Using epidemiological evidence in tort law: a practical guide

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Introduction

Epidemiology is the study of disease patterns in populations which seeks to identify and understand causes of disease. By using this data to predict how and when diseases are likely to arise, it aims to prevent the disease and its consequences through public health regulation or the development of medication. But can this data also be used to tell us something about an event that has *already occurred?* To illustrate, consider a case where a claimant is exposed to a substance due to the defendant's negligence and subsequently contracts a disease. There is not enough evidence available to satisfy the traditional 'but-for' test of causation.[1] However, an epidemiological study shows that there is a potential causal link between the substance and the disease. What is, or what should be, the significance of this study?

In *Sienkiewicz v Greif* members of the Supreme Court were sceptical that epidemiological evidence can have a useful role to play in determining questions of causation.[2] The issue in that case was a narrow one that did not present the full range of concerns raised by epidemiological evidence, but the court's general attitude was not unusual. Few English courts have thought seriously about the ways in which epidemiological evidence should be approached or interpreted; in particular, the factors which should be taken into account when applying the 'balance of probability' test, or the weight which should be attached to such factors.

As a result, this article aims to explore this question from a practical perspective. A clear test for causation in cases involving scientific uncertainty is suggested, along with an explanation as to why the test should be so applied. Although the question 'why' necessarily involves a more theoretical analysis, it is vital for understanding some of the policy decisions that are proposed. However, in order to avoid too abstract and philosophical a discussion, an attempt is made to draw the reader's attention throughout the article towards the ultimate aim of tort law: 'to define cases in which the law may justly hold one party liable to compensate another'.[3]

The article proceeds as follows. Part I briefly outlines the nature of epidemiological evidence in order to highlight the limits of using such data in law. This will lay the foundations for the subsequent discussion in Part II, in which a test for causation in cases

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1   The traditional test is laid out in *Barnett v Chelsea & Kensington Hospital Management Committee* [1969] 1 QB 428 (QB); the defendant has caused the injury if, on the balance of probabilities, the claimant's injury would not have occurred but for the defendant's wrongdoing.

2   [2011] UKSC 10, [2011] 2 AC 229 [96], [98] (Lord Phillips), [170] (Baroness Hale), [174], [186]–[187] (Lord Brown), [190] (Lord Mance), [205]–[206] (Lord Kerr).

3   *Fairchild v Glenhaven Funeral Services Ltd* [2002] UKHL 22, [2003] 1 AC 32 [8] (Lord Bingham).

that require epidemiological evidence is proposed. This test consists of the following two steps: first, the claimant must prove that there is a causal link between exposure to a substance and the alleged injury (*general causation*). Next, the claimant must prove that the exposure to the substance caused the injury *in that specific case* (*specific causation*). The legal standard of proof at each stage is the balance of probability and causation is conditional upon both stages being passed. The requirements for general and specific causation will be thoroughly examined in Parts III and IV respectively.

Furthermore, throughout the article it will be highlighted that it is crucial to identify the *question* that is being asked at the general and specific causation stages and to observe whether this lines up with the question that epidemiological evidence seeks to answer. If the questions match, there are very few problems with using epidemiological data to answer that causal query. But where the questions differ, using epidemiological evidence becomes more problematic. The significance of the 'relative risk' and 'doubles the risk' test at both stages will also be clarified, as this appears to have caused much confusion among academics and practitioners.

It is important to mention that this test is not novel; it was applied, for example, in the English case *Loveday v Renton*, in which the High Court investigated whether the administration of the pertussis (whooping cough) vaccine had caused the claimant's permanent brain damage.[4] Stuart-Smith LJ remarked, '[This] case raises the issue of causation, both general and specific, namely, whether the vaccine can cause brain damage and, if so, whether it did in the individual Plaintiff's case.'[5] As the court held that the evidence did not satisfy the legal standard of proof at the general causation stage, namely, it could not be said on the balance of probability that the vaccine can cause brain damage, the issue of specific causation did not have to be considered. Outside of England, the two-stage test is also widely recognised and applied in many American states.[6]

Although the main focus of this article is the English state of the law, other jurisdictions will nevertheless occasionally be referred to in order to shed some light on this issue.

I. Nature and limits of epidemiological studies

Ken Rothman, a renowned epidemiologist, frankly admitted in an interview for the prestigious journal Science that 'we're pushing the edge of what can be done with epidemiology'.[7] While this is arguably sensationalist writing, an awareness of the nature and limits of epidemiological evidence is crucial for understanding the extent to which it can be useful for the causation query. As a result, the problems that both the nature and the compilation of epidemiological evidence pose will be briefly outlined.

Experiments that provide the 'strongest inference of causation' are those that involve direct and rigorous control of variables.[8] The 'gold standard' is the randomised control

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4   [1990] 1 Med LR 117 (QB).
5   *Ibid*.
6   See MD Green, DM Freedman and L Gordis, ' Reference Guide to Epidemiology' in Reference Manual on Scientific Evidence, 382 (Federal Judicial Centre, 2nd ed, 2000) <http://www.fjc.gov/public/pdf.nsf/lookup/sciman06.pdf/$file/sciman06.pdf> accessed 28 August 2014.
7   G Taubes, 'Epidemiology Faces Its Limits' (1995) 269 *Science Journal* 164, 164.
8   M Rutter, 'Identifying the Environmental Causes of Disease: How Should we Decide What to Believe and When to Take Action?' (2007) Working Group Report by The Academy of Medical Sciences, 27 <*www.acmedsci.ac.uk/download.php?i=13576&f=file*> accessed 27 August 2014.

trial ('RCT'), which typically combines such control with the 'random assignment to the experimental interventions or to [the] control condition'.[9] Furthermore, both researchers and recipients are 'kept "blind" to whether they are receiving the experimental or control intervention'.[10] Together, these features effectively eliminate confounders; namely, 'any [features] other than the hypothesised disease-causing influence that might artefactually give rise to the supposedly causal associations'.[11]

However, where we wish to study the effects of certain variables on disease (eg to see whether smoking causes lung cancer), there are clearly 'manifold pragmatic and ethical obstacles' to conducting an RCT.[12] We must therefore rely on various 'observational studies'. For example, a 'cohort study' involves taking a large sample of the population and examining it after certain intervals to investigate disease outcomes.[13] A 'retrospective study', on the other hand, involves collecting a group of people who have contracted a disease ('cases') and analytically comparing them with a group of people who have not contracted the same disease ('controls').[14]

The obvious disadvantage of these studies is that very few factors can be controlled, which can lead to confounding. For instance, it is shown that poverty is linked to a number of different diseases;[15] but to what extent is poverty simply an indirect association, such that it leads to a set of conditions which are actually those attributable to the higher risks of certain illnesses? Although Smith shows that confounding can be minimised through 'statistical "adjustment" or "control"' he adds that this process is limited by missing or incorrectly measured variables;[16] thus confounding poses serious challenges to disease epidemiology.

