Factual Causation, Mesothelioma and Statistical Validity

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FACTUAL CAUSATION, MESOTHELIOMA AND STATISTICAL VALIDITY

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Introduction
In Sienkiewicz v Greif (UK) Ltd; Knowsley Metropolitan Borough Council v Willmore,¹ the Supreme Court was called upon to clarify further, in the context of the asbestos-related cancer mesothelioma, the doctrine created in Fairchild v Glenhaven Funeral Services Ltd.² Lord Phillips of Worth Matravers stated that doctrine as:

“when a victim contracts mesothelioma each person who has, in breach of duty, been responsible for exposing the victim to a significant quantity of asbestos dust and thus creating a ‘material increase in risk’ of the victim contracting the disease will be held to be jointly and severally liable for causing the disease.”³

Sienkiewicz concerned claims by the administrators of the estates of two women who had died from mesothelioma. Each administrator sued only one defendant: in one case the defendant, Greif, had employed the deceased women, Mrs Costello, for 18 years; in the other case, the defendant council had been responsible for the comprehensive school attended by Mrs Wilmore who died aged 49. It was agreed that, even if the relevant defendants had in breach of duty exposed the victim to legally significant quantities of asbestos,⁴ the victim had been exposed to a much greater volume of asbestos from other sources.

I. The Defendants’ Argument Indirectly Challenged the Fairchild Doctrine
Two features distinguished these claims from those in Fairchild. First, in these claims the claimants could only identify one blameworthy source of risk, that due to the defendant’s breach of duty; the defendants seized upon this to characterise them as “single exposure” cases. Secondly, estimates had been (or could, in theory, have been) made as to the total risk confronting the victim of this single identifiably blameworthy source of risk and of the total risk posed by the other sources of risk alone. Given these features, the defendants argued that the rationale for the Fairchild doctrine is absent in “single exposure” cases because a “conventional” approach to causation is possible by applying the “doubling of the risk” test; that is by comparing the two risk estimates and asking whether, on a balance of

³ Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [1].
⁴ There was a dispute about this in the Wilmore case: Sienkiewicz [2011] 2 W.L.R. 523 at [195].
probability, the defendant’s breach had resulted in the victim facing at least a doubling of the total risk arising from other sources.3

If the defendants’ argument that the doubling-of-the-risk approach applied in all “single exposure” claims were to be accepted, they would prevail in the instant cases because in these the breach had not resulted in a doubling of the total risk arising from other sources.6 For example, the evidence accepted by the trial judge in the Sienkiewicz claim had been that the total risk of contracting mesothelioma to which other sources had subjected the relevant public in general was 24 cases per million and that Greif’s wrongful exposure of Mrs Costello had only increased this risk to 28.39 cases per million.7

Remarkably the defendants had conceded that, in cases of multiple blameworthy exposures, causation could not be established in this “conventional”, doubling-of-the-risk way and so Fairchild applied.8 Though not alluded to by the court, this presented the defendants with a major difficulty because it was highly probable that some of the environmental asbestos pollution in the instant cases had been due to some tortious, albeit unidentified, cause: so, almost certainly, these were multiple blameworthy exposure cases. The defendants sought to avoid this difficulty with an extraordinary argument: that the breathing of polluted “ambient air … should merely be regarded as part of the ordinary vicissitudes of life” at least where such pollution was not “caused by an identifiable individual”.9 In effect this was an assertion that Fairchild only applies where there were multiple identifiable blameworthy exposures. Yet in Fairchild there was no indication that the doctrine was to be limited in such a way and, as Lord Dyson rightly noted,10 it is unclear what normative basis such a limit could have.

Since there is no plausible normative distinction between “single exposure” cases and those in Fairchild, the defendants’ argument about the availability of proof on the basis of the doubling-of-the-risk approach should be equally applicable to the latter. An estimate of the total risk faced by Mr Fairchild due to the breach of a particular defendant could, in theory, have been compared to an estimate of the total risk arising from the other sources of risk alone. If, then, proof on the basis of the doubling-of-the-risk is legitimately available when such estimates are possible, the evidentiary gap in Fairchild would be revealed to be, not an insuperable “rock of uncertainty”,11 but the mere happenstance that these risk estimates had not been gathered in those cases. The rationale for the entire doctrine would vanish.

