

**UK LEGAL APPROACH TO DISEASE CAUSATION: EXAMINING THE ROLE  
FOR EPIDEMIOLOGICAL EVIDENCE**

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## **Abstract**

The main aim of this thesis is to advocate a more scientifically informed approach towards epidemiological evidence in disease litigation. It analyses the judicial scepticism about epidemiology in UK tort law, and finds that the myth of scientific certainty lies at the heart of the devaluation of epidemiology as proof of specific causation. It traces misconceptions about epidemiology to broader misconceptions about science as a whole (including medical science and disease), and confused legal approaches to causation. To explain why legal objections to epidemiology are erroneous, the thesis clarifies fundamental aspects of science and disease causation that lawyers need to better grasp. Scientific reasoning is inherently probabilistic. Further, medical research indicates that disease causation is usually multifactorial and stochastic. Rigid and deterministic 'but for' questions are thus fundamentally unsuited for assessing disease causation. The mismatch between legal and medical causal models makes courts resort to normative, 'backwards' causal reasoning or haphazard exceptional approaches to disease causation, where the most difficult dilemmas around causation arise. This thesis argues that courts need a better test for causation for disease that can take account of probabilistic scientific and epidemiological evidence, and suggests one such principled approach. Epidemiology can be invaluable in such an assessment of disease causation.

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## INTRODUCTION

Complex disease litigation is proving to be one of the most challenging areas of modern tort law, where the most difficult legal dilemmas are prone to arise. Most problems in this area arise at the causation stage of the negligence enquiry. This thesis aims to advocate reform in the UK legal approach to scientific and epidemiological evidence in the assessment of causation in complex disease claims. Conditions of modern living indicate that we can expect litigation for diseases caused by negligent exposure to hazardous substances to grow substantially in the foreseeable future. However, legal principles in this area are confused, and remain uninformed by the advances in modern biomedical understanding of disease.

This thesis proposes the need for a more scientifically informed and evidence-based approach to such claims. It suggests that epidemiological evidence has the potential to provide clarity to this area: potential that has often remained unused because of misconceptions about epidemiology that are widely prevalent in UK courts. This has led to courts undermining the potential probative value of such evidence, largely on the grounds of its inability to provide *absolute* proof about specific causation. The thesis will illustrate that misconceptions about science in general, particularly the ‘myth of scientific certitude,’<sup>1</sup> lies at the heart of the devaluation of epidemiology as evidence of specific causation in UK tort law. This myth is particularly problematic because disease causation is an especially

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<sup>1</sup> D Faigman, *Constitutional Fictions: A Unified Theory of Constitutional Facts* (Oxford University Press 2008) 162

uncertain area of science, and it is impossible to achieve a rational understanding of this area in the absence of a probabilistic framework of reasoning.

It is important to note at the outset that the main focus of this thesis is on the use of epidemiology in toxic tort litigation. The fundamental feature of toxic torts is that these concern claims that an injury (typically, a disease rather than a trauma) was caused by exposure to a toxic agent.<sup>2</sup> However, the arguments this thesis makes about inherent causal uncertainties (and about the value of epidemiology in assessing causal mechanisms) apply equally to all diseases regardless of whether they were caused or worsened by toxic exposure, medical negligence, or any other means. Thus, where relevant to our discussion of the legal approach to disease causation, the thesis will occasionally cite cases where the disease resulted from clinical negligence rather than exposure to toxins. For purposes of simplicity, the word 'disease' and 'illness' are used synonymously in the rest of the thesis. This thesis also uses the word 'injury' as another synonym for disease or illness, in keeping with traditional legal terminology.

Causal connections between disease, death and modern forms of risk are complicated, and scientifically uncertain causal connections constitute one of the most difficult aspects of modern civil liability.<sup>3</sup> Etiological uncertainties about

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<sup>2</sup> SC Gold, MD Green and J Sanders, 'Epidemiologic Evidence in Toxic Torts' in M Freeman and M Zeegers (eds), *Forensic Epidemiology: Principles and Practice* (Elsevier 2016) 28

<sup>3</sup> L Khoury, *Uncertain Causation in Medical Liability* (Hart Publishing 2006) 3

disease often make it difficult for claimant victims of complex disease to satisfy the 'but for' causation requirement of the negligence analysis. As a result, claimants and defendants frequently adduce scientific evidence to support their contentions about causation in such claims. However, a review of UK caselaw shows that courts make a number of serious errors in the assessment and application of scientific evidence. Arguably, nowhere is this more apparent than in the UK judicial approach to epidemiology.

Epidemiology is a scientific discipline that focuses on research into the causes of disease. As such, it has much potential to aid the assessment of factual causation in disease litigation: indeed, it might seem almost tailor-made for the job.

However, courts in the UK have demonstrated a degree of scepticism about such evidence that occasionally verges on hostile. Epistemic confusion on part of the law has led to epidemiological evidence being wrongly dismissed as invalid for the purposes of proving legal causation.<sup>4</sup> Epidemiology is still struggling to gain proper recognition within the UK legal context. This is in stark contrast to jurisdictions with greater experience of toxic tort litigation such as the USA, where courts not only accept, but actually prefer epidemiological evidence where causal disputes arise.<sup>5</sup> It is an unfortunate reality that such evidence in UK tort law remains under-utilised, and where utilised, often misapplied.

This thesis examines the reasons for the judicial objections to epidemiology, and finds that these are based in wider misconceptions about science and medicine.

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<sup>4</sup> A Broadbent, *Philosophy of Epidemiology*, (London: Palgrave Macmillan, 2013),162.

<sup>5</sup> Gold et al (n 2) 30

It will trace the legal misconceptions about epidemiology to three separate underlying areas of confusion in the law, each of which will be taken up for discussion individually in different chapters of this thesis:

- (i) The legal failure to understand the inherently uncertain and inconclusive nature of science and scientific evidence in general (uncertainty that is, for biological reasons, even more pronounced when it comes to evidence about disease causation). Most of the legal objections to epidemiology, as we will examine in the thesis, pertain to the perceived epistemic deficiencies of epidemiological inferences, and stem from the fact that it cannot provide *conclusive* proof of specific causation. Lawyers, it appears, may have failed to grasp the fundamentally probabilistic nature of all scientific knowledge.
- (ii) The lack of judicial clarity about the scope and purpose of the causation element of the negligence enquiry, leading to significant infusion of normative considerations into the factual causation analysis; and
- (iii) The inability of the rigid, deterministic 'but-for' test to deal with the complex and probabilistic evidence about disease causation.

This thesis will contend that the traditional legal test approach to factual causation is fundamentally unsuited to disease litigation. Increasing scientific evidence suggests that disease causation is complex and multifactorial. Further,

because disease-causing agents work on dynamic and variable biological systems, their effects are not always universal, linear or entirely predictable. A more rational approach to complex disease claims urgently requires, this thesis contends, legal principles that are more congruent with scientific and medical understanding of disease. This would allow courts to give due consideration to empirical evidence in the assessment of causation. This thesis will thus also propose a new legal test for disease causation that is can take better account of probabilistic empirical evidence, and which is a better “fit” for this area of the law.

Chapter 1 will discuss basic aspects of epidemiology and its problematic interaction with UK tort law. It will briefly outline the primary aims of epidemiology, and will then outline the prevalent judicial views about epidemiology as expressed in UK caselaw, primarily in two cases: the Scottish Court of Session judgment in *McTear v Imperial Tobacco*<sup>6</sup> and the UK Supreme Court judgment in *Sienkiewicz v Greif*.<sup>7</sup> Both these cases expressed some remarkably dismissive views about its potential probative value in the assessment of specific causation. The chapter will draw out three distinct but related misconceptions about epidemiological evidence that, in the view of this author, most hinder its effective use in the law. These misconceptions, in summary, are that:

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<sup>6</sup> [2005] CSOH 69

<sup>7</sup> [2011] UKSC 10



1. 'Epidemiological causal inferences are based primarily on observation rather than on experimentation, which has dubious scientific validity as this can only show associations, and can *never* shed any light on causation';
2. 'Epidemiology derives its data from population studies, thus can only ever constitute "general" evidence about causation, which is entirely irrelevant to specific causation. In other words, 'general' evidence cannot tell us anything about what might have happened in a particular individual case.'
3. 'Epidemiological inferences of causation rest almost entirely on a statistical relative risk (RR) value; that is, on an  $RR > 2$ . An  $RR > 2$  is sufficient to infer causation, and any  $RR < 2$  negates causation.' (This is now applied in legal contexts as a rigid and mechanistic 'doubling of the risk' test. When courts do consider epidemiological evidence, they almost entirely focus on whether the RR is greater than 2, which is now equated to an establishment of causation on the balance of probabilities.)

In order to clearly demonstrate why these beliefs about epidemiology reflect fundamental errors, it is essential to first clarify the underlying general legal misconceptions about science and causation (particularly disease causation). The next three chapters will proceed to explore these broader legal misconceptions. After doing so, the thesis will then return, in the concluding chapter (Chapter 5) to the specific legal objections to epidemiology outlined in Chapter 1, and will subject each of these to more thorough scrutiny, in order to illustrate why they are unfair.

Chapter 2 will explore the nature of scientific reasoning, particularly the judicial myth of scientific certainty, which it will contend has led to much wrongful use of science in the law. The legal ideal of science as being able to provide absolute proof leads to a variety of erroneous legal outcomes. This chapter hopes to demystify science and the scientific enquiry for lawyers. It also underscores that it is vital for courts to modify its expectations from science, and to learn better ways of assessing the relative scientific merits of different kinds of expert evidence, selecting the most appropriate expert, and evaluating the potential flaws in the scientific testimony adduced by litigating parties. It is entirely desirable- and essential- to examine scientific data critically, and to be mindful of its methodological limitations and caveats. However, the existence of some methodological limitations does not make all data worthless. Conclusions based on limited data are a vast improvement upon conclusions based on nothing but conjecture and intuition.