Observational studies are also prone to systematic error (or bias),[17] which occurs when the selection of the sample has been tainted in some way, leading to results that are atypical of the general population. This can be a very difficult issue to overcome, because even where the bias is identified, it is often impossible to ascertain its degree.[18] Nevertheless, the risk of systematic error can be lowered by 'constructive replication', which involves repeating the study with an attempt to remove some of the weaknesses in previous studies.[19] Where the same association is found in multiple studies, it can be said with greater confidence that this is not a result of the bias.[20]

Finally, epidemiological evidence is susceptible to random error.[21] This can be reduced by increasing the sample size or through 'estimation' and 'significance testing'. Where an estimate is described as an interval (eg between 20–30% of women suffer migraines), estimation testing provides a 'confidence interval' which signifies the

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9   *Ibid* 27.
10   *Ibid* 27.
11   *Ibid* 40.
12   AP Dawid, 'The Role of Scientific and Statistical Evidence in Assessing Causality' in R Goldberg (ed), *Perspectives on Causation* (Hart Publishing 2011) 137.
13   Rutter (n 8) 29.
14   Dawid (n 12) 137.
15   Rutter (n 8) 35.
16   GD Smith, 'Reflections on the Limitations to Epidemiology' (2001) 54 Journal of Clinical Epidemiology 325, 327.
17   P Feldschreiber, L Mulcahy and S Day, 'Biostatistics and Causation in Medicinal Product Liability Suits' in R Goldberg (ed), *Perspectives on Causation* (Hart Publishing 2011) 183.
18   G Taubes (n 7) 167.
19   DT Lykken, 'Statistical Significance in Psychological Research' (1968) 70 Psychological Bulletin 151, 151.
20   Rutter (n 8) 35.
21   Feldschreiber and others (n 17) 183.

estimate's precision.[22] Significance testing, on the other hand, examines the extent to which the association between exposure and disease is attributable to random errors. This is measured by the 'p-value'. Statisticians would reject a study if the p-value was 0.05 or above – in other words, there must be no more than a one-in-twenty chance that the link between variables was due to a random association.[23] Importantly, a p-value below this threshold cannot provide 'unassailable support for a causal relationship…[as] the p-value tells us nothing about possible systematic errors'.[24]

It is clear, in light of the above, that the limits to the methodology of epidemiological studies pose many challenges to the reliability of the data collected. That notwithstanding, it is accepted that a high quality observational study that takes account of confounding, error and bias can still be used to establish a causal association between a substance and a disease.[25] Furthermore, the reader's attention is drawn to Bradford Hill's famous statement: 'all scientific work is incomplete – whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have'.[26]

It is vital to keep these issues in mind throughout the remainder of this article.

II. Suggested test for causation

In cases involving complex issues of causation, courts require the clearest guidance. The following test is therefore proposed, which would bring much needed structure and transparency to this area of law. It would also encourage consistent application of epidemiological evidence, thus ensuring fairness to litigants.

The proposed test involves two stages; causation is conditional upon both being passed. Firstly, the claimant must establish on the balance of probabilities that the substance to which he or she was exposed is *capable of causing* the injury suffered; in other words, that there is a causal link between the injury and exposure to the substance (*general causation*). Once this hurdle is passed, the claimant must prove on the balance of probabilities that the exposure to the substance actually *caused* the injury in *the specific instance* (*specific causation*). During both stages, epidemiological evidence should be weighed together with any 'particularistic evidence'[27] available and a value judgment should be reached. It will be argued that no one form of evidence should take primacy over other forms. This will be discussed in more detail in Part III below.

Although it is arguable that the general causation stage is unnecessary if specific causation can be proved, omitting the first stage risks causing injustice. This is because the wrongful act complained of must be capable of causing the injury before liability can be imposed; logic dictates that if 'substance X' is held not to be capable of causing 'disease Y' on the balance of probabilities, then in a given case it cannot be concluded that 'disease Y' was caused by 'substance X'. Thus the defendant cannot be held liable if the first hurdle is

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22   *Ibid* 184.
23   *Ibid* 184.
24   *Ibid* 184.
25   Rutter (n 8) 85.
26   AB Hill, 'The Environment and Disease: Association or Causation' (1965) 58 Proceedings of the Royal Society of Medicine 295, 30.
27   'Particularistic evidence' is defined as evidence that directly pertains to the specific case at hand, such as the medical records of the claimant taken before and after the injury, or evidence of the level of exposure that the claimant was exposed to.

not passed. This will also ensure that cases in which specific causation is not established do not set precedents regarding general causation. Furthermore, splitting the test in this way will bring much-needed transparency and coherence to this complex area of law and will generally clarify the role of the epidemiological evidence at each stage of the causal query.

However, a different approach is advocated by Barnes and Gold.[28] They argue that the test should be separated into three probabilities:

(a) 'fact probability': 'the likelihood that the defendant's actions led to the adverse outcome', commonly quantified by the 'relative risk';[29]
(b) 'belief probability': the measure of the fact finder's confidence in the evidence in support of the claimant's case;[30] and
(c) 'sampling error probability': the probability that the underlying relationship between variables is owed to a sampling error as opposed to an actual causal link between them.[31]

Barnes and Gold maintain that splitting the test this way prevents the court from 'collapsing' the probabilities, which can lead to errors.[32] McIvor adds that these measures 'are expressed as numerical values and so dealing with them separately reduces the potential for confusion with the other numerical evidence'.[33]

However, it is argued that the fact that this methodology encourages judges to attribute a numerical figure to the different elements makes it very likely that it would actually increase confusion. Firstly, such an approach could lead to insupportable quantifications of probability; as McIvor points out, 'ordinary human beings cannot differentiate between, for example, a 51% degree of belief and a 58% degree of belief'.[34] Secondly, it could lead to complicated findings of 'probabilities of probabilities of occurrence', such as, 'we may… be only forty percent sure (belief probability) that the reduction is twenty percentage points (fact probability)'.[35] Both of these situations are likely to lead to more confusion and distract from the balanced evaluation of the evidence that judges are supposed to undertake. Having supported Barnes' method, McIvor contradictorily highlighted the importance of this as well, concluding that, 'we should dispense with the practice of expressing the balance of probabilities standard in numerical terms'.[36]

A further concern with encouraging the attribution of numerical values to such evaluations is that doing so may lead to the view that the figures expressed can be

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28   DW Barnes, 'Too Many Probabilities: Statistical Evidence of Tort Causation' (2001) 64 LCP 191; S Gold, 'Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence' (1987) 96 Yale LJ 376. Gold's paper was cited favourably in *Sienkiewicz* by Lord Rodger and Lord Dyson; (n 2) [156] (Lord Rodger), [217] (Lord Dyson).
29   Barnes (n 28) 192. See in the following section the definition of relative risk.
30   *Ibid* 192.
31   This is termed the 'p-value' and was defined in Part I.
32   Gold (n 28) 378.
33   C McIvor, 'Debunking Some Judicial Myths About Epidemiology and its Relevance to UK Tort Law' (2013) 21(4) Med Law Rev 553.
34   *Ibid*.
35   Barnes (n 28) 192.
36   McIvor (n 33). Tribe makes a similar, but broader point, arguing that promoting the use of mathematical terms on a general level risks encouraging juries to give undue weight to the statistical evidence (as it is already conveniently quantified), which could likewise distract juries from undertaking the complicated task of evaluating the evidence to reach a fully reasoned conclusion regarding causation: LH Tribe, 'Trial by Mathematics: Precision and Ritual in the Legal Process' (1971) 84 *Harvard Law Review* 1329, 1363.

manipulated according to standard mathematical principles (for example, along Bayesian lines), where the correctness of this approach is far from clear. Indeed, the danger exists that the mere fact of the probabilities being expressed as numbers entices judges to play around with the figures in ways that would not be possible, or even considered, if the probabilities were expressed verbally.

