Neutral Citation Number: [2023] EWCA Civ 1377

Case No: CA-2022-002508

IN THE COURT OF APPEAL (CIVIL DIVISION)
ON TRANSFER FROM THE HIGH COURT OF JUSTICE
HIGH COURT APPEAL CENTRE CARDIFF
ON APPEAL FROM THE COUNTY COURT AT CARDIFF
HIS HONOUR JUDGE HARRISON
G14YJ691

Cardiff Civil Justice Centre
2 Park Street, Cardiff CF10 1ET

Date: 22 November 2023

Before:

LORD JUSTICE UNDERHILL
(Vice-President of the Court of Appeal Civil Division)
LORD JUSTICE PHILLIPS
and
LORD JUSTICE STUART-SMITH

- -

Between:

MICHAEL HOLMES

Claimant/Respondent

-and-

POETON HOLDINGS LIMITED

Defendant/Appellant

- -
- -

Michael Kent KC and **Jamie Clarke** (instructed by **DAC Beachcroft**) for the **Appellant**
Patrick Limb KC and **Thomas Herbert** (instructed by **Howells Solicitors**) for the **Respondent**

Hearing dates: 25-26 July 2023

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Approved Judgment

This judgment was handed down remotely at 10.30am on 22 November 2023 by circulation to the parties or their representatives by e-mail and by release to the National Archives.

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Lord Justice Stuart-Smith

Introduction

1. Between 1982 and 2020, when he was forced by ill-health to retire prematurely, Mr Holmes was a valued employee of the appellant, Poeton Holdings Ltd ["Poeton"]. In 2014 he was diagnosed as suffering from Parkinson's disease. By this action Mr Holmes claims damages from Poeton because it acted in breach of its common law and statutory duty in the period from 1982 to 1997 by exposing him to unsafe levels of Trichloroethylene ["TCE"] in the course of his employment. After a trial of the issues of breach of duty and causation, HHJ Harrison found in favour of Mr Holmes on both issues, leading him to conclude that Poeton was liable to Mr Holmes for all the consequences of his having contracted Parkinson's disease. I shall describe his claim for all the consequences as a claim for 100% damages.

2. There is no appeal against the Judge's findings of breach of duty. Poeton has, however, mounted a wide-ranging appeal against the Judge's legal analysis, factual findings and overall conclusions on the issue of causation. In briefest outline, Poeton submits that the Judge adopted the wrong legal test for establishing causation of what is acknowledged on all sides to be an "indivisible disease". Specifically, it submits that the Judge erred in failing to address the question whether Mr Holmes' development of Parkinson's disease would have happened in any event, so that the exposure to TCE made no difference. It goes on to submit that the evidence available to the Judge showed no more than that TCE may have caused an elevation of the risk of contracting Parkinson's disease: it did not demonstrate that exposure to TCE was capable of causing Parkinson's disease or that Mr Holmes' exposure had caused his contraction of the disease.

3. The breadth of Poeton's attack on the Judge's findings requires this Court to review the development of the law relating to causation of indivisible diseases in and since *Bonnington Castings v Wardlaw* [1956] 1 AC 613. I shall attempt that review later in this judgment. It may, however, be helpful to make clear at the outset that Mr Holmes has not advanced his case on the basis that he falls within what has been called "the *Fairchild* exception". As before the Judge below, he does not argue before this Court that he is entitled to damages because his exposure to TCE increased *the risk* of him developing Parkinson's disease. That might, if proved, entitle him to a measure of damages proportional to the increase in risk attributable to Poeton's tortious exposure. Perhaps optimistically, he seeks to reserve the right to advance a *Fairchild* claim at some future date if his claim to 100% damages fails. Whether or not he should or would be allowed to do so is not for this judgment to decide.

4. For the reasons I set out below I would allow the appeal.

The Factual Background

5. What follows is taken from the judgment and is largely uncontroversial. Mr Holmes was born in 1966 and spent his entire working life with Poeton. He joined as an apprentice in 1982 and was taken on as a process operator the following year. In 1990 he was promoted to foreman/supervisor but remained working on the shop floor. In 1997 he was further promoted to a managerial role and his work on the shop floor

became more limited. Having been diagnosed in 2014 he continued to work for Poeton until 2020. These proceedings were issued in February 2020.

6. TCE is an organic solvent whose properties make it particularly useful in the cleaning or degreasing of engineering components. It could be used "cold" on rags, but was more extensively used "hot" in a degreasing tank. When it was being used "hot" it would be poured into the tank and heated to produce vapour. Components would either sit or be suspended in the vapour produced in the tank. A degreasing tank would usually have a form of cooling coil near to the top of the tank above the area in which the components would sit. This would serve to condense the vapour and limit its escape. In addition, such tanks often had ventilation in the form of lip extraction near the top, which limited vapour escape further. Poeton's tank was essentially normal in these respects during Mr Holmes' relevant employment, with an additional centrifugal fan extraction unit being added in March 1991.

7. TCE is a carcinogen that is readily absorbed by the body following inhalation. Poeton was a significant user of TCE, using some 7 to 8 tonnes per annum. It was known to be potentially harmful even before it was known to be a carcinogen. By 1973 the Factories Inspectorate identified the need for breathing apparatus to be used if working with TCE in confined spaces. In and from 1976 the HSE recommended an 8 hour time weighted average day exposure limit of 100 ppm and in and from 1984 a recommended short-term limit of 150 ppm was added. These are still the recommended safe limits. In 1985 a level of 5000 ppm was identified as being immediately dangerous to life. The Judge found it to be established that TCE is neurotoxic and can act upon the dopaminergic neurons. It is also established that damage to those neurons is the mechanism by which Parkinson's disease arises, though the precise aetiology and pathogenesis of the condition is only partially understood and remains the subject of ongoing research.

8. The Judge largely accepted Mr Holmes' case on the extent of his exposure to TCE while at work. He divided his employment into three periods: first, from 1983 to about 1990 when he was a general worker; second, from 1990 to 1997 when he was general foreman; and, third, from 1997 when he was a manager. His case was that his exposure was heaviest during the first two periods. Mr Holmes did not advance a claim in respect of his employment after 1997 and the Judge made no findings about whether he was exposed at all to TCE during that third period. If he was, it was evidently taken to be at levels that were not in breach of duty and, by implication, within the then recognised safe levels.

9. The Judge held that, at all material times, the degreasing tank would be filled by operators using buckets. When the unit was in extensive operation it would need to be topped up frequently. If it was very busy, he considered it to be highly unlikely that the operatives would wait until the tank was stone cold. Equally, he considered it unlikely that new liquid would be added if the existing TCE in the tank was at its boiling temperature.

10. The method of operating the tank was that, having been filled, it would be heated and components placed in baskets below the cooling coil and in the vapour zone. When they had been there for long enough they would be removed manually using a hook or some similar implement. Ideally the components would be held in the "freeboard zone" above the cooling coil level and below the lip extraction level to allow any condensed

liquid on the components to drip off or evaporate away. This would be consistent with HSE Guidance issued in 1976 and 1985. Holding the components in this area would reduce operatives' exposure to TCE vapour. One way of holding components would be to use a mechanical hoist but, on the Judge's findings, although there may have been a hoist on the premises, it was not provided for use in the tank. There was evidence, which the Judge accepted, that operatives would therefore regularly "hoik" components (other than the most modestly sized items) out relatively quickly and rest them on the side of the tank or leave them beside the tank rather than keeping them longer in the freeboard zone, which would lead to "high levels of exposure". This practice would circumvent the effectiveness of the lip extraction. The Judge was satisfied that the general working practices described by the Claimant and those witnesses called on his behalf were allowed to persist and the TCE was not treated with sufficient caution.

11. Cold cleaning with TCE on a rag involved degreasing components outside the tank. The Claimant's case, which the Judge accepted, was that larger items such as missile tubes were a regular part of an aerospace contract enjoyed by Poeton and that they could not be dealt with in the degreasing tank. Cold cleaning, using TCE from a bucket, was the only effective means of preparing such components and the Judge found that cold TCE was frequently used as a method of degreasing during the time that Mr Holmes was a general operative and foreman.

12. The third main source of exposure of which Mr Holmes complained was while cleaning the inside of the TCE tank. Mr Holmes' case was that this was undertaken every three or four weeks by an operative getting inside it. The Defendant's evidence was that this operation would take place about monthly and would take about 2 hours on each occasion. The evidence established that, although the work was undertaken in a confined space, no breathing apparatus was provided and that the operation potentially exposed operatives to very high levels of TCE. It was accepted by Mr Holmes that he was not the only operative who undertook this task. Once again, the Judge accepted the main thrust of the Claimant's case and evidence. He found that cleaning of the tank by way of an operative getting inside it occurred approximately monthly and that the task "was not fleeting". The operative would have to have crouched or bent periodically. The task would take about 2 hours overall, though the operative would not be inside the tank for the whole of that period. The Judge found that Mr Holmes would have undertaken "a substantial proportion of the tasks."

13. Turning to the likely levels of exposure that these operations would involve, the Judge did not make any quantitative assessment of the levels involved in his assessment of the evidence about using the tank or cold cleaning. He accepted that "hoiking" out components prematurely effectively circumvented the benefits of prolonged cooling and lip extraction, whether components were rested on the rim of the tank or put on the floor next to it. He recorded the agreed expert evidence that "it was likely that the short-term limit value of 150 ppm over 10 minutes would have been exceeded and that the long-term limit would also have been exceeded on some days (100 ppm as an 8 hour time weighted average.)". He regarded this evidence as important (though not directly transferrable) when "looking at what exposure might have been like" if lip extraction was circumvented by "hoiking" out or similar. In the light of that and other evidence he concluded that "when the lip extraction was circumvented then [Mr Holmes] was likely exposed to levels in excess of the short-term limit value when the extraction was bypassed. I have already concluded that such occurred regularly."

14. Turning to cold cleaning, the Judge noted the experts' agreement that these activities were "capable of producing very high exposures indeed. The wide range of 500 to 2000 ppm appears [in their report]. Even taking the lowest level of the estimate the short-term and long-term value limits are likely to be easily exceeded."

15. It was agreed by the experts that cleaning the tank "had the potential to create very high exposure levels, even to the extent they would have been at a level that was immediately dangerous to life if exposed workers were unprotected." Levels of exposure would have been significantly in excess of the short-term limit value.

16. Drawing these strands together, the Judge found that "throughout the first and second relevant periods of the Claimant's employment with the Defendants, the Claimant was regularly exposed to levels of exposure significantly in excess of the short-term limit value and, for a significant number of days a year in excess of the long-term value."

17. In the light of these findings the Judge held that Poeton was in breach of (a) Regulation 7 of the COSHH regulations; (b) s 29 of the Factories Act 1961; and (c) their common law duty to provide a safe place and system of work. He then turned to the issue of causation.