It is also important to note that this thesis does not place the blame for the problematic law-science interactions entirely on lawyers. Scientists giving evidence in courtrooms have also occasionally displayed poor practices such as going beyond their expertise or presenting subjective opinion as scientific fact. There is much mutual mistrust and misconceptions on both sides, and scientists have not always been transparent about the potential for error in their courtroom assertions. Scientists acting as expert witnesses also need to modify some aspects of their practice.

The next chapter (Chapter 3) will discuss these issues more specifically in the context of disease causation. It will first outline the problems in the legal approach to causation, such as the inflated role of causation in the negligence analysis. Causation is often seen as synonymous with liability, and this results in courts basing factual causation decisions on normative, intuitive or 'common-sense' notions of justice: in other words, to answer factual questions in ways that accord, not necessarily with the actual facts, but with judges' preferred overall outcome. These are not, this thesis contends, matters of factual causation. The factual causation enquiry must be objective and guided by empirical evidence, and this thesis strongly advocates the value of epidemiological evidence in aiding this assessment. The chapter will then highlight the complexities of disease causation, by exploring current biomedical evidence about the causes of disease. It will aim to show why rigid tests for causation do not work in this area of the law.

Chapter 4 will explore the traditional legal test for factual causation (the but-for test), as well as the exceptional alternative tests for factual causation that have been applied to disease claims. The 'but for' test is clearly unable to deal with the causal complexities of disease. Unfortunately, most diseases can be caused in so many different ways, through so many different combinations of events, that the requirement to establish 'but-for' causation for some of the most disabling illnesses is likely to tie scientists and courts in knots for the foreseeable future. There is little prospect of a satisfactory resolution under current legal principles. This has led to the formulation of a number of somewhat haphazard exceptional

tests for factual causation that courts have devised on a piecemeal basis to deal with immediate dilemmas. The chapter will outline the applications of the exceptional tests: the 'material contribution to injury' test, the 'material contribution to risk' test and the 'doubles the risk' test. It will contend that the material contribution test for causation, despite the heavy criticism it has received, is at least a more appropriate fit for dealing with complex disease than the 'but for' test, as it is much better to take account of probabilistic causation. However, the confused way in which the exceptional tests are applied, and the arbitrary criteria that restrict them to a few select diseases are dissatisfactory. As the potential unfairness of the but-for test becomes increasingly apparent in a range of debilitating diseases, it will become increasingly harder for the law to justify restricting the exceptional approach in this way, by making fine (and somewhat artificial) distinctions between diseases. Most major diseases have uncertain, complex causal mechanisms. The law must urgently re-evaluate its simplistic rigid, approach to disease causation, and this chapter will propose a more flexible, probabilistic test of general applicability to disease litigation.

Chapter 5 will then clarify a better and more coherent role for scientific evidence generally, and epidemiological evidence more specifically, in the law. It will return to the judicial objections to epidemiology raised in chapter 1, and will take each misconception up for a detailed analysis in the light of the preceding discussions about science and disease causation. This chapter will explain how epidemiologists make causal inferences through complex statistical and non-statistical methods. The chapter will then suggest a range of strategies that could help lawyers utilise scientific evidence more effectively in legal contexts, as well

as ways in which epidemiological evidence could be better applied in disease contexts. This chapter will also contend that the law may usefully be able to borrow from evidence-based medicine, which routinely involves the application of population evidence to diagnostic and treatment decisions for individual patients.

This thesis acknowledges that epidemiology (like all scientific disciplines) has potential for error. Epidemiological studies can vary in quality, and some scientific studies can have very dubious validity. Thus, such evidence must be considered with balance and nuance, and in the context of all other available evidence. Further, such evidence cannot be determinative of causation or liability: it must be used only to *guide*, rather than to *decide*, the legal outcome. However, none of these problems constitute sufficient grounds to reject epidemiological evidence as proof of specific causation. To reject this evidence on such grounds as UK courts cite would mean that most forms of scientific evidence (including forensic testimony) should also be rejected in legal settings.

For the foreseeable future, partial and qualified evidence about disease causation is the best that we can hope for, at least in the present state of human evolution. It is crucial for the law to frame principles that can accommodate empirical evidence from disciplines that have proven their credibility through their achievements. Epidemiological evidence is, as Broadbent points out, often the only evidence that can help prove that the wrong sued for was the cause of the

harm suffered.<sup>8</sup> If the law continues to dismiss this evidence due to misconceptions about science, it 'commits itself to finding falsehoods more often than facts.'<sup>9</sup>

The aim of this thesis is to make the case that if used judiciously, epidemiology can add much rationality and robustness to the assessment of factual causation in disease litigation. Legal debates about epidemiology suggest that courts are unaware about the extent to which medical and clinical decision-making relies on data gathered from population studies. 'Generalised' evidence is not irrelevant for specific cases; but the ability to weigh and apply it in this way does require an ability to reason under conditions of uncertainty. As some legal scholars have already pointed out, there is an incoherence in the English common law of causation when the uncertainty in scientific evidence is represented in probabilistic terms. This incoherence arises primarily from an adherence to legal principles that are not compatible with the pragmatism of epidemiology.'<sup>10</sup> This thesis will thus also propose an alternative test for factual causation in disease claims that is better able to accommodate the pragmatism of science.

Lord Phillips demonstrated the extent of the judicial misconceptions about science and medicine when he stated in the important Supreme Court judgment in *Sienkiewicz v Greif*:

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<sup>8</sup> A Broadbent, *Philosophy of Epidemiology* (Palgrave Macmillan 2013) 163

<sup>9</sup> *ibid* 180

<sup>10</sup> C Miller, 'Causation in Personal Injury: Legal or Epidemiological Common Sense' (2006) 26 *Legal Studies* 544, 545

*When a scientific expert gives an opinion on causation, he is likely to do so in terms of certainty or uncertainty, rather than probability. Either medical science will enable him to postulate with confidence the chain of events that occurred, i.e. the biological cause, or it will not. In the latter case he is unlikely to be of much assistance to the judge who seeks to ascertain what occurred on a balance of probability.<sup>11</sup>*

The rest of this thesis will aim to demonstrate the multiple layers of errors that are contained in this pronouncement.

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<sup>11</sup> *Sienkiewicz v Greif* [2011] UKSC 10 at [9] (Lord Phillips)

## CHAPTER 1

### EPIDEMIOLOGY AND ITS CURRENT STATUS IN UK TORT LAW

Exponential technological and industrial advances in the last two centuries (not always accompanied by proportionate concerns about the impact of these on human health) have resulted in the fact that many people have been exposed, often for long periods, to substances or factors that are now known to be toxic. Khoury<sup>12</sup> points out that while technological advances have led to great progress for humankind, there has also been a price to pay for this. We do not yet know many long-term effects of many of the innovative processes and substances that are used in new technologies. Further, because of the complex nature of disease causation, these long-term harms may only become manifest many years or decades later. We are currently discovering more and more adverse impacts of developments that were initially thought harmless. These have led to an exponential rise in claims for negligently caused diseases in recent decades.

The sources of potentially toxic substances in our world today are multiple and varied: including industrial and motor-vehicle emissions, contamination of water by chemical spills, hormones added to animal feed, pesticide residues in food and drinking water, household products containing chemicals,<sup>13</sup> pharmaceutical products, and substances used in the manufacturing and building industry.

Much legal attention in this area has focussed on pharmaceutical products, such as Diethylstilbestrol (DES) and the Thalidomide litigation. However, the range of

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<sup>12</sup> L Khoury, *Uncertain Causation in Medical Liability* (Hart Publishing 2006) 1

<sup>13</sup> *ibid*



substances that present risks to health is far broader than this. Growing industrial power has magnified the potential for large-scale accidents and massive exposure to toxic, environmental and other hazards.<sup>14</sup> The Bhopal Gas Tragedy, one of the largest industrial disasters in human history, led to not only devastating immediate consequences, but to an unprecedented scale of residual contamination as well as long-term disease, disability and congenital malformations, that continue to damage both the environment and the health of victims even today. Since the 1960s and 1970s, there has been increasing scientific research focus on the health impact of chemical and physical agents such as volatile organic compounds, metals, particulate matter, pesticides and radiation.<sup>15</sup>

One of the most well-known examples is the vast body of litigation for diseases resulting from asbestos, a material which we now know to be a causal factor in the development of many malignant and fibrotic diseases of the lung and pleura,<sup>16</sup> including asbestosis, lung cancer and mesothelioma. Asbestos has been used extensively in the construction and manufacturing industries since several decades, but awareness of its danger to human health began to rise around the 1950s and 1960s. However, its use continued to be quite extensive even after this, although employers were required to take measures to protect employees who faced exposure to asbestos. Peto et al<sup>17</sup> point out that asbestos use in

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<sup>14</sup> A Porat and A Stein, *Tort Liability Under Uncertainty* (OUP 2001) 3

<sup>15</sup> I Hertz-Picciotto, 'Environmental Epidemiology' in KJ Rothman, S Greenland and TL Lash (ed), *Modern Epidemiology* (Lippincott, Williams and Wilkins 2008) 599

<sup>16</sup> CB Manning, V Vallyathan, and BT Mossman, 'Diseases Caused by Asbestos: Mechanisms of Injury and Disease Development' (2002) 2 *International Immunopharmacology* 191, 191

<sup>17</sup> J Peto, A Decarli, C LaVecchia, F Levi and E Negri, 'The European Mesothelioma Epidemic' (1999) 79 *British Journal of Cancer* 666-672

Western Europe remained high until 1980, and substantial quantities are still used in several European countries: Europe, they note, is in the grip of a 'mesothelioma epidemic'. Although this is a worldwide concern, it should be of particular concern in the UK: a recent study commissioned by the UK Health and Safety Executive has identified Britain as having the highest mesothelioma death-rate in the world.<sup>18</sup>

Given this scenario and the future projections, it is imperative for UK tort law to develop a coherent, principled strategy to deal with negligence claims for diseases allegedly caused by toxic exposures. Unfortunately, however, the challenges of establishing causation (an essential component of liability in negligence) in disease claims have led to a bewildering array of haphazard legal approaches towards the causation analysis in UK tort law. Disease causation is one of the most uncertain areas of science, and current medical models stress the complex and multifactorial nature of most diseases.<sup>19</sup> Thus the need for scientific and medical evidence is particularly pressing in disease and toxic tort claims, but the legal assessment of such evidence in UK law is often confused, displaying poor judicial understanding about some key aspects of science and disease causation.