II. The Statistician’s Analysis: When is the Doubling-of-the-Risk Approach Valid?

In the determination of whether a defendant’s breach contributed to a victim’s disease, the use of risk estimates, based for example on epidemiological data, raises three questions. First, as a matter of statistical analysis, when is it valid to compare

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4 Sienkiewicz [2011] 2 W.L.R. 523 at [4].
5 Sienkiewicz [2011] 2 W.L.R. 523 at [60] and [118].
6 Sienkiewicz [2011] 2 W.L.R. 523 at [208] and [213].
7 Sienkiewicz [2011] 2 W.L.R. 523 at [211], emphasis added.
9 Sienkiewicz [2011] 2 W.L.R. 523 at [212].
such risks (the statistical validity issue)? Secondly, are the data underlying such estimates reliable and a close enough fit to the facts of the case in issue (the probity issue)? Thirdly, even where statistical comparisons are valid and the data is both reliable and relevant, when if ever should the law accept such evidence, based as it is on group data, as the sole grounds for the legal determination of what actually happened in an individual’s case (a judicial policy issue)?

It deserves emphasis that only the first of these matters needed to be addressed by the court in Sienkiewicz because the principles of elementary statistics expose the defendants’ argument to be statistically invalid. The comparison of risk estimates is valid where we know that these estimates represent mutually exclusive ways the injury may have been caused and we seek to estimate the likelihood it was one way which had operated in a particular case rather than one of the other possible ways.

It follows that such comparisons are statistically valid where the estimates relate to mechanisms which, even if their details are not understood, are known to involve different agents, such as where: a collision might have been with a blue taxi or with a red taxi; a birth defect might have been due to a pharmaceutical or to the background risk; a lung cancer in an asbestos-exposed smoker might have resulted from a mechanism involving tobacco (but not asbestos) or a mechanism involving asbestos (but not tobacco), or a synergistic mechanism involving both tobacco and asbestos, or a mechanism involving neither tobacco nor asbestos but some other background carcinogen such as radon. In such cases, the comparison of separate risk estimates for each mechanism provides a measure of the likelihood that one mechanism was operative rather than another/others: for example, that it was a blue taxi not a red one involved in a collision (though, of course, it provides no indication of the likelihood it was one particular blue taxi that was involved).

Even where a disease is taken to result only from a single agent—“single-agent diseases”—such comparisons could be statistically valid in at least two contexts. One is where the figures carry information about variable susceptibility to the agent. The hypothetical example given by Lord Mackay of Clashfern in Hotson v East Berkshire Area Health Authority concerns contraction of dermatitis from brick dust:

“say that it was established that of 100 people working under the same conditions as the pursuer and without facilities for washing at the end of their shift 70 contracted dermatitis: of 100 people working in the same conditions as the pursuer when washing facilities were provided for them at the end of the shift 30 contracted dermatitis.”

Within the 70 victims in the former scenario, there are two mutually exclusive classes: a cohort of 30 whose susceptibility would have been overcome even with washing facilities; and a cohort of 40 whose higher biological resistance had only

12 Daubert v Merrell Dow Pharmaceuticals Inc., 43 F.3d 1311 (9th Cir.1991) at 1320.
14 Often risk estimates have not been made of the rival mechanisms, see e.g. Wilsher v Essex AHA [1987] UKHL 11; [1988] A.C. 1074 at 1801: excess oxygen, apnoea, hypercarbia, intraventricular haemorrhage and patent ductus arteriosus.
been overcome by the extended exposure to the dust. Where a victim, V, did not have washing facilities and contracted dermatitis, it is statistically valid to conclude that V is probably a member of the latter cohort and that, therefore, the lack of washing facilities contributed to his dermatitis.17 In other words, the data supports the conclusion as to which of two mutually exclusive scenarios had occurred: that V’s dermatitis probably followed the etiology involved when a robust individual succumbs after extended exposure, rather than the etiology involved when a susceptible individual succumbs after shorter exposure.