For the reasons highlighted above, it is argued that the test supported by Barnes and Gold should not be used. Instead, the two-stage test, which requires both general and specific causation to be established, is to be preferred.

Before the first stage of the test is considered, it is important to understand some key terms.

'Relative risk' and the 'doubles the risk' test

'Relative risk' ('RR') is a 'purely statistical measure'[37] that indicates a strength of association between two variables and that can be used to make an inference of a causal link between them. It is 'the ratio of the risk of occurrence of a disease among exposed people to that among the unexposed':[38]

$$RR = \frac{\text{Risk among exposed}}{\text{Risk among unexposed}}$$

For example, if epidemiological evidence showed that 20% of female smokers contract lung cancer, but only 2% of female non-smokers contract lung cancer, the RR is: 20/2 = 10.0.

In many jurisdictions, an RR > 2 is deemed necessary to establish causation.[39] This is termed the 'doubles the risk' test ('DTR'). It is a *legal*, not a statistical, creation. As stated by Lord Phillips in *Sienkiewicz*, the test simply follows the reasoning: if exposure to 'substance X' more than doubles my risk of developing 'disease Y', it is more likely than not that the exposure to X has caused Y.[40]

Despite its seemingly logical basis, however, McIvor claims that the DTR test demonstrates both a misunderstanding and misapplication of the RR. While there *are* statements from Lord Phillips (ironically) that suggest that the RR has been misconceived,[41] it is argued that this problem does not stem from its underlying statistical nature, such that this measurement should have no bearing on causation; rather, the confusion either originates from the courts' failure to recognise that the RR plays a different role at the general and specific causation stages, or from a simple failure to examine the two stages separately. Thus, to remedy this misunderstanding, the purpose of the RR at each step of the causal inquiry will be explained. It will be shown in the following Parts that no specific value of RR is needed to establish general causation. Further, where general causation is proved and particularistic evidence is available, an RR > 2 is not necessary to determine

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37 A Broadbent, 'Epidemiological Evidence in Proof of Specific Causation' (2011) 17 Legal Theory 237, 240.
38 *Ibid* 240; R Bonita, R Beaglehole and T Kjellström, *Basic Epidemiology* (2nd ed, World Health Organisation, 2006) 35.
39 *Merrell Dow Pharmaceuticals Inc v Hanver* 953 SW 2d 706 (Tex 1997) 718; *DeLuca v Merrell Dow Pharmaceuticals Inc* 911 F2d 941, (3d Cir 1990) 958–59.
40 *Sienkiewicz* (n 2) [72].
41 *Ibid* [82].

specific causation, as the particularistic evidence may still be strong enough to satisfy the legal standard of proof. However, where there is *only epidemiological evidence*, it will be demonstrated that by using the RR to calculate a lower bound probability of causation, an RR > 2 is sufficient to prove specific causation. Hence, despite its criticism,[42] this article supports the DTR test in the proof of specific causation in the absence of other particularistic evidence. This will be dealt with in more detail below.

The first stage of the proposed test will now be evaluated.

III. Stage one: general causation

Epidemiologists aim to find a causal link between two or more variables. Therefore, the question that epidemiological evidence seeks to answer is: 'is there a causal association between "substance X" and "disease Y"?' Or, simply, '*does* X cause Y?' At the general causation stage, we are asking the same question; namely, 'is X capable of causing Y?' As a result of this congruence, using epidemiological evidence to ascertain general causation is typically not disputed.

Brown suggests, however, that statistical evidence is 'deficient' even at the general causation step.[43] According to Brown, the 'reference class problem'[44] is so fraught with difficulties, that 'the ostensibly objective and rigorous quality of probability assessment is suspect'.[45] As a result, he argues the conductors of trials should be interviewed to validate their findings.

However, Brown fails to acknowledge that epidemiologists thoroughly analyse and account for the reference class issue in any well-researched, expertly conducted study; he therefore indicates a deep lack of appreciation of the art of epidemiology. Furthermore, his contention suggests that epidemiological evidence should be presumed to be unreliable unless it can be validated by its conductor; thus, where it is impracticable to interview the trial's conductor and a claimant's case is dependent on epidemiological evidence, the claimant must automatically lose. Given that epidemiological evidence is more often than not the only means to find a causal link between a substance and a disease, this approach is clearly unjust. Thankfully, Brown's view does not appear to be shared by the English courts, who regularly accept epidemiological evidence in the test for general causation.

How to apply the 'balance of probability' test

As noted above, typically the only data that is available that can satisfy the question of general causation is epidemiological evidence. As a result, this type of evidence can be sufficient, on its own, to pass the balance of probability threshold. However, if there is any particularistic evidence that supports or undermines the finding of general causation, this

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42   McIvor contends that the DTR test 'operates in an altogether unscientific and unsystematic function': McIvor (n 33).

43   R Brown, 'Inferring Cause in Fact and the Search for Legal "Truth"' in R Goldberg (ed), *Perspectives on Causation* (Hart Publishing 2011) 93.

44   The reference class problem, at its most basic, is the problem that arises when the epidemiological study is partitioned into groups according to various factors (age, sex, height, lifestyle etc…) and inferences are made from the data accordingly. This often skews the data, because, for instance, a female could have a different reaction to a substance than a male, or a smoker to a non-smoker.

45   Brown (n 43) 100.

should be considered alongside the epidemiological evidence and a balanced conclusion as to general causation should be reached.

Regarding the way that the epidemiological evidence should be treated, it is proposed that the evidence should be examined on the basis of the Bradford Hill Criteria.[46] As emphasised by Hill himself, however, this should not be a box-ticking exercise; the test should be flexible and should respond to the circumstances of each case.[47] Although this is often acknowledged by the courts, it is not always followed.[48] As part of this balancing exercise, it is vital that the efficacy and soundness of the epidemiological data is weighed. This involves recognising the limits of epidemiological evidence illustrated above and evaluating the extent to which these have been minimised.

Furthermore, it is not necessary for the underlying mechanism between variables to be known (ie the scientific breakdown of *how* X causes Y). Some epidemiologists prefer to see 'a highly plausible biological mechanism' before believing a study,[49] but this is undoubtedly too high a threshold. Firstly, reaching the standard of 'highly plausible' (a standard which in itself would be hotly disputed) is likely to lead to a 'mini-trial' centring on this extremely technical issue, putting a significant strain on parties' time and resources. Secondly, such an approach would inevitably raise the same evidentiary problems as the stringent 'but-for' test, as there are likely to be many instances in which current scientific knowledge is simply unable to achieve this benchmark. This problem has been recognised by many epidemiologists, such as Hill, who argued that 'biological plausibility' should be treated with caution;[50] as history has taught us, an unknown biological mechanism should not mean that the mechanism does not exist.[51] The test should therefore be negative: the alleged causal link between the variables cannot be *wholly contrary* to what is known about biology and the particular injury. It is impractical to form a more accurate standard but arguably this presents a low enough threshold to strike the correct balance between protecting the defendant from biologically unsound theories of causation on the one hand, and not rendering proof of general causation impossible through the back door on the other.