Epidemiology, with its primary focus on researching the causes of disease; as well as its multifactorial statistical and analytical tools, would seem to have much to offer to this assessment. It is only relatively recently that

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<sup>18</sup> J Peto and C Rake, *Occupational, Domestic and Environmental Mesothelioma Risks in Britain: Research Report* (Health and Safety Executive No. 696, 2009)

<sup>19</sup> As will be discussed in detail in Chapter 3 of this thesis

epidemiological evidence has started to be adduced in disease and hazardous substance litigation in the UK, in order to assess whether an exposure to particular factor caused the disease in question. Lasagna and Shulman point out that courts in the US view epidemiological evidence not only as valuable, but as crucial to the establishment of causation in disease litigation.<sup>20</sup> Many claimants in the US today find that their claims might be dismissed without epidemiological evidence to support their arguments about causation.<sup>21</sup> However, the situation is very different in the UK: the application of epidemiology in UK courts has been fraught with difficulty, much of it arising from judicial scepticism about whether epidemiological evidence is valid for aiding the analysis of specific causation.<sup>22</sup> Courts express a variety of doubts about its utility in personal injury litigation; questioning its methods, relevance and epistemic validity.<sup>23</sup> They also display substantial confusion about how to evaluate and apply such evidence to the causal analysis. On the few occasions that courts have explained the reasons for this scepticism, the objections appear to largely centre around its inability to *conclusively* prove specific causation, and the perceived lack of scientific rigour of some of its methods. However, the consequent rejection of epidemiological evidence as proof of specific causation on such grounds suggests the judicial belief that conclusive proof is possible in other sciences: in other words, from the myth of scientific certainty. This thesis contends that the UK legal devaluation of epidemiology is unfair and unjustified, if (as all available evidence indicates) it is founded on the overtly probabilistic nature of epidemiological reasoning around

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<sup>20</sup> L Lasagna and SR Shulman, 'Bendectin and the Language of Causation' in KR Foster, DE Bernstein and PW Huber (eds), *Phantom Risk: Scientific Inference and the Law* (MIT Press 1993) 110-111

<sup>21</sup> *ibid* 116

<sup>22</sup> A Broadbent, *Philosophy of Epidemiology* (Palgrave Macmillan 2013) 162

<sup>23</sup> C McIvor, 'The Use of Epidemiological Evidence in UK Tort Law' in S Loue (ed) *Forensic Epidemiology in the Global Context* (Springer 2013) 55

causation. Judges seem to lack awareness about the fundamentally uncertain nature of all science. Further, such criticisms also make it obvious that courts in the UK have little awareness about the complexities of disease causation, and about the increasingly probabilistic medical models that are emerging from recent research.

This chapter aims to introduce the basic principles of epidemiology, and to outline the current legal UK position towards epidemiological evidence. It analyses the caselaw that illustrates judicial views about epidemiology, and tries to delineate, from judicial pronouncements, the various specific problematic beliefs that courts have about this branch of science. The ultimate aim of this thesis will be to demonstrate why these judicial beliefs, and the resultant undermining of epidemiology, are erroneous. However, the thesis traces the legal misunderstandings about epidemiology in causal disputes to underlying misconceptions about wider issues than just epidemiology. It argues that misconceptions about epidemiology actually arise from misunderstandings about science as a whole, disease causation in particular, and rigid, confused legal approaches to causation in UK tort law. In order to clearly demonstrate why the judicial objections to epidemiology outlined in Chapter 1 are erroneous, it is necessary for this thesis to first clarify some fundamental issues about these wider topics that the law needs to grasp more clearly in order to develop a more rational approach to disease litigation. Thus, having introduced the problematic judicial beliefs about epidemiology in Chapter 1, the next three chapters of the thesis will focus on underlying legal misconceptions about science and disease causation. The thesis will then return, in Chapter 5, to a more thorough scrutiny

of the epidemiological issues raised in Chapter 1, and will illustrate why the devaluation of epidemiological evidence is fundamentally flawed. Section 1 begins with a preliminary introduction to epidemiology as a scientific discipline, and offers a brief summary of the aims, impact and methodology of epidemiology. Section 2 describes the current position of epidemiology in UK law. Judicial views about epidemiology remain rather vaguely articulated, but on the few occasions when courts have discussed it, the attitude towards epidemiology has been characterised mainly by substantive confusion about basic epidemiological concepts,<sup>24</sup> as well as by marked scepticism about its validity in proving specific causation. Section 2.1 will discuss in particular two recent cases which illustrate this: *McTear v Imperial Tobacco*<sup>25</sup> and *Sienkiewicz v Greif*.<sup>26</sup> Section 2.2 will describe, by way of contrast, the approach US courts take towards epidemiological evidence in the assessment of specific causation. Epidemiology is not only valued in US courts, but is sometimes seen as almost *crucial* to the assessment of disease causation. Section 3 will then delineate the various problematic assumptions about epidemiology that are highlighted by the caselaw, that in the view of this author hinder the effective use of such evidence.

## **SECTION 1: What is epidemiology: aims, scope and impact**

Epidemiology is the study of the distribution and determinants of disease, health-related states, and events in human populations.<sup>27</sup> Greenland and

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<sup>24</sup> C McIvor, 'Debunking Some Judicial Myths About Epidemiology and its Relevance to UK Tort Law' (2013) 21 *Medical Law Review* 553, 553-555 (hereafter, McIvor, 'Debunking...')

<sup>25</sup> [2005] CSOH 69

<sup>26</sup> [2011] UKSC 10

<sup>27</sup> S Greenland and KJ Rothman, 'Measures of Occurrence' in KJ Rothman, S Greenland and TL Lash (ed), *Modern Epidemiology* (Lippincott, Williams and Wilkins 2008) 32

Rothman<sup>28</sup> note that this definition of epidemiology also includes physiologic states such as blood pressure, psychological states such as depression, and positive outcomes such as disease immunity, all of which can form the subject matter of epidemiological studies.

Although there are other disciplines, such as clinical medicine, which also share the same interest in health and disease, there are some central aspects of study that are unique to epidemiology. One key difference between epidemiology and clinical medicine is in the method of study: the focus in epidemiology is on gathering data about disease causation by studying population distributions.<sup>29</sup> This, as we shall examine in detail later,<sup>30</sup> is amongst the factors that have most hindered the legal application of epidemiological evidence; and given rise to the greatest doubt about its potential probative value in the assessment of specific causation. The second key aspect of epidemiology is that it relies primarily on observation rather than experimentation to gather data. This has also aroused legal scepticism about its epistemic validity and credibility as a science, due to the seeming legal view that good science must involve experiments.<sup>31</sup>

Epidemiological studies then statistically analyse this data collected from research populations through observational research methods. Thus, statistics are a very important research tool for epidemiologists, but contrary to what many UK lawyers believe, they are only one of the many analytical tools that epidemiologists use. Epidemiological inferences of causation, as we will see

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<sup>28</sup> *ibid*

<sup>29</sup> Greenland and Rothman (n 16) 32

<sup>30</sup> Explained further below, section 3.2

<sup>31</sup> Explained further below, section 3.1

below in section 1.1 and in Chapter 5 of this thesis, are based on a number of complex quantitative and qualitative analyses. The Bradford Hill factors, outlined below, are an example of an important further non-statistical analytical process that epidemiologists use to scrutinise observational data, in order to rule out erroneous or false-positive inferences of causation.

### ***1.1: Contribution to medicine and health***

One of the primary objectives of epidemiologic research is to obtain valid and precise estimates of the effects of a potential cause on the occurrence of disease.<sup>32</sup> When epidemiologists seek to identify ‘determinants’ of a disease, the determinants they are primarily interested in are the causes of the disease.<sup>33</sup> Although epidemiology could in theory use these methodologies to study many different subject matters, its traditional focus has centred primarily on health and disease.<sup>34</sup> It is, therefore, potentially extremely relevant to legal disputes around disease causation.

Although very few of us realise it, the results of epidemiological studies make headlines more often than most other sciences.<sup>35</sup> Rising public interest about health and environmental issues has resulted in findings from epidemiologic studies receiving constant attention in the news and media. It is rarely given the credit that it is due, however, and remains poorly recognised. As Dr. Ben Goldacre, physician and senior researcher at the University of Oxford’s Centre for Evidence-Based Medicine bemoans in his bestselling book ‘Bad Science’: ‘The

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<sup>32</sup>Greenland and Rothman (n 16) 32

<sup>33</sup>Broadbent (n 11) 3

<sup>34</sup>Broadbent (n 11) 3

<sup>35</sup>ibid 1

process of obtaining and interpreting evidence isn't taught in schools, nor are the basics of evidence-based medicine and epidemiology, yet these are obviously the scientific issues that are most on people's minds....Science coverage now tends to come from the world of medicine.<sup>36</sup> Epidemiology has played a crucial role in enhancing our understanding about infectious disease, role of genetic factors in illness, drug efficacy and side effects, and the impact of nutrition, environmental factors and social factors on health. This has been invaluable for improving public health, including through the development of prevention and vaccination programmes, and of new treatments. Yet, people do not hear about epidemiology, or if they do, have little idea about what it is, or of how it is relevant or useful it is to them.