Causation in the judgment

18. The Judge focused on the fact that the parties characterised Mr Holmes' Parkinson's disease as an indivisible injury. He accepted the Claimant's formulation that "whereas a divisible injury will necessarily be the result of cumulative causes, by contrast an indivisible injury can result from a single non-cumulative cause or cumulative causes."

19. He summarised briefly the principles he intended to apply, as follows:

> "61. As a legal concept "material contribution" is a recognised extension of the "but for" test of causation. From *Wardlaw v Bonnington Castings* [1956] 1 AC 613 through *Fairchild v Glenhaven Funeral Services Ltd* [2002] UKHL 22 to *Simmons v British Steel* [2004] UKHL 20, the House of Lords confirmed that:
>
> > "if a number of factors contribute to the injury it is sufficient [for causation to be established] that the contribution which the factor attributable to the defender's fault was material."
>
> 62. For the principle to be relied upon the Claimant must prove on the balance of probabilities that the contribution was more than "*de minimis*". It is insufficient for a Claimant to identify one of a number of potential causative pathways (*Wilsher v Essex HA* [1988] AC 1074), but the principle of material contribution is equally applicable in multiple factor cases as it is in single agency cases (per Picken J in *John v Central Manchester and Manchester Children's Hospital Foundation Trust* [2016] 4 WLR 54)."

20. The Judge then summarised the respective submissions of the parties:

i) Poeton contended that the evidence available to Mr Holmes fell short of showing a causative link between exposure to TCE and the development of his Parkinson's disease "or similar symptomatology." Its case was that the evidence could only establish a material contribution to a risk of developing the disease, which would be insufficient. Poeton went further and submitted that the doctrine of material contribution that he had outlined was not available in cases of indivisible conditions. Consequently, in order to succeed, Poeton submitted that Mr Holmes had to satisfy the basic and conventional "but for" test of causation but was unable to do so;

ii) Mr Holmes placed his case on causation on the basis of a material contribution in fact and did not attempt to satisfy the "but for" test either by trying to establish that his tortious exposure to TCE had "doubled the risk" or otherwise. Mr Holmes did not ask the Court to find in his favour on the basis that Poeton's breaches of duty had made a material contribution to a risk. It is apparent from the Judge's summary that what Mr Holmes was disclaiming was any case based upon the principles established by *Fairchild v Glenhaven Funeral Services Ltd* [2003] 1 AC 32 and *Barker v Corus UK Ltd* [2006] 2 AC 572.

21. The approach to the question of material contribution which Mr Holmes urged on the Judge, and which he accepted, was set out in the judgment as follows:

> "66. … the question of whether there was a material contribution in fact to Mr Holmes' Parkinson's disease must be seen precisely as that, namely a decision for the trial judge in the case of a specific individual based upon all of the available information. As such it is not a decision that is dependent on the strict parameters applied by epidemiologists. Consequently whilst the epidemiologists in the case, namely Professor McElvenny (Claimant) and Professor Jones (Defendant) conclude that general epidemiological evidence falls short of the consistent body of evidence across a number of studies, that does not mean that in this particular case there is insufficient for causation to be made out. Equally the fact that the meta-analysis undertaken by Pezzoli and Cereda (*Exposure to pesticides or solvents and the risk of Parkinson's disease Neurology 2013;80(22):2035-41*) suggests an average increased risk factor of 1.58 (ie less than doubling the risk) for exposure to solvents generally is only part of the picture. Thus the Claimant submits that this is not a case where the court needs to be concerned with whether the risk was doubled. The proper route to resolution is to look at the extent of the evidence available in its entirety."

22. The Judge then set out what he regarded as the relevant evidence from the expert neurologists and epidemiologists, to which I shall return later. Having done so he set out his reasoning and conclusions on the issue of "material contribution in fact" in and from [76]. He started by finding that "the evidence taken together clearly shows that exposure to TCE created a material contribution to the risk of the Claimant sustaining injury." He then turned to the question whether "I can conclude on that evidence that there has been a material contribution in fact… ."

23. The steps that the Judge took and the features that he took into account in addressing that question were set out at [77]-[82] of the judgment. In summary they were: (a) his conclusion that Mr Holmes was exposed during the course of his employment to "significant levels of TCE over a protracted period"; (b) cautioning himself against "necessarily concluding that an absence of conclusive research … means that there is insufficient evidence for the court to reach a conclusion at this stage"; (c) accepting that "Parkinson's disease is usually multifactorial in its cause and the relevant factors include genetics and environment in combination"; (d) concluding that "[Mr Holmes'] workplace is an obvious potential source of an environment factor"; (e) holding that Mr Holmes' workplace used significant quantities of TCE "and [Mr Holmes] was exposed to it"; (f) "[w]hile the epidemiological studies are inconclusive, there is reliable evidence as to a plausible mechanism by which TCE could contribute to the development of Parkinson's disease"; (g) there is no evidence to contradict a causative link, Poeton's case being that it was "possible that exposure to TCE made a material contribution in fact to the development of the Claimant's condition but that such causation was not established to the requisite standard on the evidence available and the present state of scientific knowledge."

24. On this basis, the Judge expressed his conclusion as follows:

> "83. Of course, multifactorial factors might involve multiple genetic factors only, and Mr Holmes would have been exposed to many environmental factors, but if I stand back and ask myself whether the propositions set out above persuade me that in this particular case, on the balance of probabilities, was the Claimant's Parkinson's disease materially contributed to in fact by his exposure to TCE at the Defendant's works then the answer is yes. In my view to conclude otherwise would be to suspend the reality of the situation and ignore that which on any analysis seems to me to be the likely reality. Ultimately this decision is a matter for the court, guided of course by the expert evidence. It is not a matter of formal epidemiological analysis."

25. The Judge then supported his conclusion by two further factors:

i) First, he relied upon the judgment of the Court of Appeal in *Wood v MOD* [2011] EWCA 792 at [78] to the effect that a court may in appropriate cases make a finding of legal causation even when that would not be regarded as established to a scientific standard of proof.

ii) Second, he relied upon a digest of some of the scientific papers referred to during the trial. He had earlier cited a paper by Lill et al (Genetics of Parkinson's Disease 2016) which said:

> "However, the vast majority of PD is genetically complex, i.e. it is caused by the combined action of common genetic variants in concert with environmental factors. The establishment of gene-environment (GxE) interaction effects has proven to be difficult in most complex diseases, PD representing no exception." (The judge had cited the first sentence. Poeton points out that he omitted the second.)

The Judge relied upon the digest as demonstrating that his citation from Lill et al represented "mainstream science".

26. That said, he recognised that:

> "Again most of the papers are capable of criticism by way of sample size, recall bias, whether they are TCE specific, etc. They also invariably reach inconclusive results. But they do in my judgment demonstrate the common theme that Parkinson's disease has a multifactorial cause and that environmental factors are very important. Furthermore they readily demonstrate that solvents and TCE in particular were to put it colloquially "in the frame"."

27. The Judge then turned to and rejected Poeton's submission that "material contribution in fact" had no application to a case involving an indivisible disease. That led to his conclusion that Mr Holmes was entitled to succeed on the basis that he "was exposed to levels of TCE in the course of his employment with [Poeton] that amounted to a breach of duty and that such exposure made a material contribution in fact to [his] development of Parkinson's disease."

The Grounds of Appeal

28. Poeton advance five grounds of appeal. I would change the order of the grounds and summarise the issues that are raised as follows:

i) The judge adopted the wrong test in law for proving causation, namely that it was sufficient in an indivisible injury case to demonstrate a material contribution to injury regardless of whether, but for the tort, the injury would have occurred in any event: "material contribution" and the "but for" test;

ii) The judge misunderstood the evidence before him in assessing whether exposures to TCE in excess of occupational exposure limits (OELs) occurred. His findings as to the level of exposure are therefore flawed: levels of exposure;

iii) The judge made a finding that exposure to TCE was capable of causing Parkinson's disease when on the evidence that was no more than a theory in respect of which much of the relevant literature cast doubt: the evidence therefore did not support a finding of generic causation on the balance of probabilities: generic causation;

iv) The judge held that a material contribution was established when the evidence demonstrated no more than an elevated risk, falling short of doubling, of contracting Parkinson's disease as a result of exposure to TCE: individual causation;

v) The finding of individual causation in this case was speculative and unsupported by any evidence even if generic causation was established in light of the fact that PD is multifactorial and involves a complex interplay of genetic and environmental factors (which can be either internal or external to the patient) of which TCE is only one possible candidate. The case should have been treated

as falling within the ambit of the principles enunciated in *Wilsher v Essex Area Health Authority* [1988] AC 1074: Wilsher.

Issue 1: "material contribution" and the "but for" test of causation

29. Poeton submits that the Judge adopted the wrong test for proving causation. Poeton's case on this appeal has been that it was wrong to rely upon a "material contribution" test at all since such a test has no application to cases of indivisible injury such as the contraction of Parkinson's disease; and that, even if it was appropriate to consider a test based on "material contribution", that did not remove from Mr Holmes the need to prove that the tortious exposure to TCE for which Poeton was responsible was itself a "but for" cause of his Parkinson's disease.

30. This ground of appeal raises complex and difficult questions of law in an area that has been bedevilled by apparent inconsistency and imprecision at the highest level on multiple occasions. It has, not surprisingly, engendered considerable debate both in the authorities and by contributions from academic scholars, amongst whom Professors Jane Stapleton, Sandy Steel and Sarah Green have been pre-eminent in applying levels of analysis that are frequently absent in the authorities. It may therefore be convenient to start by trying to establish some preliminary points that inform the issue and its resolution.

Divisible and indivisible diseases

31. First, the terms "divisible" and "indivisible" disease or injury are ubiquitous. They are (or should be) now well understood but have been a source of confusion in the authorities. It is a characteristic of divisible diseases that, once initiated, their severity will be influenced by the total amount of the agent that has caused the disease. By contrast, once an indivisible disease is contracted, its severity will not be influenced by the total amount of the agent that caused it. The classic distinction in asbestos-related diseases is between asbestosis and mesothelioma. Mesothelioma is an indivisible disease because, although the risk of developing a mesothelioma increases in proportion to the quantity of asbestos dust and fibres inhaled, the condition once caused is not aggravated by further exposure and the severity of the condition, if it occurs, is not thought to be affected by variations in the victim's overall exposure. Asbestosis is a divisible disease because all of the victim's exposure to asbestos will contribute to the severity of his eventual disease: see *Fairchild* at [6] per Lord Bingham of Cornhill.