Similarly, comparison of risk estimates in relation to a single-agent disease would be statistically valid if it were accepted that the mechanism involved just one unit of the agent, for example where it was accepted that a disease resulted from one mosquito bite or a single injection.18 Thus, if the medical consensus were that mesothelioma was a single-agent disease and caused by a single fibre of asbestos, the law could legitimately assume the victim’s disease had either been caused by a fibre from the defendant’s breach or a fibre from one of the other sources. In such circumstances it would be statistically valid to compare risk estimates of the two scenarios (the total risk faced by the victim and the total risk from sources other than the defendant’s breach) to judge the likelihood that it was a fibre from the defendant’s breach that had operated in the particular case. For example, a comparison of the 28.39 cases per million risk faced by Mrs Costello with the 24 cases per million risk faced by the general public could validly be used to deduce, on the balance of probabilities, the degree of likelihood that it was a fibre from the defendant’s breach that had operated, and since 28.39 is not more than double 24, the conclusion would be that this was unlikely.

But there is no consensus about how mesothelioma is caused. Though there is the possibility that it may be caused by an agent other than asbestos, courts treat it, in effect, as a single-agent disease.19 It is not known how asbestos causes mesothelioma. Let alone that this process involves a single fibre:20 it is feasible that the mechanism by which asbestos caused mesothelioma in Mrs Costello involved fibres from both the defendant’s breach and from other sources.21 This means that it is not known that the two risk estimates accepted as evidence in relation to Mrs Costello represent mutually exclusive ways her injury might have occurred. So the comparison of risk estimates, which is intrinsic to the defendant’s doubling-of-the-risk approach, is statistically invalid here.22

17 As recognised by Lord Phillips, Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [29]. Where the figures are 48 and 60 respectively it is likely that the victim was in the former cohort so that lack of washing facilities did not contribute to the dermatitis and the employer would escape liability. Lord Salmon erred on the point in McGhee v National Coal Board [1972] UKHL 11; [1973] 1 W.L.R. 1 at 12–13 (see Jane Stapleton, “The Gist of Negligence: Part 2 The Relationship between ‘Damage’ and Causation” (1988) 104 L.Q.R. 389 at 399, fn.23), an error Lord Phillips notes at [26].
19 This is acceptable if one assumes that “mesothelioma is … almost always, caused by the inhalation of asbestos fibres”, Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [19], because it would then follow that for any individual case, more likely than not the mesothelioma had been asbestos-related. It is in the subsequent issue, namely which sources of asbestos were likely involved in the individual case, that the “rock of uncertainty” is located: Stapleton, “Factual Causation and Asbestos Cancers” (2010) 126 L.Q.R. 351 at 356.
20 Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [102].
22 Equally, where the one particular exposure to radiation materially increased the risk of a disease but there was background radiation (which also materially increased the risk of that disease), it is statistically meaningless (at least so long as ignorance of the disease mechanism means it is possible that the mechanism by which an individual’s disease came about might have involved contributions from both sources of radiation) to require the claimant to show that
Because the comparison of risk estimates is, in the case of a single-agent disease of unknown mechanism, simply invalid as a matter of statistics, a victim of such a disease is unable to establish that a small source of risk (i.e. small relative to the total risk) was involved in the victim’s contraction of the disease and a defendant responsible for one of those sources cannot show that it was uninvolved. (Proof of involvement of a dominant source of risk is possible: see below.) It is on this basis that the restriction of the Fairchild doctrine to diseases of unknown mechanism that are only caused by “the same agent, or an agent acting in the same causative way”23 might be rationalised: in such contexts an attempt to use risk estimates to show that the disease was likely to have been caused by a relatively small source of risk rather than another source would be statistically invalid, so the barrier to proof confronting the claimant in such a case is not simply high but insuperable as a matter of formal statistics. The Fairchild doctrine is predicated not merely on the difficulty but on the impossibility of proof.24