Moreover, it is vital that the courts do not apply the DTR test at this stage. Although a higher RR *does* go some way to indicating a causal relationship,[52] Hill explains that 'strength of association' is only one of the factors to take into account when seeking to prove a causal link between variables.[53] Accordingly, a level of RR < 2 should not automatically disprove general causation. Equally, RR > 2 should not be taken to establish a causal link, as 'relative risk takes no account of absolute risk'.[54] For example, if 1/1,000,000 people

46 Hill (n 26); namely strength of association (measured by the RR), consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment and analogy.
47 *Ibid* 299.
48 See the Scottish case *McTear v Imperial Tobacco Ltd* 2005 2 SC 1; 2005 GWD 20–365, where the trial judge insisted that all the Bradford Hill Criteria should be satisfied to establish general causation. This was heavily criticized by Miller: C Miller, 'Causation in Personal Injury: Legal or Epidemiological Common Sense' (2006) 26 *Legal Studies* 544, 566.
49 Taubes (n 7). Although this article is not as authoritative as other sources, as it is not a peer-reviewed science journal, it is argued that it is nonetheless likely to be reflective of the views of some epidemiologists.
50 Hill (n 26) 298.
51 A Broadbent, 'Inferring Causation in Epidemiology: Mechanisms, Black Boxes, and Contrasts' in PM Illari, F Russo and J Williamson (eds), *Causality in the Sciences* (OUP 2011) 57.
52 As more exposure to the substance should lead to an increase in incidents within the exposed group.
53 Hill (n 26) 296; 300.
54 Feldschreiber and others (n 17) 188.

suffer X disease *without* exposure, but 2/1,000,000 people suffer the same disease *with* exposure, the RR is 2, but the absolute risk is very low; this undermines the existence of a causal link. Therefore the RR should not be decisive of general causation.[55]

Finally, as making a policy-based decision aimed at protecting public health involves the interplay of a multitude of factors, where regulatory action based on epidemiological evidence has been or is going to be taken to protect public health, this should also not play a decisive role in the general causation inquiry.[56] For example, there are huge practical difficulties with banning cigarettes, but this certainly does not invalidate the causal link between smoking and lung cancer. Accordingly, regulatory action (or lack thereof) supported by epidemiological evidence should not be treated as conclusive proof of general causation.[57]

Taking these factors into account, if the epidemiological evidence shows that there is, on the balance of probability, a causal link between two variables, this will be enough to satisfy the general causation test. To reiterate, there is no prerequisite for particularistic evidence to be presented at this stage, but if it is, this should be evaluated in conjunction with the epidemiological evidence and a balanced conclusion regarding general causation should be reached.

Conversely, Khoury suggests a different approach. Instead of applying a balance of probabilities test, 'the [general] causation inquiry should be strictly limited to assessing a *possible* causal association between the damage and the alleged cause'.[58] Thus it does not have to be 'more likely than not' that 'substance X' causes 'disease Y'; a mere *likelihood* of a causal link is enough. This is because the epidemiological surveys 'may not be meaningful in light of the particularities of individual cases'.[59] This would also lessen the 'burdensome' task of having to establish general causation and would avoid an excessively narrow focus on the statistical evidence, which would ease the judicial process in such complex tort cases.[60]

While this is a superficially attractive argument, there are major concerns about setting the barrier so low. This is because, as noted above, imposing liability on a defendant for exposing a claimant to a substance which has not been objectively shown to cause the alleged harm (on the balance of probability) is unduly harsh and unjust. Recognising this problem, Khoury proposes that any uncertainty existing in general causation should then be taken into account at the second, specific causation, stage.[61] Yet this 'solution' of returning to general causation during the specific causation stage only serves to undermine Khoury's claim that this would be a less 'burdensome' task. On the contrary, this approach is likely to tangle and confuse the two aspects of the causal inquiry, complicating the task for the judge. As this area of law is already tainted with uncertainty and complexity, it is argued that the tests should be simplified as much as possible. Thus general causation should be independently assessed on the balance of probabilities.

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55   *Ibid* 188.
56   See R Goldberg, 'Using Scientific Evidence to Resolve Causation Problems in Product Liability: UK, US and French Experiences' in R Goldberg (ed), *Perspectives on Causation* (Hart Publishing 2011) 160.
57   Hill (n 26) 300.
58   L Khoury, *Uncertain Causation in Medical Liability* (Hart Publishing 2006) 49–50.
59   *Ibid* 50.
60   *Ibid* 50.
61   *Ibid* 44.

Furthermore, Khoury's point that the general causation test does not take into account particularistic evidence has also been noted by other academics.[62] Thus, as highlighted above – to ensure that the statistical data is not viewed in a vacuum and instead is analysed as only one element of the causal inquiry – it is argued that where particularistic evidence is available to support or challenge a finding of general causation, this evidence can and should be used.[63] This involves, for instance, ensuring that the epidemiological evidence is as *relevant* and *adapted to* the characteristics of the claimant and the circumstances of the case as possible (namely, the evidence should, as much as possible, reflect the claimant's age, sex, level of exposure to the substance in question, etc).

Role of the courts

Many academics have been vocally critical of the court's failure to utilise experts during cases involving epidemiological evidence.[64] Specifically, McIvor emphasises the importance of using qualified *statisticians* to analyse the data, as opposed to other scientists. McIvor compellingly argues that as epidemiology is a 'highly sophisticated and specialist branch of medical science',[65] judges and medical experts are ill-placed to make complicated inferences from statistics that are beyond their expertise. This can be seen in the shocking Sally Clark case,[66] in which a medical expert made a statistical error that a statistician would have recognised immediately, leading to a 'grave miscarriage of criminal justice'.[67] As a result, The Royal Statistical Society urged the Lord Chancellor 'to take steps to ensure that statistical evidence is presented only by appropriately qualified statistical experts'.[68]

More contentious, however, is what the judge's function should be. In *McTear*, Lord Nimmo Smith held that the claimants failed to explain the epidemiological methodology thoroughly enough to the court; subsequently, the judge felt unable to scrutinise the data in order to make an informed and reasoned conclusion regarding causation.[69] It is argued that this was unreasonable; in such circumstances, it is vital that the judge demands more guidance with respect to the evidence presented. To hold otherwise risks encouraging an excessively hands-off approach by the courts.[70] However, it is equally wrong for a judge to usurp the expert's role and make inferences or form conclusions from material not put to the court.[71] Instead it is proposed that judges should engage fully with the material, ask for *more* material where necessary and then form an independent judgment, recognising that the decision is ultimately in their hands.