32. Noise-induced hearing loss and pneumoconiosis are divisible diseases. *Holtby v Brigham & Cowan* [2000] 3 All ER 421 established that, in a case of a divisible disease such as asbestosis, the burden rests upon the Claimant to prove that the defendant who has been sued was responsible for the whole or a quantifiable part of his disability. This approach is conceptually straightforward with a divisible disease, though difficult evidential questions of proof may arise. It is the approach that was adopted by Mustill J in *Thompson v Smiths Shiprepairers (North Shields) Ltd* [1984] QB 405 at 437-444. Integral to that reasoning is that "I see no reason why the present impossibility of making a precise apportionment of impairment and disability in terms of time, should in justice lead to the result that the defendants are adjudged liable to pay in full, when it is known that only part of the damage was their fault." One complication in the field of noise-induced hearing loss that militates against making a precise apportionment of responsibility is that the progression of the disease is not constant; by contrast, the Court

of Appeal in *Holtby* treated the progression of asbestosis as linear depending on the amount of dust inhaled. These principles for the assessment of causation and proof of loss in divisible diseases have, to my knowledge, never been doubted, and are well established.

Wilsher and Fairchild

33. In mapping out the legal terrain for Issue 1, *Wilsher v Essex AHA* is of fundamental importance. The plaintiff was born prematurely. The Defendant negligently administered excessive oxygen to the newly born plaintiff. The plaintiff succumbed to retrolental fibroplasia ("RLF"), which is a well-known hazard of prematurity. It was common ground that the administration of excess oxygen could have caused the plaintiff's RLF and the Judge held that it increased the risk that he would suffer RLF; but there were four other potential causes of the plaintiff's RLF with the result that the Defendant's negligence was only one of a number of possible causes of the plaintiff's RLF.

34. The trial judge and the Court of Appeal found for the plaintiff. The House of Lords unanimously allowed the defendant's appeal on the grounds that they had adopted the wrong principle of causation. The House adopted the dissenting judgment of Sir Nicolas Browne-Wilkinson in the Court of Appeal, who had said at [1987] QB 730, 779:

> "To apply the principle in *McGhee v. National Coal Board* [1973] 1 W.L.R. 1 to the present case would constitute an extension of that principle. In the *McGhee* case there was no doubt that the pursuer's dermatitis was physically caused by brick dust: the only question was whether the continued presence of such brick dust on the pursuer's skin after the time when he should have been provided with a shower caused or materially contributed to the dermatitis which he contracted. There was only one possible agent which could have caused the dermatitis, viz., brick dust, and there was no doubt that the dermatitis from which he suffered was caused by that brick dust.
>
> "In the present case the question is different. There are a number of different agents which could have caused the RLF. Excess oxygen was one of them. The defendants failed to take reasonable precautions to prevent one of the possible causative agents (e.g. excess oxygen) from causing RLF. But no one can tell in this case whether excess oxygen did or did not cause or contribute to the RLF suffered by the plaintiff. The plaintiff's RLF may have been caused by some completely different agent or agents, e.g. hypercarbia, intraventricular haemorrhage, apnoea or patent ductus arteriosus. In addition to oxygen, each of those conditions has been implicated as a possible cause of RLF. This baby suffered from each of those conditions at various times in the first two months of his life. There is no satisfactory evidence that excess oxygen is more likely than any of those other four candidates to have caused RLF in this baby. To my mind, the occurrence of RLF following a failure to take a

> necessary precaution to prevent excess oxygen causing RLF provides no evidence and raises no presumption that it was excess oxygen rather than one or more of the four other possible agents which caused or contributed to RLF in this case.
>
> "The position, to my mind, is wholly different from that in the *McGhee* [1973] 1 W.L.R. 1, case where there was only one candidate (brick dust) which could have caused the dermatitis, and the failure to take a precaution against brick dust causing dermatitis was followed by dermatitis caused by brick dust. In such a case, I can see the common sense, if not the logic, of holding that, in the absence of any other evidence, the failure to take the precaution D caused or contributed to the dermatitis. To the extent that certain members of the House of Lords decided the question on inferences from evidence or presumptions, I do not consider that the present case falls within their reasoning. A failure to take preventative measures against one out of five possible causes is no evidence as to which of those five caused the injury."

35. It is to be noted that the House of Lords in *Wilsher* by adopting this statement of principle, rejected any suggestion that there could be a reversal of the burden of proof or any modified approach to causation despite the fact that the judge's finding of negligence on the part of the Defendant was not disturbed. The position was neatly encapsulated by Lord Rodger of Earlsferry in *Fairchild* at [149]: "there was nothing to show that the risk which the defendant's staff had created - that the plaintiff would develop [RLF] because of an unduly high level of oxygen – had eventuated."

36. *McGhee* was interpreted by the House of Lords in *Wilsher* as giving rise to no special principle of causation. It subsequently came to be interpreted as an application "avant la lettre" of the *Fairchild* exception: see *Barker v Corus UK Ltd* [2006] 2 AC 572 at [13] per Lord Hoffmann. Since no reliance is placed by Mr Holmes on the *Fairchild* exception, I will refer to it relatively briefly as another marker post on the causation map.

37. The essential challenge that *Fairchild* and *Barker* sought to address was outlined by Lord Bingham in [2] of *Fairchild*. There were variations in the various speeches, but the central problem was that, while it was known that a victim's mesothelioma had been caused by exposure (or exposures) to asbestos which had increased the risk of developing mesothelioma, the victim could not, because of the limits of medical science, prove on the balance of probabilities that his mesothelioma was the result of exposure to asbestos during his employment with employer A, or during his employment with employer B, or during his employment by A and B taken together. The response of the House of Lords in *Fairchild* was that there should be a modified approach to proof of causation and that proof that an employer's exposure had increased the risk that the victim would develop his mesothelioma was sufficient to satisfy the causal requirements for that employer to be held liable for the full consequences of the mesothelioma. As was made clear, no one in *Fairchild* took the point that a victim in such a case should recover less than 100% damages: see Lord Bingham at [34]; but the point was taken in *Barker*. The House of Lords' decision in *Barker* was that a victim in such a case could recover in proportion to the extent to which the sued employer had

increased the risk of his developing mesothelioma. That decision was rapidly reversed by s. 3 of the Compensation Act 2006 in relation to mesothelioma only, so that a mesothelioma victim who established against a defendant that he had been exposed to asbestos in circumstances that the common law held to be tortious would recover 100% damages.

38. One of the key features of the decisions in *McGhee, Fairchild* and *Barker* was that there was a single agent operating to cause the victim's injury – brick dust in the case of *McGhee*, asbestos in *Fairchild* and *Barker*. The significance of there being one single agent has been the subject of subsequent debate, which it is not necessary to pursue here; but it forms a central point of distinction between the *Fairchild* exception and a case such as *Wilsher* where there are a number of different potential agents (some of which may be tortious, others not) and the challenge is to decide which was operative. In a *Wilsher* case, the burden is on the claimant throughout to prove that the defendant's tort caused the injury, that being decided on the basis of the "but for" test on the balance of probability.

"Material contribution" since Bonnington

39. The phrase "material contribution" was used in a number of different legal contexts before the decision in *Bonnington Castings Ltd v Wardlaw* [1956] AC 613. There is to my mind no good reason to think that the House of Lords in *Bonnington* was adopting any particular meaning for the phrase. In particular, I would not accept that the phrase was used in *Bonnington* to denote unnecessary causes, thereby circumventing the requirement that a party must prove their case by showing that "but for" the defendant's tortious conduct they would not have suffered the damage. For that reason, it is not necessary to go back further than *Bonnington* in order to determine the origins and meaning of the phrase in the present context.

40. In *Bonnington* the pursuer contracted pneumoconiosis having been exposed to silica dust which emanated from two sources. He was exposed to dust from the pneumatic hammers without his employer being in breach of duty, because there was no known or practicable extraction plant that could prevent that exposure: his exposure was in that sense "innocent". He was also exposed to dust from the swing grinders, which exposure was "tortious" because the extraction plant fitted to the swing grinders was not kept free from obstruction, in breach of relevant statutory regulations. At page 619 Lord Reid identified the issue as being "whether this breach of the regulation caused the respondent's disease. If his disease resulted from his having inhaled part of the noxious dust from the swing grinders which should have been intercepted and removed, then the appellants are liable to him in damages: but if it did not result from that, then they are not liable."

41. The Courts below had held that, once breach of duty was established, there was an onus on the defenders to prove that the tortious dust from the swing grinders did not cause the pursuers' disease. That approach was rejected. At page 620 Lord Reid stated the correct approach:

> "It would seem obvious in principle that a pursuer or plaintiff must prove not only negligence or breach of duty but also that such fault caused or materially contributed to his injury, and there is ample authority for that proposition both in Scotland and

> in England. …The fact that Parliament imposes a duty for the protection of employees has been held to entitle an employee to sue if he is injured as a result of a breach of that duty, but it would be going a great deal farther to hold that it can be inferred from the enactment of a duty that Parliament intended that any employee suffering injury can sue his employer merely because there was a breach of duty and *it is shown to be possible that his injury may have been caused by it*. In my judgment, the employee must in all cases prove his case by the ordinary standard of proof in civil actions: he must make it appear at least that on a balance of probabilities the breach of duty caused or materially contributed *to his injury*." (Emphasis added. See also per Lord Tucker at page 624 and Lord Keith at page 625)

42. I have already referred to the fact that pneumoconiosis is a divisible disease. There is no reason to suppose that the members of the House of Lords did not know or appreciate that basic fact and there are some indications that they clearly did. For example Lord Reid said at 621 that "pneumoconiosis is caused by a gradual accumulation in the lungs of minute particles of silica inhaled over a period of years"; and Lord Keith said at page 626 that "silica dust, when inhaled, is gradual and insidious in its effects and requires to operate on the lungs for a considerable period of time before producing pneumoconiosis"; and "the disease is a disease of gradual incidence." Yet the approach in the speeches was at least equivocal and more naturally indicative of indivisible diseases. Thus, at 621 Lord Reid continued:

> "That means, I think, *that the disease is caused by the whole of the noxious material inhaled* and, if that material comes from two sources, it cannot be wholly attributed to material from one source or the other. I am in agreement with much of the Lord President's opinion in this case, but I cannot agree that the question is: which was the most probable source of the respondent's disease, the dust from the pneumatic hammers or the dust from the swing grinders? *It appears to me that the source of his disease was the dust from both sources*, and *the real question is whether the dust from the swing grinders materially contributed to the disease*. What is a material contribution must be a question of degree. A contribution which comes within the exception de minimis non curat lex is not material, but I think that any contribution which does not fall within that exception must be material. I do not see how there can be something too large to come within the de minimis principle but yet too small to be material." (Emphasis added)

43. At 622 Lord Reid held it to be established that the dust from the swing grinders made "a substantial contribution". He explained:

> "It is … probable that much the greater proportion of the noxious dust which he inhaled over the whole period came from the hammers. But, on the other hand, some certainly came from the swing grinders, and I cannot avoid the conclusion that the proportion which came from the swing grinders was not

> negligible. He was inhaling the general atmosphere all the time, and *there is no evidence to show* that his hammer gave off noxious dust so frequently or that the concentration of noxious dust above it when it was producing dust was so much greater than the concentration in the general atmosphere, *that that special concentration of dust could be said to be substantially the sole cause of his disease*." (Emphasis added).