The development of epidemiology as a science is a relatively recent phenomenon, and the growth of this discipline occurred through a process of gradual evolution. Following World War II, the randomized controlled trial (hereafter, the RCT) began to evolve as the gold-standard methodology for gathering data about disease causation. Simultaneously, the British epidemiologist Sir Austin Bradford Hill (who was also one of the foremost proponents of the RCT) carried out, along with Richard Doll, a landmark study based in observational methods demonstrating the links between smoking and lung cancer.<sup>37</sup> Subsequently, multiple epidemiologic studies were carried out to corroborate their findings that also confirmed the strong evidence for causal connections between smoking and lung cancer. This led to the publication of the Surgeon General's landmark report, *Smoking and Health*, in the United States in

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<sup>36</sup> B Goldacre, *Bad Science* (Harper Perennial 2009) 334

<sup>37</sup> R Doll and A Bradford Hill, 'Smoking and Carcinoma of the Lung' (1950) 2 *British Medical Journal* 739



1964.<sup>38</sup> Sir Austin Bradford Hill also developed, in view of the methodological concerns, proposals for implementing more systematised and justifiable methods for making causal inferences from observational research.<sup>39</sup> His seminal work led to the formulation of a list of factors or guiding criteria that should be considered when attempting to determine whether an observed association may be indicative of causation:<sup>40</sup>

- *Strength of association*, i.e. the magnitude of the risk ratio
- *Consistency* of the epidemiological and clinical data showing similar findings in different populations in diverse studies
- *Specificity* of the effect of exposure, i.e. that the causal factor should lead to only one disease
- *Temporality* of the appearance of results of exposure, i.e. that exposure precedes the onset of disease
- *Biological gradient* of the increase in intensity, increase in level and duration of the effects of exposure, i.e. a dose relationship;
- *Plausibility*: whether there is an association with known biologic facts about the pathophysiology of the disease or reaction. This is dependent on the state of scientific knowledge at the time the data is being analysed and collated. It may be inadequate to explain associations that may in fact be causal and, conversely, may be unable to provide a realistic estimate of the probability of such an association not being causally related.

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<sup>38</sup> United States Department of Health, Education and Welfare, *Smoking and Health: Report of the Advisory Committee to the Surgeon General* (Public Health Service Publication 1103, 1964)

<sup>39</sup> SS Coughlin, *Causal Inference and Scientific Paradigms in Epidemiology* (Bentham 2010) 3

<sup>40</sup> AB Hill, 'The Environment and Disease: Association or Causation' (1965) 58 *Proceedings of the Royal Society of Medicine* 295, 295-300. For a detailed explanation of these, see KJ Rothman, S Greenland, C Poole and TL Lash, 'Causation and Causal Inference' in KJ Rothman, S Greenland and TL Lash (ed), *Modern Epidemiology* (Lippincott, Williams and Wilkins 2008) 26-30

- *Coherence*: Whether there is general coherence of theories and evidence of the natural history of the material disease and associated exposure;
- *Experimental evidence*: Objective experimental models to investigate links between exposure, association and subsequent causality
- *Analogy*: This requires assessing whether there is any evidence or observations on analogous cases. For example, is there an analogous scenario that implies similarities between things that are otherwise different such a different drugs causing birth defects? If thalidomide can, so might other drugs with similarities in structure and pharmacologic properties.

Since then, epidemiology has played a vital role in helping to improve public health outcomes and our understanding about the causes of disease. Community intervention trials of fluoride supplementation in water that were carried out in the 1940s have led to widespread primary prevention of dental caries; and great strides in understanding and preventing cardiovascular disease resulted from the Framingham Heart Study, an epidemiologic follow-up study that started in 1949 and which continues to provide valuable findings more than 60 years after it first began.<sup>41</sup>

### ***1.2: Limitations and conceptual issues***

Unlike many other scientists, notes Broadbent, epidemiologists are not primarily interested in using their data to discover ‘laws of nature’ or develop theoretical

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<sup>41</sup> KJ Rothman, S Greenland and TL Lash (ed), *Modern Epidemiology* (Lippincott, Williams and Wilkins 2008) 1

frameworks: their overriding concern is with causation.<sup>42</sup> There are a number of conceptual and methodological challenges that the 'relatively young'<sup>43</sup> science of epidemiology faces in its quest for better recognition, such as, for example, its primary reliance on observational rather than experimental methods to gather data (as noted earlier), the lack of a central theory, its focus on population thinking, and its relative lack of domain sensitivity.<sup>44</sup> At a number of points during the history of epidemiology, these conceptual challenges have led to deep internal disagreements and controversy amongst epidemiologists. Rothman, Greenland and Lash<sup>45</sup> cite the example of profound differences that arose in the epidemiological world in 1978, about whether exogenous estrogens are carcinogenic. Epidemiologists debating this issue were unable to agree about some of the basic and fundamental aspects of their discipline, which the authors view as a sign that the methodologic foundations of the science had not yet been established at that stage.

However, the last third of the 20<sup>th</sup> century has seen rapid growth in the understanding and synthesis of core epidemiologic concepts and their theoretical underpinnings.<sup>46</sup> These developments, note Rothman et al, were accelerated by the tremendous increase in epidemiological activity in recent decades, such as large-scale studies of major health concerns such as cardiovascular disease, and the discovery of the link between smoking and lung cancer. These were notable milestones in the evolution of epidemiology, and led to refinements and improvements in study design and statistical methodology.

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<sup>42</sup> Broadbent (n 11) 3

<sup>43</sup> Rothman et al (n 30) 2

<sup>44</sup> For a more comprehensive review of this, and other general challenges, see Broadbent (n 11) 3-9

<sup>45</sup> Rothman, Greenland and Lash (n 30) 2

<sup>46</sup> *ibid*

However, despite its many discoveries that have transformed public health, epidemiological data (like *all* scientific data, whether observational or experimental) has its limitations. Further, epidemiological studies are highly variable in their quality and robustness. Assessing the problems with the use of epidemiologic evidence about the linkages between the anti-nausea drug Bendectin and congenital anomalies, Lasagna and Shulman<sup>47</sup> outlined the various difficulties affecting the validity of the epidemiological data. Some significant issues included recall bias (i.e. mothers of deformed babies were more likely to remember correctly the use of drugs during pregnancy), the fact that it is easy to miss modest increases in rates of congenital anomalies unless very large numbers of subjects are studied, and the fact that repeated dredging up of epidemiologic data will predictably turn up spurious correlations by chance.

These issues do not, however, make epidemiological evidence worthless: if it did, then all science would be worthless, as all empirical sciences have methodological limits and potential for error. If these judicial observations about the probabilistic nature of epidemiological evidence for assessing specific causation were simply a caution against placing *excessive* weight on such evidence, and an argument in favour of a more nuanced approach to scientific testimony, it would be difficult to argue with the substance of the criticism. The problem arises only in the UK judicial tendency to undermine and dismiss epidemiology because of its lack of certainty and 'proof'. Epidemiologists have developed study designs that are more sophisticated, and better able to control

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<sup>47</sup> Lasagna and Shulman (n 9) 102-103

sources of error such as confounding and bias in their data. Epidemiology is now much more than simple surveys of incidence and associations: a fact that US courts increasingly recognise and value.<sup>48</sup> However, awareness about this progress and about the refinements in epidemiological methods does not yet appear to have filtered through to courtrooms in the UK. Although this thesis advocates a larger role for epidemiology in UK tort law, it asserts at the same time that judges must be mindful of the potential for error in epidemiologic (and other scientific) studies: an inflated perception of the probative value of any science is as damaging to the cause of justice as the opposite attitude of undermining the value of science.<sup>49</sup> Although a detailed evaluation of all of the internal challenges epidemiology faces is outside the scope of this thesis, we will, in this chapter (section 3) and then later in greater detail in Chapter 5, examine two epistemic issues that have particular import for this thesis, as they give rise to particular legal anxieties about the use of epidemiological evidence: the extent of epidemiological reliance on observational research methods, and the focus on population data.

## **SECTION 2: Epidemiology in UK courtrooms**

In comparison with the USA, courts in the UK have so far had limited experience of toxic tort claims.<sup>50</sup> Cases relying on epidemiological evidence form a minority of those personal injury cases in which causation is disputed, notes Miller.<sup>51</sup> But these cases do require lawyers to reveal their own understanding of probabilistic

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<sup>48</sup> Below, section 2.2

<sup>49</sup> For a detailed discussion of this, see Chapter 2 (section 2)

<sup>50</sup> C Miller, 'Epidemiology in the Courtroom: Mixed Messages from Recent British Courtrooms' (2012) 11 *Law Probability and Risk* 85, 88

<sup>51</sup> C Miller, 'Causation in Personal Injury: Legal or Epidemiological Common Sense?' (2006) 26 *Legal Studies* 544, 545

concepts. Miller points out the tendency to incoherence in the English common law of causation when the uncertainty in scientific evidence is represented in probabilistic terms: incoherence arises primarily from an 'adherence to legal principles which are not compatible with the pragmatism of epidemiology or, for that matter, lay understanding of causation.'<sup>52</sup> The legal use of epidemiologic evidence is subject to a variety of errors: this includes substantive errors (such as the belief that an RR>2 equates to causation on the balance of probabilities); as well as errors about epidemiological methods that cause overarching doubts about its ability to illuminate the legal causal enquiry. These will be explored in Section 3, below.

Overall, courts appear to feel that epidemiological inferences are simply based on 'statistics', and therefore have virtually no bearing on an individual claims. This has led to a tendency to undermine such evidence. Courts tend to give greater credence to testimony provided by medical doctors (even when their testimony is either almost entirely subjective, or heavily influenced by epidemiological research), rather than epidemiologists, in the assessment of disease causation. For example, in *Novartis Grimsby Ltd. v Cookson*,<sup>53</sup> the court preferred the doctor's testimony to that of the epidemiologist, even though the issue fell directly within the specialism of the latter, and despite the fact that the scientific basis for the doctor's testimony remained unexplained and tenuous. *Novartis* involved a claim for bladder cancer, and the dispute arose because there were two potential causes: occupational exposure to carcinogenic dyes, as well as the claimant's smoking. Experts gave conflicting testimony about the likely

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<sup>52</sup> *ibid*

<sup>53</sup> [2007] EWCA Civ 1261

causal links between the occupational exposure and the cancer. The defendant's expert was an epidemiologist who was a specialist in the causes of bladder cancer, and had published a great deal of research in the area. His opinion was that the levels of occupational exposure were so low that the claimant's smoking was more likely to be the main cause. The claimant's expert was a clinician, a consultant urologist, who testified that in his opinion, the occupational exposure was more likely to be the main contributing factor, and also provided some numerical percentages to indicate the likely contribution of each factor. However, he provided no scientific bases for these numerical figures he had presented. The Court of Appeal did not ask for the evidence behind medical expert's testimony, and accepted his figures to rule that causation was established. (This tendency to accord some experts greater status than others, and to use the wrong heuristics in evaluating scientific evidence, will be explored in detail in Chapter 2, sections 2.2 and 2.3: a crucial issue because it has played a significant role in the judicial undervaluation of epidemiological evidence).