Comparing estimates of risk on the invalid assumption that the risks are independent recently led to a grave miscarriage of criminal justice in the Sally Clark case.25 This incident, in which a medical expert made a statistical error that a statistical expert would have identified as elementary, prompted the Royal Statistical Society publicly to urge the Lord Chancellor “to take steps to ensure that statistical evidence is presented only by appropriately qualified statistical experts”.26 So it was to be hoped that this fundamental flaw in the defendants’ argument in Sienkiewicz would have been identified clearly and at an early stage of the litigation. This did not happen. Indeed, the Court of Appeal in Sienkiewicz v Greif (UK) Ltd27 was much attracted to the defendants’ argument, though in the end it held that it was pre-empted by the terms of s.3 of the Compensation Act 2006 which it took to mandate the Fairchild approach in mesothelioma cases. This interpretation of the statute was roundly rejected by the Supreme Court.28

III. The Court’s Analysis: Multiple Potential Cause Cases Generally

Despite labouring without the assistance of “appropriately qualified statistical experts”, all members of the Supreme Court were able to reject the defendants’ argument that, because of the alleged availability of proof by statistics, Fairchild did not apply to “single exposure” cases. Understandably, however, the route to this conclusion was circuitous.

Only Lord Phillips squarely addressed the issue of statistical validity in detail. In the longest judgment his Lordship felt obliged first to deal with a general proposition proffered by Smith L.J. in Sienkiewicz about cases of “multiple potential causes” apart from cases involving mesothelioma:

the risk from the former was “as least twice” that from the latter: contrast B v Ministry of Defence [2010] EWCA Civ 1317; (2011) 117 B.M.L.R. 101 at [151].
23 Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [104].
26 Available at www.rss.org.uk/statsandlaw.
28 Sienkiewicz [2011] 2 W.L.R. 523 at [70], [131] and [183].
“in a case of multiple potential causes, a claimant can demonstrate causation by showing that the tortious exposure has at least doubled the risk arising from the non-tortious cause or causes.”

To the extent this statement is limited to the positive establishment of a causal connection between a dominant source of risk and the outcome, Lord Phillips agreed. 30

Certainly where the exposures were simultaneous 31 it is true that we can conclude that a dominant source that accounts for well over half the total risk made a causal contribution but it is important to understand on what basis we do this. Suppose that bladder cancer can only be caused by a particular agent (amines) and a victim had been, for exactly the same period of time, simultaneously exposed to only two sources of that agent: occupational amine exposure and amines contained within cigarette smoke. Suppose further that the volume of the agent from occupational exposure was considerably more than double that from the smoking exposure. In such circumstances, it would be possible to infer that it was probable that the dominant source of the agent, occupational exposure, contributed to the disease: this is because whatever the mechanism of the cancer, it is probable that the occupational exposure was involved—for example by being the probable source of the single unit (if that were the mechanism) or by probably contributing to the threshold (if that were the mechanism) and so on. 32 (Similar reasoning might be used to help establish that one source of “enormous quantities” of asbestos relative to other simultaneous sources probably contributed to a claimant’s mesothelioma. 33) But it is not a corollary of this proposition that the minor source of risk probably did not contribute 34; for example, if the mechanism involved a threshold, that source would have contributed to it.

To be a general method applicable to a type of injury, the doubling-of-the-risk approach must be capable of determining whether a source of risk is implicated or exculpated. It follows that the predicate for the validity of the approach is that we know that there had been mutually exclusive ways the relevant injury might have come about, one(s) that involved the source in issue and one that did not. In other words, if the doubling-of-the-risk approach is to be a general approach for a type of injury it must be valid as a way to establish that a relatively small source of risk was not causally involved in the outcome in issue: this was, after all, the use to which the defendants in Sienkiewicz wanted to put it. Because we do not know the mechanism by which the amines cause bladder cancer the doubling-of-the-risk approach is not validly applicable to this disease as a general method.