44. At 623 Lord Reid concluded:

> "No doubt the total amount from both sources in the atmosphere was small at any one time, but the combined effect over a period of eight years was to cause the respondent's disease. … In my opinion, it is proved not only that the swing grinders may well have contributed but that they did in fact contribute a quota of silica dust which was not negligible to the pursuer's lungs and therefore *did help to produce the disease*. That is sufficient to establish liability against the appellants, and I am therefore of opinion that this appeal should be dismissed." (Emphasis added)

45. Lord Tucker at 623 concluded that the silica dust from the swing grinders contributed to the harmful condition of the atmosphere, "which admittedly resulted in the pursuer contracting pneumoconiosis, and was therefore a contributory cause of *the disease*." (Emphasis added). And at 626, Lord Keith said:

> "The disease is a disease of gradual incidence. Small though the contribution of pollution may be for which the defenders are to blame, it was continuous over a long period. In cumulo it must have been substantial, though it might remain small in proportion. It was the atmosphere inhaled by the pursuer that caused his illness and *it is impossible, in my opinion, to resolve the components of that atmosphere into particles caused by the fault of the defenders and particles not caused by the fault of the defenders, as if they were separate and independent factors in his illness.* Prima facie the particles inhaled are acting cumulatively, and I think the natural inference is *that had it not been for the cumulative effect the pursuer would not have developed pneumoconiosis when he did and might not have developed it at all*." (Emphasis added)

46. The pursuer in *Bonnington* recovered 100% damages. It has been pointed out in subsequent authorities that no argument appears to have been presented to the House of Lords to the effect that pneumoconiosis was a divisible disease and that, accordingly, the defenders should in any event only be liable for a proportion of 100% damages. To my mind, that misses the point. Whatever the reason, the House of Lords treated the pursuer's claim as if it were a claim in respect of an indivisible disease: see the highlighted passages in the citations above. This may have been because the defenders did not raise the argument that pneumoconiosis was a divisible disease; or, as stated by Lord Porter, because it was not possible to resolve the components of the atmosphere into particles caused by the fault of the defenders and particles not so caused. Either way, the statements of principle in *Bonnington* about making a material contribution to

"the disease" were not expressed in terms that were appropriate to allocation of responsibility for divisible diseases. It therefore seems to me that Poeton in the present case can draw no comfort from the fact that *Bonnington* was a case of pneumoconiosis, which is known to be a divisible disease: the *Bonnington* principle was expressed in terms that were appropriate to indivisible diseases rather than to divisible ones.

47. *Bonnington* gives little guidance on what is sufficient to constitute a "material contribution". As set out above, Lord Reid said that it is a question of degree and that a contribution which comes within the exception de minimis non curat lex is not material. Otherwise, it is material; and the tortious contribution from the swing grinders was "not negligible" and therefore sufficed.

48. *Nicholson v Atlas Steel Foundry and Engineering Co Ltd* [1957] 1 WLR 613 was another case of pneumoconiosis. Once more the dressing shop where the pursuer worked was affected by silica dust which emanated from two sources: first, from the pneumatic hammer, in respect of which there was no obligation to fit appliances to intercept the dust; and, second, from swing grinders. There was no means of ventilating the shop, as a result of which the defenders were held to be in breach of their duty to secure adequate ventilation. The arguments closely followed those advanced in *Bonnington*. The tortious failure to ventilate the shop meant that the pursuer was exposed to a greater extent than he should have been. Although it was impossible to quantify, even approximately, the (innocent) particles which he must in any event have inhaled and those (tortious) particles which he inhaled but need not have, the excess was held not to be negligible and the defenders were held liable.

49. Viscount Simonds (who had been a party to the decision in *Bonnington*) held at page 616 that the question was:

> "… whether in addition to these particles he was, owing to the fault of the respondents, bound to inhale a number of other particles which made a material contribution *to his illness*. In determining whether a material contribution was made, I must apply the test recently laid down in this House in *Bonnington Castings Ltd. v. Wardlaw* and say that a contribution is material unless the maxim " de minimis " can be applied to it." (Emphasis added.)

50. Having reviewed the evidence he concluded that the effect of the defender's breach was that noxious particles were present (or, in Lord Keith's words, hung around) for longer than would have been the case in the absence of breach; and he concluded that the incremental tortious exposure, which he described as "the excess", was not de minimis and therefore the defenders were liable, following *Bonnington*.

51. It is, in my judgment, possible to detect the same ambiguity about the nature of the disease as is apparent in *Bonnington*. Thus Lord Cohen said at page 622:

> "Pneumoconiosis is a progressive disease. *The longer a workman is exposed to an intense cloud the graver must be the risk of infection.* In the present case it is clearly established by the evidence that at any rate down to 1949 the tool with which the deceased was working on dirty castings created a thick cloud

> of dust which must have necessarily included siliceous particles to an extent which cannot be classed as "de minimis." The respondents are admittedly not to blame for the generation of this cloud, but any failure to provide proper ventilation must, I think, lengthen the period during which the cloud remains intense. It seems to me to follow that *the respondents' failure to provide adequate ventilation must increase the risk to which the workmen are exposed.* Reading the evidence as a whole, I think it establishes that (to use the language of Lord Reid in Wardlaw's case) "on a balance of probabilities the breach of duty caused or materially contributed to" the injury." (Emphasis added)

52. As in *Bonnington*, it does not appear that the defender contended for an award of less than 100% damages if breach of duty were to be established. For the same reasons as set out at [46] above, I do not consider that the fact that the disease in *Nicholson* was pneumoconiosis leads to the conclusion that the statement of principle articulated in *Bonnington* and applied in *Nicholson* is directed (let alone solely directed) to cases of divisible injury or disease.

53. Conversely, I consider that *McGhee* provides substantial support for the submission that the *Bonnington* principle applies to cases of indivisible disease or injury. At 4C-E, Lord Reid restated the principle in terms that, to my mind, are only consistent with treating it as applicable to indivisible disease or injury:

> "It has always been the law that a pursuer succeeds if he can show that fault of the defender caused or materially contributed to *his injury*. There may have been two separate causes but it is enough if one of the causes arose from fault of the defender. *The pursuer does not have to prove that this cause would of itself have been enough to cause him injury*. That is well illustrated by the decision of this House in *Bonnington Castings Ltd. v. Wardlaw* [1956] A.C. 613. There the pursuer's disease was caused by an accumulation of noxious dust in his lungs. The dust which he had inhaled over a period came from two sources. The defenders were not responsible for one source but they could and ought to have prevented the other. *The dust from the latter source was not in itself sufficient to cause the disease but the pursuer succeeded because it made a material contribution to his injury*." (Emphasis added)

54. Any residual doubt that Lord Reid was applying the principle as if the injury concerned was an indivisible injury is dispelled by what follows. The respondents had sought to distinguish *Bonnington* by arguing that in that case "it was proved that every particle of dust inhaled played its part in causing the onset of the disease whereas in this case it is not proved that every minor abrasion played its part." Lord Reid then posited two alternative mechanisms. The first was that "an accumulation of minor abrasions of the horny layer of the skin is a necessary precondition for the onset of the disease. Or it may be that the disease starts at one particular abrasion and then spreads so that multiplication of abrasions merely increases the number of places where the disease can start and in that way increases the risk of its occurrence." Lord Reid inclined to think that the evidence pointed to the former view, but was not in a position to say that

it was proved. But he added that "if [the former view] were [proved], then this case would be indistinguishable from [*Bonnington*]." To my mind, this indicates that the *Bonnington* principle had been and was directed to a case where the tortious exposure contributed to the onset of the disease and not its severity. That is at least consistent with the observation of Lord Simon at page 7H that the consultants had not gone so far as to say that washing after work would have made it more probable than not that the appellant would have escaped dermatitis, with no suggestion that the case was concerned about the recovery of damages for part only of the consequences of the dermatitis; and the restatement of the *Bonnington* principle by Lord Simon at page 8C-D. And, at page 11G Lord Simon said that "a factor, by itself, *may not be sufficient to cause injury* but if, with other factors, it materially contributes to causing injury, it is clearly a cause of injury" (emphasis added). As with the passage from the speech of Lord Reid at page 4C-E cited above, this is not the language of divisible injuries.

55. The *Bonnington* principle was referred to in *Fairchild*, most pertinently for present purposes by Lord Rodger at [129]. Having identified that the idea of liability based on wrongful conduct that had materially contributed to an injury was well established before *Bonnington*, he said:

> "But [*Bonnington*] became a convenient point of reference, especially in cases of industrial disease. In such cases this basis of liability is of considerable importance. Since it is enough that the defendant's wrongful act materially contributed to the claimant's injury, the law is not applying the causa sine qua non or "but for" test of causation. In [*Bonnington*], for instance, the pursuer did not need to prove that, but for the dust from the swing hammers, he would not have developed pneumoconiosis. All he needed to prove was that the dust from the swing hammers contributed materially to the dusty atmosphere which he breathed and which caused his illness."

56. The House of Lords returned to the question of causation in different factual circumstances in *Simmons v British Steel PLC* [2004] UKHL 20, [2004] S.C. (H.L.) 94. The pursuer sustained a severe blow to his head caused by the fault of the defenders. After the accident the pursuer experienced an exacerbation of a pre-existing skin condition and developed a change in his personality which resulted in a severe depressive illness. The main discussion in the judgments centred on the issue of remoteness and whether there was a sufficient causal connection between the happening of the accident and the exacerbation of his skin condition and depressive illness. Both Lord Hope and Lord Rodger engaged in wide-ranging discussions of principle. In the course of his speech, Lord Hope at [18] added what he called "a comment on causation" to his discussion of "the grand rule on the subject of damages" (see [14]):

> "The general rule is that it must be shown that the injury would not have occurred but for the act or omission of the defender. But if a number of factors contributed to the injury it is sufficient that the contribution which the factor attributable to the defender's fault made to the injury was material ([*Bonnington*], per Lord Reid …)." (This appears to be the passage cited by the judge at [61] of the judgment below.)

57. Lord Rodger at [58] addressed the question how the pursuer's anger at the defender's treatment of him after the accident was to be treated in relation to his development of his psoriasis:

> "It is important to notice that, …, the Lord Ordinary does not single out the defenders' treatment of the pursuer after the accident. If he had, the *Graham* case would have been in point and the Lord Ordinary would have been entitled to regard his anger at the defenders' failure to visit him or show any interest in him as the, distinct, operative cause of his psoriasis and, hence, of his depressive illness. But, as the Lord Ordinary himself found, this was only one among a number of factors, all of which brought about his condition. His anger at the defenders that the accident had occurred at all, despite the warnings, also made a material contribution to the development of his condition. Before the House, senior counsel for the defenders sought to argue that the principle in *[Bonnington]* did not apply in this situation, but he cited no authority for his proposition and, in my view, it is unsound. The usual rule applies and, in the absence of any basis for identifying and apportioning the respective roles played by the various factors in the development of the pursuer's condition, the pursuer is entitled to recover damages for all of his injuries."

