Leaving aside the prescient suggestion of Dr Adler in 1912, that lung cancer is related to smoking, the first scientific articles on the connection between smoking and illness date from 1950. A series of articles by, principally, Richard Doll and A. Bradford-Hill preceded a ministerial statement made on 12 February 1954 by the Minister of Health, Mr Iain Macleod, accepting the view that the statistical evidence pointed to smoking as a factor in lung cancer, although he stated that there was so far no firm evidence of the way in which smoking caused lung cancer or of the extent to which it did so. A further ministerial statement was made in May 1956 by R. H. Turton, the Minister of Health. He stated:

“Since my predecessor made a statement in February, 1954, investigations into the possible connection of smoking and cancer of the lung have been proceeding in this and other countries. Two known cancer-producing agents have been identified in tobacco smoke, but whether they have a direct role in producing lung cancer, and if so what, has not been proved.

...

The chairman of a committee of the Medical Research Council which has been investigating the subject considers that the fact that a causal agent has not yet been recognised should not be allowed to obscure the fact that there is, statistically, and incontrovertible association between cigarette smoking and the incidents of lung cancer. The statistical evidence from this and other countries to which he refers tends to show that mortality from cancer of the lung is twenty times greater amongst heavy smokers than amongst non-smokers.

The Government will take such steps as are necessary to ensure that the public are kept informed of all the relevant information as and when it becomes available.”

2. In 1960, a Government survey, admittedly based on a small sample of 83 adults and 71 young people, found that only one elderly non-smoker had not heard of the association of smoking and lung cancer. In 1962, the Royal College of Physicians published its report entitled “Smoking and Health”. It concluded:

“Cigarette smoking is a cause of lung cancer ... Cigarette smoking is the most likely cause of the recent world-wide increase in deaths from lung cancer, the death rate from which is at present higher in Britain than in any other country in the world ... Cigarette smoking probably increases the risk of dying from coronary heart disease.”

The report stated that the risk of dying of lung cancer was 16 times greater for a smoker smoking 20 cigarettes a day than a non-smoker.

3. On 12 March 1962, Enoch Powell, again Minister for Health, said in the House of Commons that the Government accepted that the Royal College of Physicians' report “demonstrates authoritatively and crushingly the causal connection between smoking and lung cancer and the more general hazards to health of smoking”. (Hansard , 12 March 1962, column 888, and (*ibid* , column 886):

“My Right Hon. Friend the Secretary of State for Scotland and I are asking local health authorities to use all their channels of health education to make the conclusions of the report widely known and to make clear to the public the dangers to health of smoking, particularly of cigarettes. We shall be giving them guidance and providing them with publicity material”

4. In July 1962, the Government launched a publicity campaign and funded cessation clinics. In 1968, Imperial Tobacco reduced the tar content of the cigarettes on health grounds. In 1969 the Chief Medical Officer stated that cigarette smoking probably resulted in some 80,000 premature deaths in England and Wales each year, and that for the whole of the United Kingdom the number must approach 100,000. In the same year, the Radio Times implemented a ban on cigarette advertising. In October 1969, the UK's Health Education Council ran an anti-smoking campaign with posters asking: “Why learn about lung cancer the hard way?” In the following year, the World Health Organisation banned smoking at its meetings, affirmed the health hazards of smoking and asked for a ban on advertising and promotion of cigarettes.

5. In 1970, the Chief Medical Officer described the cigarette as “the most lethal instrument devised by man for peaceful use.” In 1971 the Royal College of Physicians published a report entitled “Smoking and Health Now”. The introduction stated:

“The Royal College of Physicians drew attention to the dangers of cigarette smoking in its report Smoking and Health in 1962. In spite of all the publicity it received, most smokers are still unaware of the risks they run; but doctors and other professional men are stopping smoking in large numbers.”

The report summarised the effect of smoking on shortening of life as follows:

“Death rates in relation to smoking habits

The fatal effects of tobacco smoking are almost restricted to cigarette smokers, and increase with the amounts smoked. Cigarette smokers are about twice as likely to die in middle age as are non-smokers and may have a risk similar to that of non-smokers ten years older. It is estimated that over 20,000 deaths in men between the ages of 35 and 64 are caused every year by smoking in the United Kingdom. The chances are that two out of every five heavy cigarette smokers, but only one out of every five non-smokers, will die before the age of 65. The man of 35 who is an average cigarette smoker is likely on average to lose 5½ years of life compared with a non-smoker. Those who discontinue smoking cigarettes run a steadily diminishing risk of dying from its effects, even after many years of smoking, and attain the level of non-smokers after 10 years of abstinence.”