30 Sienkiewicz [2011] 2 W.L.R. 523 at [78]: “if a defendant is responsible for a tortious exposure that has more than doubled the risk of the victim’s disease, it follows on the balance of probability that he has caused the disease”.
31 Where the exposures were sequential such a conclusion about the dominant source is not possible where the mechanism is unknown. This is because the mechanism might, for example, require only one unit of agent and be highly time dependent so that it probably involved that unit at a time other than during the dominant exposure.
32 Compare the reasoning in Novartis Grimsby Ltd v Cookson [2007] EWCA Civ 1261.
33 Margaretson v J W Roberts Ltd [1996] P.I.Q.R. P358, P360. Of course, the mesothelioma claimant would also not need the Fairchild doctrine where it was agreed that the only significant source of asbestos exposure had been due to the defendant’s breach of duty: Stapleton, “Factual Causation and Asbestos Cancers” (2010) 126 L.Q.R. 351 at 354.
34 Contrast the obiter dictum in Novartis [2007] EWCA Civ 1261 at [74] per Smith L.J.
Lord Phillips appreciated that the validity of the doubling-of-the-risk approach is critically related to what was known about the mechanism of the disease. For example even in single-agent diseases there may be data on relative susceptibility to the degree of exposure to the agent which would allow valid conclusions to be drawn as to which of two mutually exclusive scenarios had occurred as in Lord Mackay’s hypothetical about dermatitis.\textsuperscript{35}

Similarly, his Lordship noted that some diseases are known to be due to the effect of a single unit of agent resulting in a condition that is “indivisible” in the sense that the severity of the disease is not related to the amount of agent to which the body had been exposed: an example is malaria caused by a single mosquito bite.\textsuperscript{36} Lord Phillips recognised, correctly, the validity of the doubling-of-the-risk approach in such a context and wherever there are “competing alternative” mechanisms.\textsuperscript{37} This is because, as with the earlier illustration of taxis, where risk estimates are known to represent mutually exclusive ways the injury may have been caused, their comparison provides a statistically valid measure of the likelihood that one mechanism was operative rather than another/others.

In the light of this it is, with respect, striking that Lord Phillips questioned the validity of the doubling-of-the-risk approach in the particular context of lung cancer in asbestos exposed smokers.\textsuperscript{38} In fact, given suitably reliable and relevant risk estimates, such an approach is statistically valid here\textsuperscript{39} because there are four mutually exclusive mechanisms (tobacco only; asbestos only; tobacco and asbestos; a mechanism involving neither tobacco nor asbestos but some other carcinogen such as radon) only one of which could have operated in the individual’s case. His Lordship noted that epidemiological data established that asbestos and cigarette smoke can act synergistically to cause lung cancer.\textsuperscript{40} He seems to have extrapolated from this the unwarranted conclusion that, whenever an asbestos exposed smoker contracts lung cancer the operative mechanism must be one in which “asbestos and tobacco smoke [had] combined to cause lung cancer”,\textsuperscript{41} from which it follows that the asbestos will have made a material contribution to the cancer so that any doubling-of-the-risk requirement is invalid. This dictum will shock legal practitioners in this field. Had the assistance of professional statistical expertise been available to his Lordship, he may not have made this unjustified extrapolation.

In contrast, Lord Phillips correctly rejected the validity of the doubling-of-the-risk approach in diseases where the mechanism by which the disease is contracted is known to require multiple units of an agent and where there had been multiple sources of agent operating simultaneously. In such cases a tortfeasor legally responsible for only a relatively small proportion of the total

\textsuperscript{35} The doubling-of-the-risk terminology can be misleading here. For example, Lord Phillips’s statement that “the lack of washing facilities more than doubled the risk of contracting dermatitis”, Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [29], cannot refer to the doubling of an individual’s risk since the person with the higher biological resistance would likely have faced no risk had there been washing facilities, see Lord Rodger at [153].