58. Both of these statements of general principle are expressed in terms that are directly applicable to indivisible injuries. Lord Rodger was more explicit still in *Barker* where, although dissenting overall, he articulated general statements of principle that are not in doubt. At [72] he said:

> "[*Bonnington*] soon became established as the classic authority for the proposition that, to succeed and recover damages in full against any defendant, a plaintiff need prove no more than that the defendant's wrongful act materially contributed to his injury. Since anything above de minimis will do, this means that a claimant can succeed even though the injury would have occurred without the defendant's act. The "but for" or sine qua non test of causation gives way to this considerably more generous test based on the defendant's material contribution to the victim's injury."

And, at [90] he said:

> "Of course, it may seem hard if a defendant is held liable in solidum even though all that can be shown is that he made a material contribution to the risk that the victim would develop mesothelioma. *But it is also hard - and settled law - that a defendant is held liable in solidum even though all that can be shown is that he made a material, say 5%, contribution to the claimant's indivisible injury*." (Emphasis added)

59. Lord Rodger repeated the position in *Sienkiewicz v Greif (UK) Ltd* [2011] 2 AC 229 at [138]:

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

60. It need hardly be said that *Simmons*, *Barker* and *Sienkiewicz* were all cases of indivisible injuries. There is no basis for suggesting that these statements of principle at the highest level were relevant only to the causation of divisible injuries or disease. To my mind it seems clear that the *Bonnington* principle of "material contribution" should apply to indivisible diseases; divisible diseases are approached differently, as explained in *Holtby* and *Thompson v Smiths Shiprepairers (North Shields) Ltd*.

61. The interrelationship of the *Bonnington* principle and the "but for" test arose for decision in *Bailey v MOD* [2009] 1 WLR 1052 (CA). The claimant suffered a cardiac arrest and consequent brain damage because she vomited, was unable to clear her throat, and aspirated her vomit. The Judge found that she was unable to clear her throat because of weakness that had two cumulative causes. One cause was pancreatitis, which was not attributable to fault on the part of the defendant; the other cause was her stormy passage over a number of days, which was the result of the defendant's negligent failure to provide proper care. The outcome was therefore an indivisible injury, the cause of which was the claimant's weakened state which in turn was attributable to two cumulative features, one "innocent" and one "tortious".

62. The defendant's third ground of appeal was that the right test for the judge to have applied was the "but for" test and that, unless the claimant could establish that, but for the defendant's negligence, the claimant's injury would not have happened, she could not succeed. That proposition was rejected. Waller LJ (with whom Sedley and Smith LJJ agreed) endorsed Lord Rodger's statement of principle at [129] of *Fairchild*, which I have set out above. The ratio for his decision on this point is at [46]-[47] and included the following:

> "46. … In a case where medical science cannot establish the probability that "but for" an act of negligence the injury would not have happened but can establish that the contribution of the negligent cause was more than negligible, the "but for" test is modified, and the claimant will succeed."
>
> 47. The instant case involved cumulative causes acting so as to create a weakness and thus the judge in my view applied the right test, and was entitled to reach the conclusion he did."

63. The reasoning in *Bailey* has been subjected to sustained criticism, not least by Professor Stapleton (Unnecessary Causes, LQR 2013, 39-65 at 50-54). It may well be asked how a principle first enunciated in a case involving a quintessential divisible disease has come to be established as a test that bypasses the need to satisfy the "but for" test in cases of individual injury that do not come within the *Fairchild* exception. It might, as Professor Stapleton suggests, derive from a response to an evidentiary gap in divisible

injury cases where there is no satisfactory acceptable evidentiary basis from which the disability due to the separate insults to the body could be apportioned to the individual sources. But, if so, its transference to the field of indivisible injury or disease has not been explained apart from the recognition that the principle is generous to claimants. I would, however, hold that we are bound in the light of *Bailey* to find that the *Bonnington* "material contribution" principle applies to cases of indivisible injury and that, where the principle applies, the claimant does not have to show that the injury would not have happened but for the tortious exposure for which the defendant is responsible.

64. Poeton relies upon a passage from the judgment of the Court of Appeal in the atomic veterans case (*AB v Ministry of Defence* [2010] EWCA Civ 1317, (2011) 117 BMLR 101). The case came before the Court of Appeal on applications to strike out and questions of limitation. The claimants sued in respect of a wide variety of conditions for which they alleged the defendant to be responsible because they had been exposed to ionising radiation during tests of thermonuclear devices in the region of the Pacific Ocean between 1952 and 1958. It was common ground that all the conditions of which the claimants complained had several different possible causes besides radiation: see [130]. Unsurprisingly, the Court of Appeal's first reason for finding against the claimants was that the case fell within the ambit of *Wilsher* and, unless there were to be an extension of *Fairchild*, the claimants would have to show "but for" causation, which they could not do. However, in response to a further submission from the defendant, the Court of Appeal said at [130] of the *Bonnington* principle:

> "This principle applies only where the disease or condition is 'divisible' so that an increased dose of the harmful agent worsens the disease. As is well known, in *Bonnington*, the claim succeeded because the tortious exposure to silica dust had materially aggravated (to an unknown degree) the pneumoconiosis which the claimant might well have developed in any event as the result of non-tortious exposure to the same type of dust. The tort did not increase the risk of harm; it increased the actual harm. Similarly in *Bailey*, the tort (a failure of medical care) increased the claimant's physical weakness. She would have been quite weak in any event as the result of a condition she had developed naturally. No one could say how great a contribution each had made to the overall weakness save that each was material. It was the overall weakness which led to the claimant's failure to protect her airway when she vomited with the result that she inhaled her vomit and suffered a cardiac arrest and brain damage. In those cases, the pneumoconiosis and the weakness were divisible conditions."

65. With the utmost respect for the Court of Appeal in that case, I am unable to accept or adopt this analysis. For the reasons I have tried to explain, *Bonnington* itself was expressed in terms that were appropriate to indivisible rather than divisible diseases and the principle has since been accepted at the highest level on multiple occasions as applying to indivisible diseases; and *Bailey* was a case directly in point because the injury suffered by the claimant was indivisible. Nor did [98] of the speech of Lord Phillips in *AB v Ministry of Defence* [2013] 1 AC 78 on appeal to the Supreme Court assist the claimants. He there pointed out that, in circumstances where there were other

potential causes of their injuries, there was no known basis for concluding that the exposure would have gone so far as to double the risk of sustaining the injury of which the individual claimants complained. Proof that exposure had increased the risk (short of doubling it) was insufficient because the claimants were not within the *Fairchild* exception. In other words, these were claims falling within the ambit of *Wilsher*.

66. In *Williams v Bermuda Hospitals Board* [2016] AC 888 the Privy Council explained *Bonnington* on the basis that there had been no suggestion that the pneumoconiosis was "divisible", meaning that the severity of the disease depended on the quantity of dust inhaled; and that Lord Reid had interpreted the medical evidence as meaning that the particles from the swing grinders were a cause of the entire disease: see [32]. I agree, for the reasons I have set out above. In *Williams* there had been a culpable delay of at least 140 minutes before a necessary operation was performed, as a result of which sepsis continued to develop incrementally for longer than would otherwise have been the case. At [42] the Privy Council held that "it is right to infer … that the hospital board's negligence materially contributed of the process [of accumulation of sepsis increasing the oxygen requirement] and therefore materially contributed to the injury to the heart and lungs." In context it is plain that the Privy Council treated the delay as materially contributing to the causation of the resulting injury to the patient's heart and lungs, which was treated as an indivisible injury.

67. Before leaving *Williams* it should also be noted that the Privy Council in an obiter passage at [47] interpreted *Bailey* as being a case where "but-for" causation was established. In my judgment this passage is liable to cause confusion and should not be adopted: see Stapleton and Steel (Causes and Contributions LQR 2016, 132, 363-369 at 366-367), Steel (Material contribution to damage, again LQR 2022 138, 540-545 at 543).

68. The final authority to which I think it may be helpful to refer is *Heneghan v Manchester Dry Docks Ltd* [2016] 1 WLR 2036. The deceased died of lung cancer. The claimant sued six former employers who were alleged to have exposed the deceased to asbestos, which exposure was alleged to have caused his lung cancer. Earlier employers who had also exposed the deceased to asbestos were not sued. It was common ground that the lung cancer was caused by exposure to asbestos fibres. However, it was not possible to say which, if any, of the defendants' tortious exposures led to the deceased contracting the disease; all that could be said was that each defendant increased the risk that he would contract it: see [3]. At first instance Jay J applied the *Fairchild* exception and awarded an aliquot share of 100% damages against each defendant in proportion to their tortious contribution to the deceased's overall exposure. His decision was upheld by the Court of Appeal.

69. Before both the trial judge and the Court of Appeal the claimant submitted that the evidence demonstrated that each of the six defendants materially contributed to the deceased's lung cancer and that the *Bonnington* principle applied so that he should recover 100% damages. In discussing and rejecting that submission, Lord Dyson MR at [34] adopted the reasoning of the High Court of Australia in *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111, including the passage:

> "The issue in *Bonnington Castings* was whether one source of an injurious substance contributed to a gradual accumulation of dust that resulted in disease. … Questions of material

> contribution arise only if a connection between [the victim's] inhaling asbestos and his developing cancer was established. Knowing that inhaling asbestos can cause cancer does not entail that in this case it probably did. For the reasons given earlier, that inference was not to be drawn in this case. Questions of what is a material contribution do not arise."

70. Turning to the evidence before him, Lord Dyson concluded that "the epidemiological evidence permitted the contribution to the risk of cancer attributable to an individual defendant to be quantified. But it went no further than that": see [42]. That led him to the conclusion that *Bonnington* was inapplicable and that "the response of the law to the problem posed in a case where the scientific evidence does not permit a finding that the exposure attributable to a particular defendant contributed to the injury is to apply the *Fairchild* exception": see [47]. I respectfully agree even though I also agree with Professor Sarah Green that the decision in *Heneghan* involved an extension of *Fairchild*: see Fairchild and the Single Agent Criterion LQR 2017 25-31.

71. For these reasons I reject Poeton's submission on Issue 1 that *Bonnington* is only applicable to cases of divisible injury.

Issue 2: levels of exposure

72. I have summarised the judge's findings on the frequency and levels of Mr Holmes' exposure to TCE at [13] to [16] above. Two features immediately emerge. First, the judge did not make specific findings either about the duration or levels of the exposure. While it would be unreasonable to have expected mathematical precision, the likelihood of the TCE exposure having contributed to a mechanism involving the destruction of dopaminergic neurons must be affected by both the frequency and the levels of exposure. Thus the extreme generality of the judge's findings about the duration and levels of exposure has a knock-on effect when considering what findings were open to the judge when he came to the issues of general and individual causation. To give specific examples, the judge made no finding about what proportion of Mr Holmes' time was spent during periods 1 and 2 operating the degreasing tank in the way described or in its immediate vicinity; nor did he make any finding about how long in a given week Mr Holmes would have spent doing cold degreasing or being in the immediate vicinity while others did it; nor did he resolve the question of how frequently and for how long Mr Holmes would have been exposed to very high levels of TCE (including levels that would carry an immediate risk to life) while cleaning the inside of the tank: he merely accepted that a "substantial" period would have been spent inside and that Mr Holmes carried out "a substantial proportion" of the tasks. More generally, the judge did not draw any distinction between periods 1 and 2, though Mr Holmes accepted that his exposure was less during period 2 than period 1 despite him being a "hands-on" foreman.