6. *In the same year, government health warnings were put on cigarette packets in the following terms: “Warning by H.M. Government: Smoking can damage your health.”*

7. *In 1975 two Thames Television documentaries in successive weeks resulted in 160,000 (i.e. 5 per cent of the audience) stopping smoking according to a Gallup poll. In 1976, Thames Television broadcast a film entitled “Death in the West — The Marlborough Story” showing cowboys dying of lung cancer. The film referred to the view of the World Health Organisation that “Cigarettes are the cause of a world-wide epidemic of a disease (lung cancer) which at present kills hundreds of thousands of smokers per year.” In January of that year the Health Education Council launched an anti-smoking campaign aimed at young people. In the spring, the BBC programme “Nationwide” launched a wide-spread campaign to “Stop smoking with Nationwide” which continued for several months. The Advertising Standards Authority implemented a new code of practice in relation to cigarette advertising, which led to the Marlborough cowboy advertising campaign being withdrawn.*

8. *In 1977, the Royal College of Physicians issued a report entitled “Smoking or Health” which concluded that, “Deaths from coronary heart disease are responsible for about half of the excess deaths among cigarette smokers ... The association between smoking and heart disease is largely one of cause and effect.” In March 1977, a Department of Health and NOP Poll showed that 70 per cent of the population favoured further restrictions on smoking in public places. In 1978, cigarette advertising was banned on commercial radio. In 1979 the World Health Organisation published a report entitled “Controlling the Smoking Epidemic” which received widespread press coverage. Later that year, in July, main post offices were made smoke-free.*

9. *The tobacco companies have largely been in denial as to the connection between smoking and bad health. However, in 1980 Dr Green, a scientist formerly employed by British American Tobacco, admitted publicly on the BBC's Panorama programme that he was quite sure that smoking was a major factor in lung cancer.*

10. *In 1981, the Trades Union Congress endorsed an anti-smoking campaign to help union members give up smoking. Mr Badger was a member of the Boilermakers Union, which was affiliated to the TUC.*

11. *In August 1982, the British Medical Association asked the Government to ban all forms of tobacco advertising. In November of the following year, the Royal College of Physicians stated in its report “Health or Smoking”: “Smoking still kills ... some 100,000 of our citizens are dying prematurely from its effects every year.” In February 1984, a national no smoking day was adopted.*

12. *In July 1984 and in February 1985 smoking was prohibited on all London Underground trains and in all London Underground stations respectively. However, this may well have been due to the risk of fire rather than concern as to the effect of smoking on health.*

13. *In March 1986, new health warnings were introduced for cigarette packets. They included “Smoking can cause fatal diseases” and “Stopping smoking reduces the risk of serious diseases” and “Smoking can cause lung cancer, bronchitis and other chest diseases”. At the same time, tobacco advertising in womens magazines was banned. In April 1986, the [Protection of Children \(Tobacco\) Act](#) made it illegal to sell any tobacco product to children under 16 years of age. In December of that year, statistics were published by the World Health Organisation showing that Britain had the world's worst death rates from lung cancer and heart disease. Two months later, in January 1987, Cannon-ABC made all its cinemas smoke-free. British Airways banned smoking on domestic flights in October 1988.*

14. *In 1990, Philip Morris, the cigarette manufacturer, stated in its annual report:*

“We have acknowledged that smoking is a risk factor in the development of lung cancer and certain diseases, because a statistical relationship exists between smoking and the occurrence of these diseases. Accordingly we insist that the decision to smoke, like many other life style decisions, should be made by informed adults. We believe that smokers around the world are well aware of the potential risks associated with tobacco use, and have the knowledge necessary to make an informed decision.”

15. In October 1990, an EC directive banning tobacco advertising on television came into force. In February 1991, London Regional Transport made all its buses smoke-free. In the budget of the following month, the tax on cigarettes was raised, the Chancellor of the Exchequer saying: "There are strong health arguments for a big duty increase on tobacco." Later that year, the Government published its Green Paper "The Health of the Nation" which included a smoking reduction programme. The UK Government changed the text of its health warning on cigarette packets to "Smoking Kills". In November 1991, the Health Education Authority published "The Smoking Epidemic" stating that in the UK 110,000 people die every year from smoking-related lung and other cancers/ heart disease, arterial disease, chronic bronchitis and emphysema.

16. In May 1992, Midland Bank made all its premises smoke-free. In June 1992, Transdermal nicotine patches became available on prescription to assist smokers to stop smoking. In July 1992, the Government White Paper "The Health of the Nation" was published. In the following month, Roy Castle, who later died of lung cancer, ran a high profile campaign against smoking. In the following month, the Home Office for the first time permitted doctors to enter "smoking" as a cause of death on death certificates. In October 1992, 800 doctors took out a four page advertisement in the Independent asking for a total ban on tobacco advertising. In November 1992, National Express banned smoking on all its coaches. Network Southeast banned smoking on its long distance commuter trains in January 1993. In the same month, employers were obliged to provide smoke-free areas of work for employees. Abbey National introduced a total ban on smoking in its premises in February 1993, and in the same month British Midland banned smoking on all its flights. In March 1993 the British Agency for Adoption and Fostering recommended that babies and children under two years should not be placed in households with smokers if equally suitable non-smoking households were available. In April 1993, J. D. Weatherspoons introduced smoke-free zones in 54 pubs. In the following month, all Cathay Pacific flights between Hong Kong and London became non-smoking, as did all National Health Service premises.

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APPENDIX 2: EXTRACT FROM THE JUDGMENT OF MACKAY J IN SHORTELL ON SMOKING KNOWLEDGE

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 inconsiderate 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.