This section will begin (in 2.1) with an overview of some key UK cases where courts have expounded their views about epidemiology and its relevance for the legal analysis of disease causation. These cases are important because they help understand the reasons for the UK judicial scepticism. Section 2.2 will examine, by way of contrast, the approach towards epidemiological evidence in US law, which must be understood also within the context of its general approach towards scientific evidence in disease claims. This section highlights that US courts place greater responsibility on judges to properly assess the reliability of scientific evidence generally (through clear admissibility criteria), and also have significantly more regard for epidemiological evidence than UK courts do (while

appreciating both its strengths and limitations).

### ***2.1: Judicial perceptions of epidemiology:***

Two important cases that help illustrate the issues that obstruct the legal use of epidemiology are the Scottish case of *McTear v Imperial Tobacco Limited*<sup>54</sup> and the Supreme Court decision in *Sienkiewicz v Greif*.<sup>55</sup> The cases involved causal disputes around the claimants' lung cancer and mesothelioma respectively.

Although epidemiological evidence has been adduced in a number of other cases, these two cases will be taken up for particular discussion because members of the judiciary discussed, at some length, not just the evidence placed before them, but their general views about epidemiology and its potential probative value for assessing disease causation in legal contexts. (Interestingly, *Sienkiewicz* was decided largely on the basis of common-law and statutory principles governing mesothelioma claims, and epidemiological evidence did not play any role, as it was not adduced by either of the parties. However, this case is illuminating for the purposes of our discussion because their Lordships took the opportunity, obiter, to advance their views about epidemiology and its relevance to specific causation).<sup>56</sup>

#### ***2.1.1 McTear v Imperial Tobacco Ltd***<sup>57</sup>

This claim was brought by the widow of a lung cancer victim against the manufacturer of the cigarettes her husband smoked. Her contention was that

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<sup>54</sup> *McTear* (n 14)

<sup>55</sup> *Sienkiewicz* (n 15)

<sup>56</sup> McIvor, 'Debunking...' (n 13) 562

<sup>57</sup> n 14



smoking had caused her husband's cancer, and that the cigarette manufacturers were negligent in manufacturing and selling them to members of the public knowing that they could cause lung cancer. The claimant brought expert witnesses (epidemiologists and doctors) in order to show the overwhelming epidemiological evidence for the causal links between smoking and lung cancer. The manufacturers admitted that the evidence for the causal link between smoking and lung cancer was strong enough to have been accepted by the World Health Organization, the UK government and the US government. However, the court held (despite the overwhelming epidemiological evidence about the links between smoking and lung cancer) that the claim failed to satisfy the causation requirement. One reason advanced for this was that no amount of 'general' evidence could be sufficient to establish causation in the *specific* individual claim at hand. Following a long and elaborate discussion about epidemiology, its methods and core concepts, Lord Nimmo Smith then proceeded, surprisingly, to not only outrightly reject the possibility that epidemiological evidence could offer proof of *specific* causation, but also rejected it as sufficient to prove *general* causal links between smoking and lung cancer (Miller notes, as an interesting related point, that: "Quite apart from public understanding of the strength of the link between smoking and lung cancer, it is somewhat ironic that a Scottish judge should come to a contrary conclusion at a time when the Scottish Parliament was passing legislation which, to protect public health, would ban smoking in public places").<sup>58</sup> Further, citing with approval the submissions of the defendant's expert, Lord Nimmo Smith astonishingly stated that not only could epidemiology not prove individual causation, but that it could not even provide information on

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<sup>58</sup> Miller, 'Causation in Personal Injury..' (n 40) 544

the *likelihood* that an exposure produced an individual's condition. The population attributable risk was, in His Lordship's view, simply a measure for 'populations' and therefore did not even imply a likelihood of disease occurrence within an individual:

*It is accordingly sufficient in my view to state that I accept, for the reasons given by him... that epidemiological evidence cannot be used to make statements about individual causation. The information provided in an observational epidemiology is generally such that it can neither confirm nor refute a causal relationship, particularly when the exposure in question is not specifically associated with a certain condition... Epidemiology cannot provide information on the likelihood that an exposure produced an individual's condition. The population attributable risk is a measure for populations only and does not imply a likelihood of disease occurrence within an individual, contingent upon that individual's exposure.<sup>59</sup>*

Lord Nimmo Smith dismissed overwhelming epidemiological evidence about general causal links between smoking and lung cancer for the purposes of assessing causation in the specific claim despite accepting overwhelming evidence that suggested the general causal link might be as high as 90%. The Court's approach in *McTear*, notes Turton, virtually amounted to requiring certainty.<sup>60</sup> In any event, his Lordship maintained, no general causal links were sufficient to prove that smoking had caused *that particular individual claimant's* lung cancer.

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<sup>59</sup> *McTear* (n 14) at [6.180]

<sup>60</sup> G Turton, *Evidential Uncertainty in Causation in Negligence* (Hart 2016) 92

Courts, this case illustrates, can draw sharp lines between what they term proof of ‘general causation’ and that of ‘specific causation’- in this instance, viewing them as utterly irrelevant to each other. Although population and individual evidence are not the same thing, to view them as so utterly disconnected and dichotomous entities is extraordinary. It is difficult to believe that His Lordship was apparently unaware about how much current medical and scientific knowledge, and how many of our day-to-day, *individual* decisions about a number of *specific* issues, are based on statistical, ‘population’ data. This level of disdain for probabilistic epidemiological evidence is startling, and a deeply worrying indication of the depth of judicial ignorance about science and scientific thinking. This myth of good science, and good evidence as having to possess the quality of “certainty” underlies much of the erroneous legal approach to epidemiology. The myth of scientific certainty and its operation in UK law will be explored at length in Chapter 2.

### ***2.1.2 Sienkiewicz v Greif<sup>61</sup>***

This was a recent Supreme Court case that was brought by the daughter of a woman who had died from mesothelioma, against her mother’s employer. The deceased had been exposed to asbestos at her workplace by her employer, but there was an additional complicating factor in regard to causation: she had also had a significant background exposure to asbestos in her environment due to the location where she lived. Statistical evidence indicated that the tortious exposure at the workplace had increased her risk of contracting mesothelioma by only 18%. The trial judge had dismissed the claim on the grounds that causation had

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<sup>61</sup> *Sienkiewicz* (n 15)

not been established on the balance of probabilities. However, the Supreme Court held that normal rules of causation did not apply in mesothelioma claims, and therefore the defendant was liable. In keeping with common law principles established in the earlier case of *Fairchild v Glenhaven Funeral Services Ltd.*,<sup>62</sup> all that was needed to satisfy causation in claims for mesothelioma caused by asbestos was proof of a material increase in risk. (A review of the alternative or exceptional approaches for factual causation, and the causal reasoning applied in situations of evidentiary uncertainty such as mesothelioma, will be presented later in detail in Chapter 4 section 2).

Although epidemiological evidence was not at issue in *Sienkiewicz*,<sup>63</sup> as neither the claimant nor the defendant had submitted epidemiological evidence, the court entered into an obiter discussion of their view of the application of epidemiology to such claims. The Supreme Court broached this topic, as McIvor<sup>64</sup> points out, mainly in response Lady Justice Smith's view expressed in the Court of Appeal that the 'doubling of the risk' test should operate as the default test for causation in evidentiary gap cases. The Supreme Court disagreed with this approach, but then thought it was necessary to cast doubt on epidemiology as a whole in order to explain why.<sup>65</sup>

Lord Phillips made several references to the purported lack of robustness and reliability of epidemiology.<sup>66</sup> Many other members of the Supreme Court, such as

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<sup>62</sup> [2002] UKHL 22.

<sup>63</sup> *Sienkiewicz* (n 15)

<sup>64</sup> McIvor, 'Debunking...' (n 13) 562

<sup>65</sup> *ibid* 562-563

<sup>66</sup> *Sienkiewicz* (n 15) at [97]-[99] and [101]-[103] (Lord Phillips)

Lord Mance and Lord Kerr, echoed the attitude of general scepticism. Lord Phillips also discussed the statistical measures that epidemiological inferences of causation are based on and propagated, as McIvor points out, the myth put forward by Mackay J in *XYZ v Schering*<sup>67</sup> and frequently repeated in toxic tort contexts, that epidemiologists treat evidence of a 'doubling of the risk' as adequate proof of causation."<sup>68</sup> Further:<sup>69</sup> 'In a set of comments apt to cause great offence to epidemiologists, Lord Kerr even goes so far as to suggest that epidemiology lacks credibility as a scientific discipline:'

*There is a real danger that so-called "epidemiological evidence" will carry a false air of authority. It is necessary to guard against treating a theory based on assumptions as a workable benchmark against which an estimate of the increase in risk could be measured.*<sup>70</sup>

Section 3 below will examine the specific methodological issues that courts have alluded to in their discussion of epidemiology, and the fundamental (erroneous) assumptions about epidemiology that are illustrated particularly by these two important cases.

## ***2.2: US judicial approaches to epidemiology: the contrast with UK***

Within the US, there has been a steady growth in hazardous substance litigation since the early 1980s, and thus a growing use of scientific expert evidence. This includes the Bendectin litigation,<sup>71</sup> toxic tort cases involving radiation

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<sup>67</sup> [2002] EWHC 1420 (QB)

<sup>68</sup> McIvor, 'Debunking...' (n 13) 553-554

<sup>69</sup> *ibid* 568

<sup>70</sup> *Sienkiewicz* (n 15) at [206] (Lord Kerr)

<sup>71</sup> for a detailed review, see Lasagna and Shulman (n 9) 101-122

exposure,<sup>72</sup> the Agent Orange litigation,<sup>73</sup> swine flu vaccination<sup>74</sup> and cases involving contamination of water supplies.<sup>75</sup> Epidemiological evidence is frequently admitted as proof of specific causation in such litigation, and is accorded a great deal more respect than in the UK.