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62   R Goldberg, 'The Contraceptive Pill, Negligence and Causation: Views on Vadera v Shaw' (2000) 8 *Med L Rev* 316.

63   *Ibid* 316–317.

64   See Khoury (n 58) 60.

65   McIvor (n 33).

66   *Meadow v General Medical Council* [2006] EWCA Civ 1390.

67   J Stapleton, 'Factual Causation, Mesothelioma and Statistical Validity' (2012) 128 LQR 221, 225.

68   Letter from The Royal Statistical Society to the Lord Chancellor (23 January 2002) <http://www.rss.org.uk/statsandlaw> accessed 28 August 2014.

69   *McTear* (n 48) [6.155], [6.162]–[6.163].

70   Goldberg, 'Using Scientific Evidence' (n 56) 160.

71   See *Hotson v East Berkshire Area Health Authority* [1987] AC 750 (HL)*,* where the trial judge, unconvinced by figures presented to him by either side's expert witness, settled with an average of the two (see the Court of Appeal decision: [1987] 2 WLR 287 (CA) 301–3).

IV. Stage two: specific causation

The usefulness of epidemiological evidence at the general causation stage was discussed above. Here, the usefulness of the same epidemiological evidence at the specific causation stage will be examined. Due to the nature of the data, this issue is far more contested.

As demonstrated, epidemiological evidence aims to find causal associations between variables; it asks, '*does* X cause Y?' This coincides with the general causation query. However, specific causation is asking a different question, namely, '*did* X cause Y *in this particular circumstance*?' As the former query is 'forward-looking' and the latter is 'backward-looking',[72] using epidemiological evidence to establish specific causation can be artificial and can suffer major constraints. According to Dawid, this misalignment 'leads to very significant differences in how the two questions are to be formulated and addressed'.[73]

However, it is maintained that these concerns are not strong enough to justify the exclusion of epidemiological evidence from the assessment of specific causation. Assuming that the epidemiological study has been expertly executed and the data is suitably similar to the claimant's case, it will be shown that epidemiological evidence should be used in the assessment of specific causation not only (a) *with*, but also (b) *in the absence of*, particularistic evidence. A subsequent suggestion for how the balance of probability test should be applied in both of these instances will be made.

(a) Where particularistic evidence is available

Despite the English courts' general scepticism of statistics, all the judges in *Sienkiewicz* accepted that 'with proper caution' using epidemiological evidence 'in conjunction with specific evidence related to the individual circumstances and parties is…common ground and clearly right'.[74] Many academics agree that epidemiological evidence should be used in tandem with other evidence to establish causation.[75]

Nevertheless, Wright, an 'ardent "particularist"',[76] argues that epidemiological evidence is irrelevant to the proof of specific causation; particularistic evidence 'is all that counts'.[77] To explain this theory, Wright distinguishes between causal laws and causal generalisations. A causal law describes the 'relation between some minimal set of abstractly described antecedent conditions and some abstractly described consequent condition, such that the concrete instantiation of all the antecedent conditions will always immediately result in the concrete instantiation of the consequent condition'.[78] As our knowledge of causal laws is generally 'incomplete', we employ causal generalisations instead, which 'refer to only some of the antecedent conditions in the relevant causal laws'. An example is given by Broadbent: 'a causal generalisation might be that smoking

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72 Dawid (n 12) 134; also noted by Baroness Hale in *Sienkiewicz* (n 2) [170].
73 *Ibid* 134.
74 *Sienkiewicz* (n 2) [191] (Lord Mance).
75 Broadbent, 'Epidemiological Evidence' (n 37).
76 C Miller, 'Epidemiology in the Courtroom: Mixed Messages from Recent British Experience' (2012) 11(1) *Law Probability and Risk* 85, 91.
77 RW Wright, 'Proving Causation: Probability Versus Belief' in R Goldberg (ed), *Perspectives on Causation* (Hart Publishing 2011) 207.
78 *Ibid* 205.

causes lung cancer. A causal law would be the full specification of all those factors that are present when smoking causes lung cancer'.[79]

Wright reasons that our knowledge that all the antecedent conditions of a causal generalisation were instantiated only provides a 'probability' that the underlying causal law was completely instantiated; thus only provides a probability that the relevant condition actually caused the alleged consequence.[80] Although such a probability *can* be sufficient to establish specific causation, only *particularistic evidence* is able to show whether a causal generalisation and its underlying causal law was, in fact, instantiated.[81] Consequently, following this logic, *only particularistic evidence* can be relevant to the probability that the underlying causal laws were completely instantiated; epidemiological evidence plays no role here.[82] Hence, any slight real evidence that a roll of a dice has yielded a six 'would outweigh all the probability otherwise'.[83]

Wright's theory seeks to clarify judges' differing approaches to particularistic and epidemiological evidence. Although it appears sound, however, it has been criticised for being based on 'speculative metaphysics'.[84] Unfortunately, the philosophy of causation is outside the scope of this article and, ultimately, no theory of causation has been positively validated. However, it remains a compelling argument that dismissing epidemiological evidence on account of highly disputed metaphysical presumption is 'wrong, epistemically and morally'.[85] Moreover, Broadbent persuasively argues that it is 'surely rational to take into account background frequency with which events occur…when deciding whether a particular event did in fact occur. To deny this is to make an epistemic error'.[86] It therefore seems intuitive that epidemiological evidence should be weighted together with particularistic evidence in the assessment of specific causation. As Baroness Hale helpfully remarks, 'why should what [an unrepresentative] judge thinks probable in any given situation be thought more helpful than well-researched statistical associations in deciding where the overall probabilities lie?'[87]

A slightly less radical view is held by Gold, who does not argue that epidemiological evidence is irrelevant to the question of specific causation, but rather claims that the nature of particularistic evidence requires it to have primacy over epidemiological evidence.[88] This is because 'the power of particularistic proof to generate belief probabilities regardless of known fact probabilities requires us to treat particularistic evidence differently from group-based evidence'.[89] However, Trite disagrees, arguing that '*all* factual evidence is ultimately "statistical"' and that all 'empirical data…[needs] some step of inductive inference';[90] thus irrespective of the type of evidence, it will always be based on some

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79   Broadbent, 'Epidemiological Evidence' (n 37) 261.
80   Wright (n 77) 208.
81   Particularistic evidence in this context means evidence that '[instantiates] an element in a proven or accepted causal generalisation'; Wright (n 77) 210, fn 67.
82   *Ibid* 206.
83   *Day v Boston & Maine Railroad* (1902) 52 A 771, 774 (Supreme Court of Maine) cited in Wright (n 77) 208.
84   Broadbent, 'Epidemiological Evidence' (n 37) 263–264.
85   *Ibid* 264. It is also noteworthy that Wright's view of a causal law as an exceptionless regularity has been losing support since the first half of the 20th century: see M Parascandola, 'Causes, Risks, and Probabilities: Probabilistic Concepts of Causation in Chronic Disease Epidemiology' (2011) 53 *Preventative Medicine* 232.
86   Broadbent, 'Epidemiological Evidence' (n 37) 265.
87   *Sienkiewicz* (n 2) [172] (Baroness Hale).
88   Gold (n 28).
89   *Ibid* 384, fn 42.
90   Tribe (n 36) 1330, fn 2 (emphasis in original).