\textsuperscript{36} Sienkiewicz [2011] 2 W.L.R. 523 at [12]. His Lordship contrasts these “diseases … caused by a single agent” [12] with “diseases where the contraction is dose related” [13]. This unorthodox use of the terms “single agent” and “dose related” might obscure the contrast intended, namely between a disease which is contracted by a single unit of agent and a disease which requires multiple units of agents before contraction occurs.

\textsuperscript{37} Sienkiewicz [2011] 2 W.L.R. 523 at [93].

\textsuperscript{38} Sienkiewicz [2011] 2 W.L.R. 523 at [76].


\textsuperscript{40} Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [75].

\textsuperscript{41} Sienkiewicz [2011] 2 W.L.R. 523 at [76].
amount of agent that had assaulted the body cannot use the doubling-of-the-risk approach to exculpate himself. This is because the predicate for the validity of the approach is absent: it is not the case that there had been independent alternative ways the relevant injury might have come about, one that involved the small source and one that did not. Where the disease is divisible—such as is the case with asbestosis—the severity of the disease is related to the amount of agent that assaulted the body, so we know that the small source had been involved in producing some injury albeit only a portion of the total condition suffered by the victim. So the tortfeasor would “be liable in respect of the share of the disease for which he is responsible”\textsuperscript{42}. Similarly in a case where the disease mechanism is known\textsuperscript{43} to require multiple units of an agent forming a threshold which, once passed, triggers a disease that is indivisible—as seems to be the case with many cancers—the tortfeasor would have contributed some amount to the threshold and therefore to the contraction of the disease: he was, therefore, a cause and legally responsible for the entire indivisible disease.\textsuperscript{44}

The simple fact that the doubling-of-the-risk approach is invalid in the above context would have been enough to demolish the notion that the approach is valid as a general method either to implicate or exculpate a minor source of risk in a case of multiple sources of risk. However, Lord Phillips went on to state, in a puzzling dictum about consecutive exposures, that he saw no reason in principle why the doubling-of-the-risk approach should not be applied

“where the initiation of the disease is dose related [i.e. requires multiple units], and there have been consecutive exposures to an agent or agents that cause the disease, one innocent and one tortious … [because] where the innocent exposure came first, there may be an issue as to whether this was sufficient to trigger the disease or whether the subsequent, tortious, exposure contributed to the cause.”\textsuperscript{45}

With respect, much more would need to be known about the mechanism for the doubling-of-the-risk approach to be validly used to exculpate the later exposure. For example, it would not be valid if the threshold “sufficient to trigger the disease” only becomes potent at a time that postdates both exposures: such dormancy in the mechanism would permit the involvement of all sources of agent including a small tortious contribution that followed a large innocent one. Again, the predicate for the validity of the doubling-of-the-risk approach would be absent.

To illustrate, suppose a person is injured in a road accident and a passing doctor without breach of duty administers an emergency dose of a certain medication. The victim is immediately transferred to a hospital where the admitting doctor, failing to discover what medications had already been administered to the victim, negligently administers another, albeit smaller, dose of the medication. Later the patient suffers an indivisible adverse reaction to the medication, such as death. Of course, it is theoretically possible that the mechanism that produced death required only a single molecule of the medicine, in which case it is likely to have come

\textsuperscript{42} Sienkiewicz [2011] 2 W.L.R. 523 at [90]. These diseases are discussed by his Lordship at [14].
\textsuperscript{43} A fortiori, the doubling-of-the-risk approach is invalid where the mechanism is unknown but might involve such a threshold.
\textsuperscript{44} Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [90]. These diseases are discussed by his Lordship at [13].
\textsuperscript{45} Sienkiewicz [2011] 2 W.L.R. 523 at [91].
from the larger lawful dose and we can validly conclude that the later smaller dose would have played no causal role. But such a mechanism is far-fetched. Much more likely is that the mechanism required a certain concentration of molecules, in which case at least some from the later smaller dose, being comingled with the innocent molecules, would have been involved in the process leading to death. In other words, the tortious administration of the medication by the admitting doctor would have made a causal contribution to the death, even though another larger and earlier dose would alone have been sufficient for death to follow.