73. Second, though the judge gave general descriptions of the levels of exposure to which Mr Holmes was exposed, he did not attempt to identify the extent to which any exposure was not tortious (because not falling outside the accepted safe limits) and what the incremental quantity and effect of the tortious exposures may have been. One response could be that the evidence did not permit of such an analysis; but even that response may have implications when assessing whether causation has been proved on the basis of that evidence.

The degreasing tank

74. Poeton makes three criticisms of the judge's findings about exposure from the degreasing tank. First, in the light of the judge's acceptance that the degreasing tank was not a "rogue" tank, they point to the acceptance by Professor Cherrie (the expert on Occupational Hygiene instructed on behalf of Mr Holmes) that the starting point was that an operator working at this design of degreaser tank would have been exposed to levels that were well within the short- and long-term threshold limit values. This acceptance is not specifically mentioned in the judgment; but the point was addressed by the judge on the basis that, although the tank was not a "rogue" tank, levels of exposure would be higher if, as he found, the system of "hoiking out" or similar work practices took place. In his judgment, whether the components were rested on the rim of the tank or put on the floor next to the tank (rather than being left in the freeboard zone) the effectiveness of the lip extraction would be "circumvented", by which he meant that its effectiveness would be compromised. That, as a general proposition, was a conclusion that was open to him on the evidence though it does not answer the question of what levels of exposure and with what frequency Mr Holmes was exposed to as a result of these practices. What is plain from Professor Cherrie's evidence is that the level of exposure would have to be increased by a significant margin before the short-term limit was breached.

75. Second, Poeton criticises the judge's finding that the absence of a hoist led to tortious exposure. The judge relied upon agreed evidence from the Occupational Hygienists at paragraph 2.5 of their Joint Statement:

> "It is our joint opinion that if the Court accepts the account of the Claimant and his witnesses, *and that conditions were as described above*, the TCE exposure of the Claimant while involved in using the degreasing tank during Period 1 would have been high (Professor Cherrie will say probably around 80 ppm or 420 mg/m^3 as an 8-hour time-weighted average) with peak TCE concentrations around 300 ppm (1,700 mg/m^3). Under these circumstances, such concentrations could have been amongst the highest in degreasing processes in the UK during the 1980s (based on the scientific paper by Shipman and Winn, …). We agree that if these circumstances prevailed, it is likely that the TCE short-term limit value (ppm, 802 mg/m^3 over 10-minutes would have been exceeded and exposures may have exceeded the long-term limit on some days (100 ppm, 535 mg/m^3 as an 8-hour time-weighted average." (emphasis added)

76. Poeton submits that the emphasised words in this passage are critical, because the "conditions as described above" were as set out at paragraph 2.3 of the Joint Statement, where the Claimant's witnesses referred to a "rogue" tank, i.e. one without effective means of extracting fumes such as would immediately have been subject to an enforcement notice from HSE if encountered by one of their inspectors. Poeton rightly submits that the judge did not accept that its degreasing tank was "rogue" in this sense. Accordingly it submits that the premise for paragraph 2.5 of the joint statement is absent and the judge should not have relied upon it.

77. What the judge said, at [49] was:

> "What the expert evidence does show is that without effective lip extraction concentrations for TCE exposure to an operative can become high very quickly. The experts agree at paragraph 2.5 of the joint statement TB 404, that it was likely that the short-term limit value of 150ppm or 802mg/m3 over 10 minutes would have been exceeded and that the long-term limit would also have been exceeded on some days. (100ppm as an 8-hour time weighted average.) Whilst not a direct correlation these figures are in my judgment important in looking at what exposure might have been like if lip extraction was circumvented by "hoiking" out or similar. When this evidence is combined with the documented recognition at TB 1093 that excessive drag out causes high exposure and the fact that Dr Phillips' view was that reported dizziness (as was complained of) might suggest exposure in excess of 100ppm allows the court to conclude that when the lip extraction was circumvented then the Claimant was likely exposed to levels in excess of the short term limit value when the extraction was bypassed. I have already concluded that such occurred regularly."

78. On a fair reading of this passage, it is plain that the judge appreciated that there was no "direct correlation" between the situation if there was a rogue tank with no effective lip extraction of fumes and the situation that prevailed on "hoiking out". Instead, he was using paragraph 2.5 of the joint statement to provide indicative evidence of "what exposure might have been like if lip extraction was circumvented by "hoiking" out or similar". He also took into account (as he was entitled to do) the evidence that excessive drag out (by which is meant as a result of "hoiking out") causes "high exposure" and that reported dizziness (the evidence of which he clearly accepted) might suggest exposure in excess of 100 ppm, two thirds of the short-term limit. The reference to TB1093 is, however, instructive in explaining the reference to excessive drag out causing "high exposure". It is a reference to a paper by Shipman and Whim (Occupational Exposure to Trichloroethylene in Metal Cleaning Processes and to Tetrachloroethylene in the Drycleaning Industry in the UK, Annals of the Annual Conference of the British Occupational Hygiene Society 1979). The authors took 212 measurements over 8-hour work periods from 32 installations. "[T]he results indicate[d] clearly that the exposure levels are well below the [Threshold Limit Value]. Only in one case did the level exceed 100 ppm and this was caused by too rapid removal of work from the plant. Most of the measurements were below 50 ppm (97%) and 91% were below 30 ppm. … The practices which lead to high exposures … are well known: (1) incorrect siting of plant; (2) excessive drag out due to incorrect operation; (3) inadequate plant maintenance; (4) overloading of equipment and incorrect jigging of work leading to solvent trapping." Given these "multiple practices" that lead to "high exposures" and the fact that only one reading out of 212 exceeded 100 ppm, it is plain that the authors must have included readings below 100 ppm within the phrase "high exposures". They were not, and cannot have been, saying that excessive drag out or too rapid removal of work from plant routinely led to readings in excess of 100 ppm.

79. Once again, the judge's conclusion is expressed in very general terms, namely that Mr Holmes was likely to have been exposed to levels in excess of the short-term limit value when the lip extraction was circumvented/bypassed, which occurred "regularly".

Given the limited scope of the evidence upon which he relied, the lack of precision in his findings was necessary: it is not of itself a ground for criticism.

80. The judge was clearly right to accept that there was no direct correlation between the exposure that would have occurred with a "rogue" tank that had no effective lip extraction (so that there was no impediment to the escape of TCE vapour from the tank throughout its operation) and on "hoiking" out components without their having shed TCE while being suspended in the freeboard zone. Figures were available in the evidence for the levels of likely exposure in certain circumstances: see the references to Shipman and Whim in paragraph 2.5 of the joint statement and [49] of the judgment. What is lacking in the judgment is any quantitative analysis of the exposure that would occur if a component were "hoiked" and either rested on the rim or put on the floor. In the absence of any such analysis the use by analogy of figures relating to a "rogue" tank, including the reference to a peak concentration of 300 ppm, seems tenuous: not irrelevant but of limited value in determining whether and, if so, to what extent the safe limits were breached.

81. In addition, the reference to dizziness being indicative of a level that would be ⅔ of the short-term limit provides little support for a finding that the short-term limit was exceeded when components were "hoiked" out. On behalf of Mr Holmes it is submitted that an additional problem because of the absence of a hoist was that it caused operatives to lean over the open tank when placing components in the tank and subsequently removing them. No analysis along these lines appears in the judgment where the emphasis is on the exposure caused by proximity to components that have been removed from the tank without a sufficiently long period in the freeboard zone, which is different.

82. Although I agree that the evidence upon which the judge relied was tenuous, I would not go so far as to say that the judge was not entitled to conclude that the short-term limit would be exceeded on occasions. However, this highlights the understandable vagueness of the judge's finding, which went no further than to say that Mr Holmes was exposed to levels "in excess of the short-term limit value" without giving any indication of the extent to which the short-term limit value would have been exceeded.

83. The third area of criticism levelled by Poeton in relation to the use of the degreasing tank concerns the practice of replenishing the tank by pouring TCE from buckets. Until 2005 the use of buckets for this purpose was accepted practice provided that liquid should not be added to hot sump liquor at or near its boiling point (87 deg C) because of the risk of sudden and violent vaporisation. The evidence of Dr Phillips (the Occupational Hygiene expert instructed on behalf of Poeton) was that, although filling from buckets might appear crude, it was unlikely that such a brief event would materially have increased the daily exposure levels. The judge found it to be unlikely that buckets would be used to add new liquid to liquid already at the boiling temperature. He made no finding that the use of buckets made any material difference to exposure levels or led to safe limits being exceeded.

84. Taking these criticisms together, I would accept that the judge was entitled to find that the process of "hoiking" out led to levels of exposure that were higher than would have happened if there had been a hoist. However, the materials relied upon by the judge can only justify the most generalised conclusions. That may explain why the judge did not attempt any more detailed assessment of the extent of any tortious exposure but it

significantly weakens the evidential base that was available to the judge when he went on to consider the impact of his findings on the difficult issues of causation to which this case gave rise.

Cold degreasing

85. At [50] of the judgment the judge said:

> "… accepting the level of inaccuracy that inevitably arises in estimating the likely exposure from cold degreasing, it is again to be noted that the experts agree that such activities are capable of producing very high exposures indeed. The wide range of 500 to 2000 ppm appears at paragraph 4.11 of their report. Even taking the lowest level of the estimate the short term and long-term value limits are likely to be easily exceeded."

86. What the experts said in their report, after referring to the potential range of exposures was:

> "Such concentrations would have exceeded the appropriate short-term occupational exposure limit for TCE and may have resulted in the long-term (8-hr) limit being exceeded if the work lasted for more than about an hour or other work involving TCE was carried out in the workroom. We also agree that at times, concentrations immediately dangerous to life may have developed."

87. Poeton raises two criticisms. First, the judge made no finding that cold degreasing work lasted for more than an hour or about other work being carried out in the vicinity. The conditions for a finding that the long-term value limit was likely to have been easily exceeded were not satisfied. Second, the judge made no finding about whether and to what extent cold degreasing caused an increase in tortious exposure other than his finding that the limits were likely to be "easily exceeded".

88. On behalf of Mr Holmes it is accepted that the experts' opinion that the long term limit may have been exceeded was contingent on the work lasting for more than about an hour. In that regard, Mr Holmes said in a witness statement that between 1982 and 1997 he spent between 10 and 60 minutes cold degreasing, with an average of 35 minutes, per week. The contingency was therefore not satisfied and the judgment does not provide a basis for the finding that the long-term limit was exceeded. The judge's finding that the short-term limit "was likely to be easily exceeded" remains.