prior knowledge that we have. Miller offers an example: a woman claims that she has witnessed a negligent driver drinking heavily in a bar an hour before he is involved in an accident. Miller writes, '[the accident-specific information's] probative value still relies on prior knowledge of the effect of alcohol consumption upon driver behaviour'.[91] This suggests that distinguishing between statistically collected evidence by experts in the epidemiological or scientific field and evidence gathered from, say, a witness in an interview would be arbitrary and unjustified. Furthermore, such an inflexible rule could potentially cause injustice. Green provides an example of this:

> '[A] high quality statistical study showed a very high risk – in excess of 90 percent – that someone exposed to an agent would contract the disease from which the plaintiff suffers and in which an eyewitness claims to have observed the plaintiff contract the disease from an alien bite.'[92]

Although it has been criticised,[93] Green's example illustrates the fundamental point that there will be instances where the particularistic evidence is not as *reliable*; therefore a strict rule that prioritises it above epidemiological evidence would be wrong in principle.

How to apply the 'balance of probability' test

In view of the above, epidemiological evidence should not be dismissed as irrelevant to specific causation on the basis of metaphysical theories of causation. Instead, such conceptual disputes should give way to policy concerns, which in this case strongly support the value of epidemiological evidence in the specific causation query. Furthermore, it would be both unjust and irrational to automatically disregard epidemiological data where particularistic evidence is available, as the latter equally relies on inference derived from group-based data and both are prone to fallibility.

Instead, the balance of probability test should be applied flexibly; all the circumstances of the case should be taken into account and the different forms of evidence should be weighed against one another.[94] The reliability of the epidemiological data should also be considered and added to the overall assessment. Moreover, the DTR test has no application here because where the RR < 2, there may still be compelling particularistic evidence that indicates that exposure to a substance was the cause of the injury.

(b) Where no particularistic evidence is available

In this section, it will be demonstrated that epidemiological evidence cannot *on its own* definitively prove specific causation. However, it will be argued that there are nonetheless compelling policy justifications for allowing strong and reliable epidemiological evidence to satisfy the legal standard of proof.

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91   Miller, 'Epidemiology in the Courtroom' (n 76) 93.
92   MD Green, 'The Future of Proportional Liability: The Lessons of Toxic Substances Causation' in M Madden (ed), *Exploring Tort Law* (Cambridge University Press, 2005) 352, 362 fn 38.
93   Broadbent, 'Epidemiological Evidence' (n 37) 265, fn 106, rightly observing that the eyewitness 'cannot *see* the bite causing the disease' (emphasis added).
94   *Ibid* 265.

To begin, it is necessary to explore the infamous 'Blue Cab Problem', first expounded by Brachtenbach J in *Herskovits v Group Health Cooperative of Pudget Sound*, a decision of the Supreme Court of Washington.[95] This is used by a number of 'particularists' to illustrate the fallacy of using 'naked statistics' to establish causation and a number of courts have referred to it positively.[96] The problem is as follows: a man is hit from behind by a cab. He does not see it. There are no eyewitnesses. It is known that 75% of the cabs in the town are owned by the 'Blue Cab Company' and 25% owned by the 'Yellow Cab Company'. If nothing else is known, applying the statistics directly to the case suggests that the Blue Cab Company should be held liable because it is more likely than not that the victim was hit by a blue cab. As most people (including 'probabilists') would not proceed on this basis, this arguably evidences the fallacy of using bare statistics to prove specific causation.

However, Posner argues that the main issue is not the inherent problem of applying statistics to a case; rather, it is the extreme improbability of the fact that the claimant is unable to gather more evidence.[97] This, he maintains, 'powers the intuition that the plaintiff must lose'.[98] Alternatively, and far more persuasively, Broadbent argues that the principal error in this example is that it does not accurately reflect epidemiology and the relevance of epidemiological data. He remarks, a 'closer analogy would be a case where 75% of injuries relevantly like the claimant's are *caused* by blue [cabs]'.[99] This is significant, because if statistics did show that, the intuitive response that the claimant must lose is no longer so immediate. For instance, in *Sienkiewicz*, after citing the Blue Cab Problem as support for the finding that 'statistical evidence may be an inadequate basis upon which to found a finding of causation', Lord Phillips then made the contradictory observation that, '*much more significant would have been…the past accident record of the firms in question*'.[100] Following Broadbent's reasoning, this is precisely what the epidemiological evidence would show if the Blue Cab Problem was more comparable to a real case. Broadbent notes that epidemiological evidence is not 'naked', rather 'clothed in general causal inference'.[101] He concludes, 'It is the general causal claim, quantified but not exhausted by the statistical fact, that licenses the inference to the specific causal claim.'[102] This suggests that if there was a greater understanding of the nature of epidemiological evidence, there would be substantially less opposition towards utilising it at the specific causation stage.

More fundamentally, however, the Blue Cab Problem's fatal flaw is its *general* implausibility. It seems extremely unlikely that there will be no other evidence except the number of cabs that there are in the town: no camera footage, no forensic evidence, etc. Thus there is no real *impossibility* of proof, as scientific methods make it *possible* to uncover this evidence. Therefore, using the statistical evidence on its own to establish causation defies all common sense. However, 'diseases do not operate like cabs'.[103] Owing

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95   (1983) 664 P 2d 474.
96   See for instance, *Sienkiewicz* (n 2) [96].
97   R Posner, 'An Economic Approach to the Law of Evidence' (1999) University of Chicago Law School, John M Olin Law & Economics Working Paper No 66, 40 <http://papers.ssrn.com/paper.taf?abstract_id=165176> accessed 28 August 2014.
98   *Ibid* 40.
99   Broadbent, 'Epidemiological Evidence' (n 37) 286.
100   *Sienkiewicz* (n 2) [96] (emphasis added).
101   Broadbent, 'Epidemiological Evidence' (n 37) 286.
102   *Ibid* 286.
103   *Ibid* 269.

to long latency periods and lack of medical knowledge, often there really can be no other evidence apart from statistical evidence; hence the 'impossibility' of proving causation. Moreover, in such complex tort cases, the defendant's negligence is often already established; consequently, if the data is sound, utilising epidemiological evidence is not so 'counter-intuitive' as the Blue Cab Problem suggests. As a result, the problem has no relation to cases requiring epidemiological evidence at all and offers nothing to the debate at hand.