Lord Phillips’ dictum seems based on the very common, but misleading, conflation of two distinct issues. One is whether the tortious contribution of the agent made a causal contribution to the occurrence of the injury. The other issue is whether that injury represents “damage” relative to the position that the law determines the victim had been entitled to be in. The road accident victim’s death would not represent such “damage” if the innocent contributions of medication would alone have been “sufficient to trigger” the death anyway.

IV. Statistical Data: Probity and Judicial Policy

Since it does not seem to have been made clear to the Court that it could simply have dismissed both the defendants’ argument and the general applicability of the doubling-of-the-risk approach on the grounds of statistical invalidity, it is understandable that Lord Phillips and the other justices also commented on the general issue concerning the probity of the underlying data. For example, even though the approach is statistically valid in the taxi example because the relevant mechanisms are known to be mutually exclusive, it may not be judged sufficiently probative in the individual case given the importance of other available evidence such as the past accident records of the taxi firms in question. In particular, epidemiological data may be unreliable because it is too thin or because its parameters are not a close fit to that of the individual case in hand.

Especially attention was also paid to the third issue: the general judicial policy question of whether the law should permit reliance solely on risk estimates in a suitable case. Lord Phillips saw no reason in principle why this should not be permitted in certain cases. In contrast, Lord Rodger of Earlsferry noted that risk estimates alone (even if entirely reliable and a tight fit to the facts in issue) could at most go to the question of whether the court was satisfied, on the balance of probability, that the defendant’s breach had probably caused the result, whereas “in civil proceedings for damages the role of the judge is to decide, on the balance of probability, what actually happened” in the individual case. In his Lordship’s

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46. It was the latter issue that was at stake in X v Schering Health Care Ltd [2002] EWHC 1420 (QB); (2003) 70 B.M.R. 88.
47. Sienkiewicz v Greiff (UK) Ltd [2011] 2 W.L.R. 523 at [83], [96] and [205].
51. Sienkiewicz [2011] 2 W.L.R. 523 at [91] and [93].
view, the introduction of a rule allowing reliance solely on evidence based on group statistics to judge the question of actual causation in an individual’s case would so radically change the nature of civil proof that it should be the province of the legislature.53 Baroness Hale agreed with Lord Rodger.54 Lord Mance and Lord Kerr were cautious but ultimately agnostic55 while Lord Dyson shrewdly contemplated the possibility that in a particular case such evidence might on the facts be so reliable and such a compelling fit with the facts of the case that a court could form the necessary degree of belief in relation to the issue of what had actually happened in an individual’s case.56

V. The Court’s Analysis: “Single Exposure” Mesothelioma Cases

In relation to the defendants’ argument that the doubling-of-the-risk approach was available in “single exposure” mesothelioma cases, Lord Phillips pointed to a series of passages in Barker v Corus UK Ltd57: these seemed to imply that in relation to mesothelioma one could deduce from the relative volume of fibre or the relative degree of risk contributed from one source a measure of the likelihood that this source had or had not been a cause of the victim’s disease.58 For example, Lord Hoffmann had implied that where parties had “a very small share in exposing the claimant to risk” there was “a relatively small chance” that they had contributed to the mesothelioma.59 If the implication in these passages was well founded, then Lord Phillips saw an apparent conundrum:

“Lord Bingham observed [in Fairchild] … that it is accepted that the risk of developing mesothelioma increases in proportion to the quantity of asbestos dust and fibres inhaled. If this is so why should one not determine the probability that a particular defendant caused a claimant’s mesothelioma by analysing the extent to which he wrongfully contributed to the exposure of the claimant to asbestos dust and fibres? … Why was there any need to apply the Fairchild/Barker rule where epidemiological evidence enabled one to use statistics to determine causation on balance of probability?”60