The US approach to epidemiology presents a stark contrast to that seen in UK tort law. Note Lasagna and Shulman:<sup>76</sup> ‘..(A)n expanding body of US law openly acknowledges the importance of epidemiological evidence, particularly as a foundation for expert evidence where no direct evidence is available on the issue of causation. In *Brock v Merrell Dow Pharmaceuticals, Inc.*<sup>77</sup> (which involved birth defects allegedly stemming from the drug Bendectin), the Fifth Circuit concluded that epidemiologic studies constitute “the most useful and conclusive type of evidence.” The court went on further to emphasise that in cases of this type, “speculation unconfirmed by epidemiologic proof cannot form the basis for causation in a court of law.”<sup>78</sup>

However, the respect for epidemiological evidence in US courts is not founded on any illusions that it is a ‘perfect’ science: courts appear to value epidemiology even as they are able to engage with methodological limitations and weaknesses. The court in *Brock* also displayed a growing judicial willingness to scrutinize the expert evidence more closely. Courts took an active role in examining whether the foundations of the evidence placed before them were adequate and

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<sup>72</sup> *Allen v United States*, 588 F. Supp. 247 (D. Utah 1984) (US)

<sup>73</sup> In Re. “Agent Orange” Products Liability Litigation, 597 F. Supp. 740 (E.D.N.Y. 1984) (US)

<sup>74</sup> e.g. *Thompson v United States*, 533 F. Supp 581 (1981); *Heyman v United States*, 506 F. Supp. 1145 (S.D. Fl. 1981) (US)

<sup>75</sup> *Sterling v Velsicol Chemical Corp.*, 855 F. 2d 1188 (6<sup>th</sup> Cir. 1988) (US)

<sup>76</sup> Lasagna and Shulman (n 9) 110-111

<sup>77</sup> 874 F. 2d 307 (5<sup>th</sup> Cir. 1989) cited from Lasagna and Shulman (ibid)

<sup>78</sup> Lasagna and Shulman (n 9) 110-111

sufficient, raising questions about such methodological issues as Relative Risk (RR) and confidence intervals. This occurred even as courts acknowledged that epidemiological evidence could have methodological limitations, and were able to engage with this.<sup>79</sup> In *Amorgianos v. Nat'l R.R. Passenger Corp.*,<sup>80</sup> the court showed impressive awareness of the need for discrimination in the application of epidemiological evidence, distinguished between different forms of epidemiologic research, and pointed out that each differed in the evidentiary weight it could add to questions of causal connection.<sup>81</sup> This case drew a distinction between uncontrolled case studies or case-series reports, which it viewed as simply identification or surveys of disease incidence and “not formal epidemiologic investigations,” and contrasted these with more sophisticated epidemiological study designs such as cohort studies and case-control studies.<sup>82</sup> Designs such as cohort and case-control studies are, the court opined, “the most informative investigations used to test specific etiological hypotheses and to confirm and quantify degrees of health risk related to causal exposures...”<sup>83</sup>

The approach to epidemiology in US courts must, however, be understood in the context of the generally more informed approach towards scientific evidence, where courts are exhorted to assess the reliability of evidence, but are directed to take into account a range of factors when making this assessment. Since the

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<sup>79</sup> *ibid* 114

<sup>80</sup> *Amorgianos v National Railroad Passenger Corp*, 137 FSupp2d 147 at [163]; [167]-[168] (ED NY 2001) (US)

<sup>81</sup> See also D Sinclair, ‘Epidemiology in the Courtroom: An Evidence-Based Paradigm for the Determination of Causation in Compensation Environments’ (2010) 52 *Journal of Occupational and Environmental Medicine* 456, 458-459

<sup>82</sup> *Amorgianos* (n 69) [167]-[168]

<sup>83</sup> *ibid* [168]

Supreme Court decision in *Daubert*<sup>84</sup> in 1993, US courts now have an important 'gatekeeping' role to play in assessing the admissibility of scientific expert evidence. The *Daubert* decision charged courts with "the task of ensuring that an expert's testimony both rests on a reliable foundation and is relevant to the task at hand."<sup>85</sup> The 'reliability' test was formulated partially in response to rising concerns about 'junk science'<sup>86</sup> being freely admitted as courtroom testimony in toxic tort litigation, that were attributed in turn to the lax admissibility standards that had ensued from the earlier US Court of Appeals decision in *Frye v United States*.<sup>87</sup>

The legal issue in *Frye* pertained to the admissibility of evidence of a systolic blood pressure test. In deciding whether such evidence could be admitted, *Frye* formulated the 'general acceptance' test, which simply requires a litigating party to demonstrate that the scientific theory or technique on which their expert relies has been endorsed by a substantial majority of specialists in his or her field.<sup>88</sup> Although widely applied, note Heffernan and Coen,<sup>89</sup> the *Frye* general acceptance test was controversial, as it often became the cloak for an enormous variety of judicial practices: it was 'malleable enough to be pursued with rigour, applied perfunctorily as a matter of form, or sidestepped altogether.' Popular judicial sleights of hand, they point out, included limiting the scope of the test to the so-called hard sciences (as opposed to so-called soft sciences such as mental

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<sup>84</sup> *Daubert v Merrell Dow Pharmaceuticals, Inc.* 509 US 579 (1993)

<sup>85</sup> *Daubert* (ibid) at [597]

<sup>86</sup> E Beecher-Monas, *Evaluating Scientific Evidence: An Interdisciplinary Framework for Intellectual Due Process*, (Cambridge University Press, 2007) 8

<sup>87</sup> *Frye* 293 F 1013 (DC Cir 1923)

<sup>88</sup> ibid 1014

<sup>89</sup> L Heffernan and M Coen, 'The Reliability of Expert Evidence' (2009), 73 *Journal of Criminal Law* 488, 493



health)<sup>90</sup>; defining the relevant field of expertise so narrowly that a general consensus among experts is easy to reach; and simplifying the judicial task by relying on peer review and publication as the exclusive yardsticks for general acceptance. These are issues that continue to plague the application of scientific evidence in UK courtroom settings, and are magnified because there is little guidance for courts about the admissibility of such evidence. This leaves courts often applying the wrong criteria to assess the weight of the testimony placed before them: Chapter 2 (section 2) will discuss general errors in the UK legal approach towards scientific evidence.

*Daubert* was one of many by the claims brought against the pharmaceutical company that had manufactured the anti-nausea drug Bendectin. The claimants were children who had been born with limb defects, and whose mothers who had consumed the drug during their pregnancy. The US Supreme Court upheld the claim, and formulated a new standard for the admissibility of scientific evidence, holding that rigid 'general acceptance' rules such as that in *Frye* were unsatisfactory. Emphasising that the inquiry into the reliability of expert evidence is a flexible one, Justice Blackmun suggested a non-exhaustive list of factors for courts to take into account when determining admissibility:

- whether the theory or technique can be tested;
- whether the theory or technique has been tested;
- whether the theory or technique has been subjected to peer review and publication;
- whether the theory or technique has a known or potential rate of error;

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<sup>90</sup> A distinction that will be explored further (Chapter 2 section 1.3)

- whether the theory or technique has been generally accepted within the relevant scientific community.<sup>91</sup>

The *Daubert* decision, note Heffernan and Coen, revolutionised expert evidentiary practice: the validation standard holds sway not only in the federal courts, but also in the majority of the states.<sup>92</sup> Subsequently, the Supreme Court has further clarified the reliability test for admissibility in the cases of *General Electric Co v Joiner*<sup>93</sup> and *Kumho Tire Co v Carmichael*.<sup>94</sup> The current version of the test is codified in Federal Rule of Evidence 702, as amended in 2000. The rule contains the following core requirements: (i) the evidence must be helpful to the factfinder; (ii) it must be based on sufficient facts or data; (iii) it must be the product of reliable principles and methods; and (iv) the expert must have reliably applied the principles and methods to the facts of the case.<sup>95</sup> However, this test has also led to its own problems: most significantly, the rigid and over-enthusiastic applications of the reliability test, whereby each piece of scientific evidence is subjected to individual scrutiny and often rejected if it, on its own, fails to meet all the requirements of Rule 702.<sup>96</sup> This rigid and ‘atomistic’<sup>97</sup> approach extends to the use of epidemiology, and has occasionally led to the prioritisation of epidemiologic evidence over other forms of toxicity evidence.<sup>98</sup> The First Circuit Court of Appeals decision in the more recent case of *Milward v*

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<sup>91</sup> *Daubert* (n 73) [593]-[594]

<sup>92</sup> Heffernan and Coen (n 78) 495

<sup>93</sup> 522 US 136 (1997)

<sup>94</sup> 526 US 137 (1999)

<sup>95</sup> C McIvor and J Ahuja, ‘Obstacles to the Effective Use of Epidemiological Evidence’ (draft article, 2016)

<sup>96</sup> *ibid*

<sup>97</sup> S Haack, *Evidence Matters: Science, Truth and Proof in the Law* (Cambridge University Press 2014) 235-237

<sup>98</sup> CF Cranor, ‘*Millard v. Acuity Speciality Products*: Advances in General Causation Testimony in Toxic Tort Litigation’ (2013) 3 *Wake Forest Journal of Law & Policy* 105

*Acuity Specialty Products*<sup>99</sup> has, however, further offered further clarity about the use of scientific evidence. *Milward* clarifies that the gatekeeping role of the court is limited to ensuring that the expert testimony is supported by evidence and sound scientific reasoning, and that questions concerning the relative persuasiveness of competing opinions fall within the exclusive remit of the jury: as such, the First Circuit draws a clear distinction between admissibility and weight.<sup>100</sup>

The legal approach to disease causation in the UK may benefit from emulating such an approach: both in the responsibility placed on judges to scrutinise the reliability of scientific testimony (requiring some basic judicial understanding of the science),<sup>101</sup> as well as in the enlightened acknowledgement of the value of probabilistic epidemiological evidence. However, it is important to clarify that this thesis does not advocate that courts should go to the opposite extreme of insisting that epidemiological evidence must *always* be adduced to prove disease. Sometimes epidemiological evidence may simply be unnecessary, unavailable, or less useful than other kinds of evidence. Which evidence is the best is not a question that can be answered by rigid rules: in science, context is all-important; and what is best in one situation could well be pointless in a different situation. The crucial point this thesis makes is that the assessment of factual causation should rest, simply, on the best available evidence.