An alternative argument recently raised in *McTear* by Lord Nimmo Smith is that epidemiological evidence concerns patterns in 'populations' and not 'individuals'; accordingly, it is irrelevant to individual cases.[104] However, this is unpersuasive because it does not take into consideration the forceful argument made above that 'all evidence, even if styled "particularistic", involves inference from observed probability patterns'.[105] Accordingly, if I drink alcohol and become drunk, years of having observed others get drunk from some alcoholic beverage helps me deduce that the alcohol was the cause of my drunkenness. Thus it is 'at least sometimes possible to conclude something about an individua', particularly in the realm of causation, 'on the basis of a fact about a population of which the individual is a member'.[106]

A more sophisticated contention is that the inherent nature of epidemiological evidence means that it is unequipped to satisfy the question of specific causation. As previously highlighted, the evidence aims to answer the broader question: '*does* X cause Y?' Specific causation, on the other hand, seeks to answer the far narrower question: '*did* X cause Y *in this instance*?' This misalignment supports the claim that epidemiological evidence cannot, on its own, conclusively support a finding of specific causation. To demonstrate this more clearly, Dawid offers the example of a simple experimental study conducted to assess the effect of aspirin on the recovery of a headache.[107] For the purposes of simplicity, 'recovery' in this context means that the headache disappears within 30 minutes.[108] The study comprises two cohorts: a treatment group, who are given an aspirin; and a control group, who are instead given a chalk tablet.[109] The results show that from the treatment group, 30/100 people recover from the headache. From the control group, only 12/100 people recover. The RR is therefore $(30/100)/(12/100) = 30/12 = 2.5$. This shows a relatively strong correlation between taking the aspirin and recovery. Assuming this experiment is constructed on a scale that would provide meaningful results, I can use this data to predict what would happen if I develop a headache and subsequently take an aspirin: I could expect my headache to recover more quickly than if I do not take an aspirin.[110] However, at the specific causation stage, the question asked is: if I take an aspirin, and my headache disappears within 30 minutes, can I use the epidemiological data to conclusively determine whether the recovery was *caused* by my taking the aspirin?

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104 *McTear* (n 48) 6.180; Lord Nimmo Smith held that the 'population attributable risk is a measure for populations only and does not imply a likelihood of disease occurrence within an individual'.

105 See above (n 90); D Rosenberg, 'The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System' (1984) 97 Harvard Law Review 849, 870 cited in *Gold* (n 28) 384 fn 42. Broadbent writes that this comes down to the simple fact that 'populations are composed of individuals': Broadbent, 'Epidemiological Evidence' (n 37) 246.

106 Broadbent, 'Epidemiological Evidence' (n 37) 246.

107 Dawid (n 12) 135–136, 140–143.

108 *Ibid* 136.

109 Note that this is an example of a RCT and not an observational study, which would be susceptible to the limitations listed in Part I.

110 Dawid (n 12) 136.

Clearly I cannot, because that is not what the data was created to do. It is on account of this that Lord Rodger in *Sienkiewicz* strongly emphasised that some *particularistic evidence* that links the epidemiological data to the case at hand is necessary to establish specific causation.[111]

Acknowledgement of the point above is vital for understanding both the nature of epidemiological evidence and its usefulness for the causation query. However, the crucial question remains: what, if anything, *can* epidemiological evidence show us at this stage?

'Attributable fraction' and the 'probability of causation'

From the data in Dawid's example, the 'attributable fraction' ('AF') can be calculated.[112] This is a statistical measure used to indicate the 'proportion of a disease burden within a population that is "attributable" to a given exposure'.[113] It is calculated as:[114]

$$AF = \frac{\text{Incidence rate if exposed} - \text{incidence rate if not exposed}}{\text{Incidence rate if exposed}}$$

This can also be written as:

$$AF = 1 - 1/RR$$

Here this would be the number of people who recovered having taken the aspirin minus the number of people who recovered without having taken aspirin, divided by the number of people who recovered having taken the aspirin: AF = (30–12)/30 = 0.6 = 60%. Or: $1 - (1/2.5) = 0.6 = 60\%$.[115] This therefore shows the extent to which recovery of the headache is *attributable* to the ingestion of the aspirin. But what exactly does this mean? Is this equivalent to saying that it is 60% likely that taking the aspirin resulted in, or *caused*, the recovery, such that the AF is equivalent to the 'probability of causation' ('PC')?[116]

The answer to this question depends on the 'type of outcome' at issue. If the outcome is one in which 'time of incidence is crucial',[117] then Robins and Greenland demonstrate that directly equating the AF to the PC is a fundamental methodological error that often leads to an underestimate of the probability of causation.[118] Alternatively, if the outcome is 'all-or-none' (such as birth defects), then 'risks but not rates are important'.[119] This article will focus on the outcome in which time of incidence is crucial (it will be noted that Dawid's example falls into this category).

To demonstrate why directly equating the AF with the PC is a methodological error, Robins and Greenland begin by explaining that the concepts '*excess fraction*' and

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111 *Sienkiewicz* (n 2) [163] (Lord Rodger).
112 Dawid (n 12) 142–143.
113 Broadbent, 'Epidemiological Evidence' (n 37) 241.
114 S Greenland, 'Relation of Probability of Causation to Relative Risk and Doubling Dose: A Methodological Error That Has Become a Social Problem' (1999) 89 *American Journal of Public Health* 1166, 1167.
115 Dawid (n 12) 143.
116 If this is so, it would explain why some jurisdictions accept that RR > 2, in the absence of other evidence, is enough to satisfy the legal burden of proof (because where the RR > 2, the PC exceeds 50%).
117 Greenland (n 114) 1166.
118 S Greenland and JM Robins, 'Epidemiology, Justice, and the Probability of Causation' (2000) 40 *Jurimetrics* 321.
119 Greenland, 'Relation of Probability of Causation' (n 114) 1166.

'etiologic fraction' must be kept distinct.[120] *Excess fraction* is the 'proportion of exposed disease occurrences that would not have taken place in the absence of exposure'.[121] *Etiologic fraction* denotes the 'cases in which the exposure makes a *causal contribution*'.[122] They show that, assuming that general causation has been satisfactorily established, it is reasonable to infer that the excess cases are caused by the exposure. However, it is unreasonable to subsequently infer that the remaining cases are *not* caused by the exposure without the use of strong biological assumptions. In fact, the etiologic fraction could be *larger* than the excess fraction, and often is, because 'it is possible that the exposure could contribute to cases that would have occurred even without the exposure'.[123] As a result, in most cases the AF 'will *underestimate* the probability of causation'.[124] However, it can equally be said that the AF can never *overstate* the PC.[125] Accordingly, the AF is actually equivalent to a *lower bound* PC:

$$PC \geq AF$$

To illustrate this point, its reasoning will be applied to the facts of *Sienkiewicz*. The Supreme Court held in this case that epidemiological evidence on its own cannot be used to establish specific causation in cases involving mesothelioma due to the etiology of the disease;[126] therefore, for the purposes of this example, it must be assumed that the etiology of mesothelioma is known and unproblematic. The basic facts are as follows: Mrs Costello was diagnosed with mesothelioma. She had been both tortiously exposed to asbestos dust during her employment and non-tortiously exposed to a low level of asbestos in the general environment. The relevant epidemiological evidence showed that 24/1,000,000 cases of mesothelioma were identified following only environmental exposure, whereas 28.39/1,000,000 cases of mesothelioma were identified following environmental exposure *and* occupational exposure.[127] From this data we calculate:

$$RR = (28.39/1,000,000)/(24/1,000,000) = 28.39/24 = 1.18.$$

$$AF = (28.39{-}24)/28.39 = 4.39/28.39 = 0.15 = 15\%.$$

The excess fraction is 4.39/1,000,000.[128] However, it would be fallacious to equate the etiologic fraction with the excess fraction and thereby to conclude that there was a 15% chance that the mesothelioma was caused by the excess exposure. This is because, in the absence of specific biological data derived from complex biological models, it is *impossible* to say that the etiologic fraction is no larger than 4.39/1,000,000 – in other words, it can only be shown that the etiologic fraction is *at least* 4.39/1,000,000. Indeed, it is highly likely that the etiologic fraction *is* larger than this, as holding otherwise leads to the