Had the justices had assistance from statistical experts, they would have been able to reject the defendants’ argument on the simple basis that it was statistically invalid. Though risk of mesothelioma does indeed increase with the quantity of asbestos inhaled, this simply does not provide the necessary information about the mechanism by which mesothelioma is caused which would render statistically valid the comparison of risk estimates at the heart of the doubling-of-the-risk approach, namely information that the sources of risk represent mutually exclusive ways the cancer might have come about. The mechanism of mesothelioma is unknown so it cannot be said that the sources of risk are “rivals” or in “competition” with each other.61 This means that, though risk increases with the quantity of

54 Sienkiewicz [2011] 1 W.L.R. 523 at [170].
55 Sienkiewicz [2011] 2 W.L.R. 523 at [192] and [204].
56 Sienkiewicz [2011] 2 W.L.R. 523 at [222].
58 Sienkiewicz v Greif (UK) Ltd [2011] 1 W.L.R. 523 at [48]–[54]. See also [52].
60 Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [45] and [94].
61 Sienkiewicz [2011] 1 W.L.R. 523 at [104] and [61].
asbestos inhaled, this is no evidence that a defendant’s relative contribution to the
volume of fibre inhaled or to the relative degree of risk correlates to the relative
likelihood that the defendant was or was not involved in the causation of the
victim’s mesothelioma. The implication in Lord Hoffmann’s comment has no
foundation in the available science. The conundrum is revealed to be a false one.
Though the Supreme Court did unanimously reject the defence argument, no
judgment was based on the straightforward basis that it was statistically invalid.
Lord Phillips’ reasoning came closest but it was admixed with comments on
probity and judicial policy. His conclusion was that

“there are special features about mesothelioma, and the gaps in our knowledge
in relation to it, that render it inappropriate to decide causation on epidemiological data as to exposure.”

With respect, a preferable, because simpler and more powerful, explanation would
have been simply to expose the argument as statistically invalid.

In the other substantial judgment, Lord Rodger argued that there was no room
for introducing the doubling-of-the-risk approach in mesothelioma cases now that
Fairchild had intervened to assist claimants who would, due to gaps in knowledge,
have failed under such an approach. Baroness Hale made a similar argument.
Again, with respect, these passages obscure the fundamental reason the approach
has no place in mesothelioma claims against defendants legally responsible for
minor sources of exposure: this reason, which has nothing to do with whether or
not a jurisdiction has adopted a rule such as Fairchild, is simply that the approach
is statistically invalid in such cases.

VI. In Summary

The Supreme Court considered and rejected propositions that the
doubling-of-the-risk approach was a legitimate general method of proving and
disproving factual causation: in mesothelioma cases where there was only one
identifiable tortious exposure; and in other multiple potential cause cases. Both
propositions could have been rejected as invalid on elementary statistical principles.
As the Sally Clark case showed, the proper handling of such principles is not a
matter of common sense: even medical experts make elementary mistakes. It is,
therefore, much to be regretted that in Sienkiewicz the Supreme Court was left to
be confronted with a toxic tort case of such importance without the assistance of,
or at least access to, professional expertise on statistical validity which would have
greatly simplified the issues. It is also to be regretted that in such circumstances,
and where it was not necessary for the decision, the court speculated on the
appropriate role of epidemiological evidence in general.

It is another matter entirely whether the law chooses to use the separate risk estimates for the sources as a device
to divide responsibility between defendants: Barker v Corus UK Ltd [2006] UKHL 20; [2006] 2 A.C. 572 at [109]
per Lord Walker.

Sienkiewicz v Greif (UK) Ltd [2011] 2 W.L.R. 523 at [104].
*Sienkiewicz [2011] 2 W.L.R. 523 at [94]–[106].
Sienkiewicz [2011] 2 W.L.R. 523 at [106], emphasis added.
Sienkiewicz [2011] 2 W.L.R. 523 at [159]–[160].
Sienkiewicz [2011] 2 W.L.R. 523 at [169].
Asbestos; Causation; Mesothelioma; Proof; Risk; Statistics