### **SECTION 3: Misconceptions about epidemiology**

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<sup>99</sup> 639 F3d 11 (2011) (US)

<sup>100</sup> *McIvor and Ahuja* (n 84)

<sup>101</sup> The situation regarding UK tort law's approach to scientific evidence as a whole is discussed in Chapter 2.

Crucially, the cases in Section 2.1 above show that most of the objections to epidemiology pertain to its overtly probabilistic nature: that it is unable to provide conclusive proof of specific causation. The singling out of epidemiology for such judicial criticism, on grounds of the uncertain nature of its conclusions, reflects the legal illusion that probabilistic reasoning is unique to epidemiology as a scientific discipline, and the corollary that other sciences are capable of providing more certain or conclusive evidence. The excessive (arguably, over-inflated, as Chapter 2 will later discuss) weight given to other forms of scientific evidence in other legal contexts, for example in criminal law, demonstrates this. Judges who rather uncritically welcome DNA/fingerprint expert evidence to secure convictions in criminal law seem, by contrast, rather untroubled by the degree of subjectivity and uncertainty in the interpretation of such data.

Broadbent distils, “from the rather convoluted jurisprudence and commentary on the topic”, 3 main attitudes towards epidemiological evidence in proof of specific causation:<sup>102</sup>

- Epidemiological evidence is irrelevant to proof of specific causation. Lord Nimmo Smith took this line of reasoning in *McTear v Imperial Tobacco Ltd*<sup>103</sup>
- Where only epidemiological evidence is available, normal causation rules may sometimes be relaxed, so increase in risk can satisfy the causation element; and

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<sup>102</sup> Broadbent (n 11) 163

<sup>103</sup> (n 14)

- That the threshold for proof of specific causation using epidemiological evidence is an RR (Relative Risk) greater than 2.

This thesis focuses on three judicial beliefs about epidemiology, which in the view of this author most significantly hinder its application and appropriate use in litigation. All of these judicial beliefs, this thesis contends, are based in erroneous or incomplete understanding of scientific evidentiary models, especially the evidence about disease causation. Thus it is particularly unfair and unfortunate that these have led to a rejection of this form of empirical evidence that could be invaluable to the law. Subsections 3.1-3.3 below will briefly outline the three problematic judicial beliefs about epidemiology that prevent its effective legal application.

### ***3.1: "Epidemiology can only show associations, never causation"***

One of the most significant problems arises from the fact that epidemiology relies substantially on data drawn from 'observational' methods, rather than from experimental research. Generally speaking, the more controlled the conditions under which a study is conducted, the less the potential for error, confounding, and bias in the results. Epidemiologists, however, largely tend to use observational use study designs (such as, for example, cohort studies and case-control studies) to gather data about exposures and outcomes, and use this data to make hypotheses about causation.<sup>104</sup> (There are, as Broadbent notes, some exceptions to this, such as RCTs, where epidemiological researchers might divide subjects into groups and then actively intervene: for example they might administer pharmaceutical drug treatments to one group, and a placebo to the

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<sup>104</sup> See Broadbent (n 11) 4-5

other, in order to assess the effect of the drug on the disease in question. Even here, however, because of the research and ethical restrictions which studies involving human participants are subject to, the situations where such research is possible, and the extent to which researchers can control the 'experiment', is restricted).

This 'nonconformity to standard philosophical images of science'<sup>105</sup> appears to have diminished the legal regard for epidemiology. Lord Nimmo Smith emphasised somewhat disdainfully in *McTear* that 'The information provided in an observational epidemiology is generally such that it can neither confirm nor refute a causal relationship.'<sup>106</sup> Lord Dyson noted in *Sienkiewicz v Greif* that epidemiological methods can only establish associations between alleged causes and effects, which are insufficient to *conclusively* prove causation.<sup>107</sup> Lord Phillips alluded to the methodological problems with epidemiological research that limited its reliability.<sup>108</sup> The dismissiveness towards observational research is not restricted to legal settings, and the scientific world has had a similar tendency, for a long period, to view observational research as somehow inferior in epistemic validity.<sup>109</sup> This is because of the intrinsic limitation of observational data: no matter how many times we observe an association between two events we suspect to be causally linked, we cannot know for certain that one causes the other, or that the same association will occur the next time.

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<sup>105</sup> *ibid* 3

<sup>106</sup> *McTear* (n 14) [6.180]

<sup>107</sup> *Sienkiewicz* (n 15) [302]

<sup>108</sup> *ibid* [267] (Lord Phillips)

<sup>109</sup> As explored later in Chapter 5, section 2.1

However, it is naïve to dismiss observational data without due consideration of its value, and without understanding the practical reasons why scientists need different methodologies. While it is certainly true that scientists should select the most stringent methodologies they can, there are situations where experimentation or heavily controlled research conditions are impossible for practical reasons. Toxic tort and disease-related research is an important example. It is, for various good reasons, often impossible to experiment by intentionally administering suspected hazardous substances to a study participant. Many other branches of science e.g. physical sciences, chemistry etc. are often less subject to similar constraints, because the focus of the research is often on chemical substances or physical objects. Rigid and blinkered perceptions of science that hold that strictly controlled experimentation is always the ‘gold standard’ often puts at a disadvantage sciences that focus on subjects where experimentation is more difficult (astronomy is another example of such a science). Epidemiology appears to have drawn the short straw in this flawed ‘hierarchy’ of sciences (for a detailed discussion see later, Chapter 2, section 1.3). This judicial undermining of observational research will be taken up for detailed discussion in Chapter 5 (s 2.1).

### ***3.2: “Population studies are entirely irrelevant to ‘specific’ causation”***

The second major legal objection to epidemiological evidence is that epidemiologists derive their knowledge primarily from the study of populations. UK courts appear to have concluded that “generalistic” evidence is almost disconnected from individual cases, and has practically no relevance to the causation question in the specific claim at hand. This view is also supported by

some legal academics. Wright<sup>110</sup> argues, for example, that population or 'general' evidence can never illuminate the question of specific causation, and that this assessment always requires 'particularistic' evidence. See reiterates his view that such evidence cannot establish whether the factor in question caused disease or injury in a specific individual.<sup>111</sup>

Lord Nimmo Smith took this dismissal of epidemiology to an extraordinary extreme, stating that not only could epidemiology not prove causation, but also that it could not even provide information on the *likelihood* that there was a causal connection.<sup>112</sup> Thus, in his opinion, measures such as RR (Relative Risk) and attributable fractions only applied to populations. This is an odd and naïve view. Population studies may not provide proof or certainty about what happens in every specific instance, but to even deny that they can provide robust information about likelihoods and probabilities is difficult to defend as a logical stand. There is undoubted truth in the judicial observation that it is necessary to exercise caution when extrapolating from population data to individual cases. This, however, should simply indicate a need for nuance when assessing data from population studies. While it is certainly true that what is true of the majority of the population is not necessarily true of every single individual within it, neither is it true that data drawn from populations provide *no* information at all about what might have happened in an individual instance. To state that facts we know to be true of the general population tell us nothing

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<sup>110</sup> See Richard Wright, 'Liability for Possible Wrongs: Causation, Statistical Probability, and the Burden of Proof' (2008) 41 *Loy. L.A. L. Rev.* 1295; and Richard Wright, 'Statistical Causation, Responsibility, Risk, Probability, Naked Statistics and Proof: Pruning the Bramble Bush by Clarifying the Concepts' (1988) 73 *Iowa L. Rev.* 1001

<sup>111</sup> For example, Andrew See, 'Use of Human Epidemiology Studies in Proving Causation' (2000) 67 *Defence Counsel Journal* 478, 478.

<sup>112</sup> *McTear* (n 14) [6.180]



about specific individuals is, as Broadbent rightly puts it, a non-sequitur: such reasoning would require us to ‘ignore great swathes of advice about not just epidemiology, but about health, nutrition, house prices, and the prospects of survival upon leaping from a third-storey window!’<sup>113</sup>

Populations are ultimately composed of individuals:<sup>114</sup> a ‘population’ is not some mythical being conjured out of the ethers. Thus it is at least sometimes possible to make inferences about an individual on the basis of information we have about the population of which that individual is a member.<sup>115</sup> This epistemic concern on part of courts appears to be related to a vague distaste for making legal decisions on the basis of statistical evidence, with the concomitant belief that because epidemiology studies populations, its causal inferences are based on no more than the statistics. Lord Phillips in *Sienkiewicz* uses the word epidemiology interchangeably with the term ‘statistical analysis’<sup>116</sup> and opines, later, that it therefore cannot illuminate the further question of whether these statistical associations demonstrate a causal relationship.<sup>117</sup> Several other members of the Supreme Court in the same case expressed the view that reliance on population data or on statistical measures such as associations and correlations in order to assess what happened in the specific case was illogical, unfair, and even, almost, dehumanising. Lord Phillips<sup>118</sup> cited a variation of the famous Blue Bus Paradox<sup>119</sup> in order to make the point that using statistics in legal decision-making could lead to arbitrary outcomes. Lord Mance, in the same

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<sup>113</sup> Broadbent (n 11) 165

<sup>114</sup> A Broadbent, ‘Epidemiological Evidence in Proof of Specific Causation’ (2011) 17 (4) *Legal Theory* 237, 246