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120   *Ibid* 1167–1168.
121   *Ibid* 1168.
122   Broadbent, 'Epidemiological Evidence' (n 37) 242 (emphasis added).
123   *Ibid* 242.
124   Greenland, 'Relation of Probability of Causation' (n 114) 1167 (emphasis added).
125   Broadbent, 'Epidemiological Evidence' (n 37) 257.
126   The court concluded that as long as the claimant can show that the exposure increased his or her *risk* of developing mesothelioma by more than *de minimis*, this is enough to satisfy the burden of proof for causation; *Sienkiewicz* (n 2) [106] (Lord Phillips).
127   *Sienkiewicz* (n 2) [60].
128   Calculated as (28.39–24)/1,000,000.

'astonishing' conclusion that the people who would have suffered mesothelioma even without the excess exposure would have been somehow protected from the effects of the excess exposure.[129] This cannot be right.

Consequently, a conclusion that there was a 15% chance that the additional occupational exposure was the cause of the disease is incorrect.[130] Following Greenland's very persuasive argument, the PC must actually be *at least* 15%.[131]

What is the significance of this?

The above analysis has sought to show that although epidemiological evidence cannot conclusively prove specific causation, the calculation of the AF can give a lower bound probability of causation. This leads to the crux of the matter: if a claimant is exposed to a substance due to the defendant's negligence and is only able to present relevant and high quality epidemiological evidence that shows that there is a strong causal link between the particular substance and the disease, should this be enough to satisfy the legal standard of proof?

It is argued it should be. Fundamentally, 'but-for' causation only requires the claimant to prove causation on the *balance of probabilities*; thus, doubts may remain about causation, but as long as it is *more likely than not* that the defendant caused the injury, the claimant should win.[132] This legal standard of proof aims to do justice to both parties; as highlighted in *Merrell Dow Pharmaceuticals Inc v Hanver*, 'the law must balance the need to compensate those who have been injured by the wrongful actions of another with the concept deeply imbedded in our jurisprudence that a defendant cannot be found liable for an injury unless the preponderance of the evidence supports cause in fact'.[133] Accordingly, a system that always demands particularistic evidence before specific causation can be established tilts the balance too far in favour of the defendant. This is illuminatingly demonstrated by Lord Nimmo Smith's position in *McTear*. The judge argued that, as epidemiological evidence can never on its own be relevant to specific causation, even if general causation had been established with an RR of 10 (which is equivalent to a probability of causation of *at least* 90%), this would *not* have been enough to find the defendant liable.[134] Broadbent compellingly remarks that, faced with ten such claimants, Lord Nimmo Smith's approach would lead him to make an incorrect judgment in 9/10 of them.[135] It is not difficult to see the injustice in such a result.

In fact, it seems that even Wright recognises the overwhelming unfairness of this, as, despite being an 'ardent particularist', he attempts to curb the most extreme effects of

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129 Broadbent, 'Epidemiological Evidence' (n 37) 256.

130 See S Steel and D Ibbetson, 'More Grief on Causation in Tort' (2011) 70 *Cambridge Law Journal* 451, 451, 460, fn 44, making precisely this methodological error.

131 Greenland suggests a number of ways that the AF would underestimate the PC. For instance, in this example, the additional exposure may have led to *accelerated* occurrences; namely, the remaining 24 people may have developed the disease earlier in life than they would have but for the additional exposure; Greenland, 'Relation of Probability of Causation' (n 114).

132 C Miller, 'Liability for Negligently Increased Risk: the Repercussions of *Barker v Corus UK (plc)*' (2009) 8 *Law, Probability and Risk* 39, 51.

133 (n 39) 718.

134 *McTear* (n 48) [6.180].

135 Broadbent, 'Epidemiological Evidence' (n 37) 270.

his own position by widening the definition of particularistic evidence. This is seen in his response to Green's 'alien-bite example' above,[136] in which Wright granted that,

> '[assuming] there is no proven or accepted alien-bite causal generalisation and no other possibly applicable causal generalisation, then the only possibly applicable causal generalisation with at least some particularistic instantiation in the particular situation is the toxic-agent causal generalisation, which fact could support the formation of a belief that it was the causal process actually at work in the particular situation'.[137]

It will be recalled that the only possible 'particularistic evidence' in that example could have been the fact that the claimant was exposed to the particular agent. Thus, remarkably, Wright would support a finding of specific causation where epidemiological evidence is adduced *as long as the claimant can show that he or she was exposed to the agent that allegedly caused the injury*. This is significant for two reasons. Firstly, exposure to the alleged injury–causing substance is not particularistic evidence for the purposes of the test here proposed. Secondly, it has been emphasised that the epidemiological evidence must be *relevant* to the case at hand; as a result, evidence of exposure to the substance in question has been an unquestionably basic requirement of the two-stage test.

This striking concession by Wright suggests that despite his inflexible antipathy to using epidemiological evidence to establish specific, he acknowledges the fact that complex tort cases involving diseases and grievous injuries are simply not as black and white as examples such as the 'Blue Cab Problem' would lead one to believe. It is argued that his remedy for this injustice – an expansive definition of particularistic evidence – undermines the basis of his opposition to epidemiological evidence and thereby undercuts its own justification. Far more straightforward would be to accept epidemiological evidence at both the general causation and specific causation stages. Therefore epidemiological evidence on its own should be enough to satisfy the legal standard of proof.

How to apply the 'balance of probability' test

In light of the above, how should the balance of probabilities test be applied to prove specific causation where there is only epidemiological evidence? This can be dealt with in brief. As the data is able to indicate a lower bound probability of causation, there is no fairer or more objective conclusion than to base liability on a probability of causation of over 50%.[138] A higher threshold would favour defendants and a lower one would favour claimants; thus a 50% threshold is the only way to sustain impartiality.[139] Accordingly, where the RR > 2 (hence the PC > 50%), it is more likely than not that exposure to a substance has caused the claimant's injury; an RR > 2 should be enough to satisfy specific causation.

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136 (n 92).

137 Wright (n 77) 210, fn 67.

138 Although in Part II an aversion to mathematical quantification of the balance of probabilities was expressed, it was the attribution of a numerical value to a 'degree of belief' and the mathematical errors and confusion that this may cause that was being criticised. Here, however, these values have scientific grounding.

139 Broadbent, 'Epidemiological Evidence' (n 37) 272, 272, fn 132.

Conclusion

This article has sought to offer a functional guide to the usefulness of epidemiological evidence to causation in tort law. In doing so, it has aimed to elucidate the nature and limits of epidemiological evidence. Although the general structure of the test is not novel, it is hoped that the practical suggestions made to assist in its application will serve to clarify this complex issue for future cases.

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