Tank cleaning

89. The judge set out his conclusions on the level of exposure from tank cleaning in [51] of the judgment:

> "The experts agree that the task, if undertaken, had the potential to create very high exposure levels indeed. (TB 409 paragraph 3.1). There is no doubt that the experts identify this as a task that would have required breathing apparatus if it was to be

> undertaken. Levels of exposure would have been significantly in excess of the short-term limit value. (6,600 mg/m^3 as a peak averaged at 3,300 mg/m^3 over a two-hour period.)"

90. "TB 409 paragraph 3.1" is a reference to paragraph 3.1 of the Occupational Hygienists' joint statement. The levels quoted at the end of the paragraph were Professor Cherrie's estimate of what Mr Holmes was likely to be exposed to if he entered the tank to clean it. They approximately equate to 1,200 ppm and 600 ppm respectively. Dr Phillips considered that Professor Cherrie's estimates were realistic and that they could have been even higher in a previously undisturbed setting. The experts agreed that such exposures would be in breach of Poeton's legal obligations and would have presented a risk of accidental death. The risk was of a sudden loss of consciousness. It is not suggested that the sudden loss of consciousness in this extreme situation would be attributable to a mechanism involving damage to the dopaminergic neurons.

91. Poeton submitted to the judge that the operation would not take very long and that the highest risk was when an operative was in the tank and bent over, which was said to be for a short period only. The Judge held that the entire operation of cleaning the tank took place approximately monthly and that the time taken for the task was "up to about 2 hours". No breathing apparatus was provided. The judge found that, although it may have been possible to carry out some of the work from an external access point people did get inside the tank to clean it. He accepted Mr Holmes' evidence and held that cleaning was by way of an operative getting inside the tank. He accepted that not all of the 2 hours would be within the tank but that a "substantial" period would have been; and that, although the operative would not have been bent double within the tank for the entire period he would have had to have either crouched or bent periodically to achieve the cleaning task. It appears that, as Poeton now submits, Mr Holmes would not always have been the person who carried out the tank cleaning: the inference appears to be that he will have cleaned it less than about 6 times a year but with some degree of regularity.

92. On the information that was available to him, the judge was fully entitled to reach these conclusions. There can be no doubt that Poeton's breaches of duty in relation to cleaning the tank led to operatives who carried out the cleaning being exposed to very high levels of TCE that would have far exceeded the short-term limits, as the judge held.

93. In my judgment, the criticisms made by Poeton do not justify this court in reversing his conclusion that "the Claimant was regularly exposed to levels of exposure significantly in excess of the short-term limit value and for a significant number of days a year in excess of the long-term value." But this is because the extreme generality of the conclusion provides little or no real insight into the extent to which Mr Holmes was tortiously exposed to TCE. This is not necessarily a criticism of the judge, because, at least to some extent, this lack of specificity may have been because of deficiencies and lack of precision in the evidence, but it is a significant feature when one comes to consider issues of causation, to which I now turn.

Issue 3: generic causation

94. Legal issues of causation have to be decided on the basis of current knowledge: see Lord Rodger at [142] of *Sienkiewicz*; and I bear in mind at all times that proof of causation for the purposes of legal liability is different from "scientific proof".

95. The causation of Parkinson's disease is poorly understood. It was common ground, as the judge recorded, that it "probably involves the loss or damage of dopaminergic neurons in the brain. Professor Edwards (the expert on neurology instructed on behalf of Mr Holmes) went on to describe how this [damage to dopaminergic neurons] can set in train a cascade of degeneration of nerve cells resulting when a critical point is reached (70%) in the development of the condition." The generic causation question is therefore whether exposure to TCE can cause (or materially contribute to the causing of) Parkinson's disease, the mechanism of interest being the destruction of the patient's dopaminergic neurons. The fact that it may be toxic in other ways or by other mechanisms is not informative.

96. The judge accepted that the likelihood is that those who develop Parkinson's disease have a genetic susceptibility but that susceptibility does not make it inevitable that the condition will develop. Furthermore, he said that "the mainstream hypothesis amongst neurologists is that there is a complex interplay between genetic and environmental factors and that both factors work together to produce the condition." What the neurologists agreed in their joint statement was that, because Mr Holmes first developed symptoms of a tremor aged 46 and was diagnosed with Parkinson's disease at 48, they would classify him as a "young onset" of Parkinson's disease: "We agree that genetic causes of Parkinson disease are most often found in the young onset group. We note that Mr Holmes has not had genetic testing. We agree that current genetic analysis for Parkinson disease will probably only identify a minority of causative mutations as the majority likely remain to be discovered." The evidence of Professor Schapira (the expert on neurology instructed on behalf of Poeton) was that the only unequivocal causes of Parkinson's disease identified to date are genetic mutations. Overall these account for approximately 10-15% of Parkinson's disease with the remainder being "idiopathic".

97. Although the Judge was fully entitled to accept that the majority of cases of Parkinson's disease were multi-factorial, he was correct to observe in [83] of the judgment (which I have set out above) that "of course multifactorial factors might involve multiple genetic factors only, and Mr Holmes would have been exposed to many environmental factors."

98. TCE is one of many compounds that have been suggested to be capable of causing Parkinson's disease. This may be illustrated by reference to Ascherio and Schwarzschild (The epidemiology of Parkinson's disease: risk factors and prevention. Lancet Neural. 2016; 15: 1257-1265), which identified numerous risk factors at the end of which the authors said "Finally, there is growing interest, but no longitudinal data, in the potential role of solvents (eg [TCE]) as an adverse risk factor and the gut microbiome as a modulator of Parkinson's disease risk."

99. There is no doubt that TCE is neurotoxic. There is also no doubt that it operates in different ways and with different results. The evidence of Professor McElvenny (the epidemiologist instructed on behalf of Mr Holmes) was that:

> "TCE is a mild skin, eye and respiratory tract irritant. TCE is thought to depress the central nervous system via a solvent effect on lipids and protein compounds of neural membranes. Typical symptoms of exposure to lower levels of TCE (>500 ppm) include excitation, lightheadedness, headache, nausea, incoordination, and impaired ability to concentrate. At higher doses (>100 ppm) lack of muscle tone, decreased deep-tendon reflexes, drowsiness, dizziness, impaired gait and stupor may develop. In extreme cases death may result from respiratory depression. In a few cases, TCE has been associated with peripheral and cranial nerve damage. … TCE is also responsible for effects on other parts of the human body inducing cardiovascular, respiratory, hepatic, renal, dermal and ocular effects. Some survivors of ingestion or severe inhalation have experienced chronic nerve disorders, including blindness due to inflammation of nerves of the eyes."

He might have added that TCE is now recognised as a carcinogen. While this does not purport to be a comprehensive description of the workings and effects of exposure to TCE, it does not provide a justification for assuming that, because TCE is neuro-toxic, it is capable of affecting the dopaminergic neurons at the dose levels to which Mr Holmes was exposed.

100. The evidence as summarised by the judge in support of the hypothesis that exposure to TCE can cause (or materially contribute to the causing of) Parkinson's disease, is weak.

101. Professor McElvenny and Professor Jones (the epidemiologist instructed on behalf of Poeton) included in their joint statement:

> "2.1 We agree that causal associations in epidemiology require a consistent body of epidemiological evidence across a number of studies. In our view, the epidemiological evidence for solvents in general and PD, for chlorinated solvents and PD and for TCE and PD all far short of that required to deduce causal associations."

102. The high point of the epidemiological evidence for Mr Holmes' case is the meta-analysis carried out by Pezzoli and Cereda (Exposure to pesticides or solvents and risk of Parkinson's disease. Neurology. 2013 80(22) 2035-2041). The objective was to investigate the risk of Parkinson's disease associated with exposure to pesticides and solvents using meta-analysis of data from cohort and case-control studies. A total of 104 studies were included in the meta-analysis. Solvents were treated as a class: TCE was not separately identified or analysed. The outcome results from cohort studies included two that collected data on exposure to solvents and both reported no association. The available case-control studies were of variable quality. Selection of case-control studies of higher quality resulted in an elevated risk of Parkinson's disease for exposure to solvents. The adjusted risk for solvents, which has become the headline figure to which most reference has been made, was 1.58 which, it is common ground, falls well short of evidencing a causal association. The discussion section of the paper concluded that "the literature supports the hypothesis that exposure to pesticides or

solvents is a risk factor for PD. However, further prospective and high-quality case-control studies are required to substantiate a cause-effect relationship."

103. It is common ground that, to date, such high-quality case-control studies have not substantiated the cause-effect relationship. It is also common ground that results from the studies that have been carried out have been inconsistent. Professor McElvenny had relied upon what is known as the Geoparkinson Study (Dick, De Palma et al. Environmental risk factors for Parkinson's disease and parkinsonism the Geoparkinson Study Occup Environ Med 2007, 666, 673) but subsequently accepted that he had been wrong to say that the study did not examine organic solvents. It did. As recorded in the Joint Statement, "the study addressed lifetime occupational history and hobby exposure to solvents and provides an adjusted odds ratio for an exposure to solvents of 1.01 (95% CL: 0.84-1.23), for low exposure to solvents of 1.17 (0.92 to 1.50), and for high exposure 0.88 (0.69 to 1.12). This study does not provide evidence in support of solvent being a cause of PD and parkinsonism." The Geoparkinson study was not included in the meta-analysis of Pezzoli and Cereda.

104. Professor Jones identified the study by Van der Mark et al (Occupational exposure to solvents, metals and welding fumes and risk of Parkinson's disease. Parkinsonism and Related Disorders 21 2015). The study is a case-control study involving 444 cases of Parkinson's disease identified between 2010 and 2012 at hospitals in the Netherlands. No evidence was found for an association between exposure to chlorinated solvents and Parkinson's disease.

105. As recorded by the judge at [75] of the judgment, neither of the expert epidemiologists thought that application of the Bradford Hill criteria provided an answer. The judge concluded that strict application of the criteria to the facts of the present case "does not really take matters very much further." Referring to "the deficit in clear research" he considered it arguable that the strongest arguments for Mr Holmes were temporality, biological gradient and plausibility. He did not explain his references to temporality or biological gradient other than to say "Some of the studies seem to suggest a biological gradient and there is a temporal relationship in that, to borrow a phrase from Sir Austin [Bradford Hill] the cart is not put before the horse." We have not been taken to any evidence that suggests temporality or biological gradient in relation to damage to dopaminergic neurons rather than to other acknowledged effects of TCE exposure. As for plausibility, the judge said that it was agreed that a plausible mechanism could be envisaged.

106. A central element of the judge's reasoning was that TCE is neurotoxic and has been shown to damage dopaminergic cells in animals. He cited two papers which he said demonstrated "a plausible neurological pathway or mechanism whereby TCE exposure specifically could act in a way that would materially contribute to the development of Parkinson's disease." In the course of doing so he noted that such animal studies must be approached with care because the scientists concerned are trying to replicate in weeks or months the sort of exposures that might occur occupationally over the many years of a working life, with the possibility that the results were skewed by "comparatively (very) high doses".

107. The first study to which he referred was Gash et al (Trichloroethylene: Parkinsonism and Complex 1 Mitochondrial Neurotoxicity 2008; 63(2): 184-192). The judge cited the paper's conclusion that Neurotoxic actions of TCE were demonstrated in animal

studies showing that oral administration of TCE for 6 weeks instigated loss of dopamine neurons. The paper recorded (but the judgment did not) that peak TCE blood levels in rats may have been at least 35 times greater than those in typical industrial workers. The paper did not report behavioural alterations in the rats.

108. The second study cited by the judge was Keane et al (Trichloroethylene and its metabolite TaClo lead to degeneration of substantia nigra dopaminergic neurones: Effects in wild type and human A30P mutant α-synuclein mice Neuroscience Letters 711. 2019 134437). The judge expressly recognised that the exposures to TCE in the study had not led to acceleration of motor or cognitive deficits but cited the conclusion of the paper, including that "exposure to TCE … can cause DA neuronal cell death in the SNpc in vivo, suggesting TCE exposure as a possible contributory factor in development of PD."

109. The judge was also referred to a study by Liu et al (Trichloroethylene induces dopaminergic neurodegeneration in Fisher 344 rats J Neurochem 2010; 112(3): 773) where the administration of TCE in high doses induced a significant loss of dopaminergic neurons in the substantia nigra pars compacta in a dose dependent manner.

110. In the light of these studies the judge was entitled to conclude that a plausible neurological pathway had been established; and Professor Schapira was correct to concede that a causative link between TCE and PD could not be disproved so that, in that sense, it was *possible* that TCE was a cause of the condition. But the evidence had significant limitations.

111. Professor Edwards accepted that animal studies of this sort "are always complex to interpret in medical practice, typically because they do short exposure typically at very very high doses and that it is extremely hard to mimic the nature of chronic lower levels of exposure in human beings." Consequently what he was prepared to take from the studies was that there is "an interaction between TCE and the relevant processes in the brain and, in particular, relevant processes in dopaminergic neurons." He considered this to be "interesting information that adds weight to a potential mechanism, and that's as far as I take … that interpretation." He also accepted that one explanation could be that a different mechanism was in play and that what could be happening was that the rats were being overwhelmed by the high doses that were being administered. The very high dosing of the rodents may also be relevant because there may be a threshold below which damage is not caused to the dopaminergic neurons. He made clear in his oral evidence that he was not proposing "a sort of single high level very hyper sort of toxic exposure which immediately causes cell death for example". Rather, he envisaged "something where chronic exposure to a … stressor, a toxic stressor causes … potentially cumulative damage over time or just pushes the system into a state where it's more likely to trigger into a state of progressive degeneration."

112. I have summarised the propositions on which the judge based his finding of (individual) causation at [24] above and the main features of the scientific evidence on which he relied. In my judgment, the propositions and evidence on which he relied did no more than to establish that TCE was a risk factor for Parkinson's disease and that there is a plausible mechanism based on the rodent studies for a finding that TCE may cause or materially contribute to the development of Parkinson's disease. Although TCE has long been identified as a compound of interest, the evidence to prove generic causation

is lacking whether one is applying the legal or a scientific standard of proof. To my mind the critical gap, given the absence of epidemiological evidence supporting a causative link, is the present inability to extrapolate from the rodent studies to the impact of TCE exposure in humans, not least because of the great disparity in relative exposure levels.

113. I would therefore uphold Poeton's submissions and challenge on Issue 3 on the ground that the evidence before the judge did not justify a finding of generic causation.

Issue 4: individual causation

114. If I am right about the issue of generic causation, it would follow that Mr Holmes cannot prove that the tortious exposure to TCE to which he was subjected caused or materially contributed to his developing Parkinson's disease unless, possibly, there were features of his case that are not reflected in the generic evidence that compel a finding of causation, such as a relevant and repeated response to challenge and rechallenge by exposure to TCE. Such features are absent in this case. Even if I am wrong, there are other difficulties in Mr Holmes' way to proving individual causation.

115. The first difficulty derives from the judge's (correct) acceptance that Mr Holmes would have been exposed to many environmental factors. Attention has concentrated on TCE because of the known history of exposure and Mr Holmes' ability to prove breaches of duty on the part of Poeton so that he can point to tortious exposure. Nothing is known about any other environmental factor in his case and whether or to what extent such other environmental factors increased the risk of him developing Parkinson's disease. A further difficulty is that it is *tortious* exposure which must be shown to have played a relevant causative role. Proof of tortious exposure does not prove that the tortious exposure has caused or materially contributed to damage: see, for example, *Wilsher* where the defendant's lack of care was proved but was not shown to be causative. Furthermore, although it is recognised that our knowledge of genetic factors affecting the development of Parkinson's disease is incomplete, such that genetic testing may provide false negative returns, and that a majority of cases of Parkinson's disease implicate both genetic and environmental factors, numerous genetic risk factors have been identified but Mr Holmes has not been tested.

116. I have commented already on the vagueness of the findings about the level of exposure in Mr Holmes' case and that this may well be a function of the limitations of the evidence that was available to the judge. That said, no evidence was identified by the judge (and no evidence has been drawn to our attention) to substantiate a causative link between the tortious exposure and damage to Mr Holmes' dopaminergic neurons. The difficulty this creates comes into sharp focus when one returns to the question: have Poeton's breaches of duty caused *or materially contributed to* the development of Mr Holmes' disease? Take a simplified model and assume that an individual who has developed Parkinson's disease had 100 units of dopaminergic neurons and that the destruction of 70 of those units has caused his disease. Assume that exposure to TCE has damaged or destroyed 35 units: it would not be difficult for a court to conclude that the exposure to TCE materially contributed to the development of the disease. Assume exposure to TCE has damaged or destroyed 1 unit: it is not obvious that the same answer would be given. Yet in the present case there is nothing to indicate even at a most general level whether Poeton's tortious exposure has damaged or destroyed 0 units, or 70 units or some number of units in between.

117. Nor can this absence of evidence be remedied by appealing to "the reality of the situation" or "what seems to be the likely reality". Tempting though it may be to do so, it is not permissible for the court to speculate in the absence of sufficient evidence that Poeton's tortious exposure of Mr Holmes to TCE has caused or materially contributed to his infinitely regrettable disease.

118. I have not lost sight of the fact that Professor Edwards maintained his opinion that Mr Holmes' disease was caused or materially contributed to by his exposure to TCE. However, being confronted by opposing views from distinguished experts, the judge manfully identified and analysed the features that he considered to be relevant to the questions of proof that he had to decide. For the reasons I have given, I consider that he reached the wrong conclusion in this difficult case.

119. I would uphold Poeton's submissions on Issue 4 and allow the appeal on the basis that, although it was established that exposure to TCE is a risk factor for the development of Parkinson's disease, the judge's finding that tortious exposure to TCE caused or materially contributed to Mr Holmes' disease was not sustainable on the evidence and was wrong.

120. Perhaps as something of a fall-back position, those representing Mr Holmes submitted that there might in some circumstances be a rational or logical way in which responsibility might be allocated even where the injury or disease is of a type that is generally regarded as "indivisible". We were referred to dicta in *Rahman v Arearose Ltd* [2001] QB 351 at [19] per Laws LJ, *Hatton v Sutherland* [2002] ICR 613 at [36]-[42] per Hale LJ, *Dickins v O2 plc* [2009] IRLR 58 at [45]-[47] per Smith LJ and *BAE Systems (Operations) Ltd v Konczak* [2018] ICR 1 at [65]-[72] per Underhill LJ. While, in my respectful opinion, those dicta raise questions that might be important (and difficult) in another case, no question of apportioning liability arises in this case since no causative contribution has been shown. For similar reasons, although the prospect was raised by the Court during the hearing, this is not a case which involves oversubscribed causes. These important and difficult questions should therefore be left alone until a case in which they actually arise.

Issue 5: Wilsher

121. In these circumstances, if my lords agree with my conclusions on issue 4, it is not necessary to decide whether this is a *Wilsher* case and I do not do so. My reluctance is based on the lack of necessity and because I consider that the present understanding of Parkinson's disease makes a decision on this issue particularly difficult and probably unreliable. Even if it is accepted that genetics and both internal and external environmental factors may all be relevant to the causation of Parkinson's disease, it is not clear on current understanding to what extent they may combine or, alternatively, may be discrete potential causes that could bring the case within the ambit of the principle established by *Wilsher*.

Lord Justice Phillips

122. I agree.

Lord Justice Underhill

123. I agree with Stuart-Smith LJ that this appeal should be allowed, for the reasons that he gives. That is, I do not believe that it was open to the Judge on the evidence before him to conclude that such exposure as the Claimant had experienced to TCE made a material contribution to his developing Parkinson's disease. I agree with Stuart-Smith LJ that the evidence about a possible causative link between TCE exposure and Parkinson's disease amounts to no more than the identification of a plausible mechanism and that that by itself is not enough. Further and in any event, even if, contrary to our view, it were established that TCE exposure could contribute to the causation of Parkinson's disease, I agree that, given the range of other possible genetic and environmental causes, including non-tortious exposure to TCE, the evidence does not justify a conclusion that on the balance of probabilities such wrongful exposure as the Claimant experienced in this case made a material contribution to his developing the disease.

124. I also agree with Stuart-Smith LJ's conclusion on Issue 1 of the appeal. His comprehensive analysis of the authorities shows conclusively that in the case of an indivisible injury a tortfeasor who makes a material contribution to the injury is liable for the whole. I respectfully agree with him that it is clear from the passages which he quotes at paras. 42-45 above that that is the *ratio* of *Bonnington*, notwithstanding that pneumoconiosis is a disease whose severity relates to the extent of exposure. And even if there were room for doubt, the subsequent case-law, as he shows, authoritatively establish that that is to be treated as the ratio. The point is put succinctly by Lord Toulson at para. 32 of his judgment in *Williams*:

> "Lord Reid interpreted the medical evidence as meaning that the particles from the swing grinders were a cause of the entire disease. True, they were only part of the cause, but they were a partial cause of the entire injury, as distinct from being a cause of only part of the injury."

125. I would only add one footnote on Issue 1. Stuart-Smith LJ refers at para. 63 of his judgment to Professor Stapleton's criticisms of the reasoning (though not the outcome) in *Bailey*. Her preferred analysis of cases of this kind appears now to have been approved by the Supreme Court: see paras. 182-185 of the judgment of Lord Leggatt and Lord Hamblen in *Financial Conduct Authority v Arch Insurance (UK) Ltd* [2021] UKSC 1, [2021] AC 649, and her recent article "Unnecessary and Insufficient Factual Causes", *Journal of Tort Law* 2023.