<sup>115</sup> *ibid* 246

<sup>116</sup> *Sienkiewicz* (n 15) at [242]

<sup>117</sup> *ibid* at [267]

<sup>118</sup> *ibid* [266]- [267]

<sup>119</sup> E.g., L. Jonathan Cohen, *The Probable and the Provable* (Clarendon Press 1977) 74-81

case, cautioned that the law needed to concern itself with the rights and wrongs of situations, and that individuals and companies must “not be treated like statistics.”<sup>120</sup>

The epidemiological method has been referred to somewhat disparagingly by a section of lawyers as “naked statistics”<sup>121</sup> (it is unclear what exactly this term means, or, as Miller wonders,<sup>122</sup> what the other types are). We assume this term encapsulates the legal idea that epidemiologists simply put observational data through statistical programs, and mechanistically apply numerical criteria in order to infer (or reject) causation. However, as McIvor<sup>123</sup> points out, this is quite simply untrue: epidemiologists are more than just statisticians. They use statistics as a tool for analysis of data, but also use a variety of other sophisticated techniques to determine whether the associations indicate a causal effect. The idea that epidemiological inferences are no more than statistical measures seriously misconceives epidemiological inferences of causation, a complex reasoning process that will be explored in Chapter 5 (section 1). Miller argues that the judicial aversion to statistical evidence in establishing causation stems from a ‘vain search for a certainty that can somehow finesse the problem of induction.’<sup>124</sup> The Supreme Court case of *Sienkiewicz v Greif* shows, in Miller’s view, that courts still remain equally immune to developments in the worlds of science, epidemiology or other legal jurisdictions such as the US that have vastly revaluated their views about epidemiological evidence. The *McTear*

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<sup>120</sup> *Sienkiewicz* (n 15) at [295]

<sup>121</sup> R Wright, ‘Liability for Possible Wrongs: Causation, Statistical Probability, and the Burden of Proof’ (2008) 41 *Loy. L.A. L. Rev.* 1295-1344

<sup>122</sup> Miller, ‘Epidemiology in the Courtroom...’ (n 39) 90

<sup>123</sup> C McIvor, ‘The ‘Doubles the Risk’ Test for Causation and Other Related Judicial Misconceptions About Epidemiology’ in *Tort Law: Challenging Orthodoxy*, S Pitel, JW Neyers and E Chamberlain (eds) (Hart 2013) 219

<sup>124</sup> Miller, ‘Causation in Personal Injury’ (n 40) 545

reasoning, that insists on absolute proof of specific causation, would imply that almost any disease claim should fail for want of causation, and that all previous cases that have imposed liability for diseases allegedly caused by exposure to harmful substances are wrong: because such certainty of proof in complex disease claims is rare enough to be near impossible.

Wright's<sup>125</sup> exaggerated idealisation of 'particularistic' evidence, to the exclusion of all other considerations such as context, practicality, or robustness of the data is irrational and illogical. Particularistic data (such as eyewitness testimony) can also be inherently unreliable. Further, in disease litigation, there is often no particularistic evidence available. No expert can ever be *certain* that the patient's smoking played a causative role in their disease: a tumour caused by smoking looks no different, on a scan, from that caused by genetic or other factors. In most disease causation disputes, 'general' evidence may well be all we have, and to reject the best available evidence in the futile quest for **certain** evidence is illogical and futile. The fervor for particularistic evidence has led to a tendency to accept assertions from experts who often offer no more than subjective or 'guesswork' opinions about the claimant. Unfortunately, courts might end up favouring, under such an approach, evidence from experts who make less-than-honest assertions about specific causation over those who offer more transparent and well-grounded (but nuanced) testimony. All of these issues will be explored at greater length in Chapter 5 (section 2.2).

### ***3.3: 'Epidemiologists infer causation from RR values, and RR>2 is causation on the balance of probabilities':***

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<sup>125</sup> Wright (n 99)

Broadbent<sup>126</sup> notes that there are two important epidemiological measures that are employed to measure the strength of associations while assessing potential causation. These are the relative risk (RR) and the attributable fraction (AF) or attributable risk. The relative risk (RR) is well-known in the law, and is a measure of how much more common a given condition is among people who are subject to a given exposure than among those who are not. It is calculated as:

$$RR = \text{risk among exposed} / \text{risk among unexposed}$$

The AF is the proportion of a disease burden within a population that is “attributable” to a given exposure. This is calculated as:

$$AF = (\text{risk among exposed} - \text{risk among unexposed}) / \text{risk among exposed}^{127}$$

In addition to the above measures, epidemiologists also use a variety of other statistical as well as qualitative methods to assess data. However, lawyers appear to have focused exclusively on RR values, and this value has become synonymous, in legal minds, with epidemiological inferences of causation. This has led to a further mistaken idea within the legal profession that epidemiologists infer a causal link between an agent and an outcome based on relative risk thresholds alone, and see causation as established when the RR value is greater than 2. The result is a mantra-like insistence on an  $RR > 2$  as a magical number that confers instant significance and respectability on epidemiological inferences of causation, and this is often sufficient for courts to conclude that causation is established on the balance of probabilities. On the

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<sup>126</sup> Broadbent, ‘Epidemiological Evidence in Proof of Specific Causation’ (n 103) 240-242

<sup>127</sup> *ibid* 241-242

other hand, any RR value that is below 2 is dismissed straightaway as negating causation.

However, the interpretation of the RR value is more complex, and depends on context and background, much like all numerical data in science (a general issue that will be discussed in Chapter 5 section 3.2). Further, this view misrepresents that epidemiology uses such simplistic criteria to infer causation does the discipline a disservice. Lord Phillips states in *Sienkiewicz* that where RR exceeds 2, the 'statistical' likelihood is that the particular exposure was the cause of the disease. As McIvor points out,<sup>128</sup> this statement, coming as it does after Lord Phillips' earlier statement that epidemiology is concerned with the question of statistical associations and not with the question of whether such associations demonstrate an underlying biological relationship,<sup>129</sup> implies the belief that epidemiologists would not look any further than the statistics when formulating their conclusions about causation. In fact, epidemiologists do not treat an RR>2 as automatically indicative of causation, and do not base findings of causation on RR rates alone.<sup>130</sup> This view of epidemiology has been translated, in its legal application, into the 'doubles the risk' test for causation: the idea that an RR>2 equates to proof of causation of 51% or more, or in other words to proof on the balance of probabilities. Lady Justice Smith in *Novartis*<sup>131</sup> treated the 'doubles the risk' test as an application of the standard 'but for' test on the balance of

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<sup>128</sup> McIvor, 'Debunking...' (n 13) 572

<sup>129</sup> *Sienkiewicz* (n 15) at [84]

<sup>130</sup> McIvor, 'The 'Doubles the Risk' Test..' (n 112) 219-220

<sup>131</sup> n 42

probabilities. The claim in *XYZ & Others v Schering Health Care Ltd*<sup>132</sup> failed because the RR value was computed at just below 2, and the claimants did not contest that it was necessary to have established an RR>2 to enable their claim to succeed.

The law has made a serious error in its reading of epidemiology, and in the way it has applied this to the legal question. As Broadbent notes,<sup>133</sup> the RR value is not determinative: the significance of the epidemiological evidence depends on the context. An RR>2 can be sufficient for proof of specific causation where there is no other evidence, but may not be sufficient if there is other evidence against that proposition. Similarly, an RR<2 does not disprove causation. Further, it is illogical to apply RR in this mechanistic way to conclude causation on the balance of probabilities.

By contrast, at least some US courts must be credited for taking a more flexible and informed approach to RR values, as well as for rejecting the atomistic approach to data. Viewing several pieces of toxicity evidence in conjunction with the epidemiologic evidence, the court in *Oxendine* imposed liability in a claim where the RR value was between 1.3-1.8. The court concluded it was satisfied that all the evidence put together with the epidemiological evidence (including in

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<sup>132</sup> [2002] EWHC 1420 (Q.B.)

<sup>133</sup> Broadbent, 'Epidemiological Evidence in Proof of Specific Causation' (n 103) 239

vivo and in vitro studies) was sufficient to establish causation on then balance of probabilities, even though the RR value was less than 2.<sup>134</sup>

Chapter 4 (section 2.3) and Chapter 5 (section 2.3) will further elaborate on the problems with the view that epidemiological inference of causation are based just on an  $RR > 2$ , as well as the problems with the doubling of risk test.

#### **SECTION 4: Facilitating legal clarity about the value of epidemiology: the importance of clearer understanding of science and disease causation**

Unfortunately, the overtly probabilistic nature of epidemiology has led UK courts to devalue and dismiss sciences such as epidemiology that offer robust evidence of general causal links. This thesis will attempt to show that none of the concerns about epidemiology that courts have expressed so far constitute valid grounds to dismiss its credibility as a science, or its probative value in the assessment of specific causation.

A key contention of this thesis is that that the myth of scientific certainty is central to the to the misconceived and sceptical judicial attitude towards epidemiology. The judicial pronouncements cited above illustrate that UK courts appear to have fundamental misconceptions about science in general, and about what scientific evidence can realistically provide to the legal enquiry. Science, as a whole, is fundamentally probabilistic and uncertain. The misalignment

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<sup>134</sup> *Oxendine v Merrell Dow Pharmaceuticals, Inc.*, 563 A. 2d 330 (DC App 1989), cited from Lasagna and Shulman (n 9) 115

between the myth and reality is even more magnified in disease litigation, because disease causation is one of the most uncertain areas of science. A sensible approach to disease litigation can only be formulated when the law learns to use probabilistic evidence about causation with greater rectitude.

Specifically, this thesis locates the scepticism about epidemiology in UK tort law within three broader problematic contexts, each of which will be taken up in turn in the next three chapters of this thesis:

- (i) *Judicial ignorance about the basic philosophy scientific reasoning.* Of particular concern is the fact that courts seem to think probabilistic reasoning is unique to epidemiology, when in actuality it is a defining feature of science in general. The ‘myth of scientific certainty’ is a matter of serious concern, not just in disease litigation, but also in many other areas of the law where judges frequently and routinely rely on scientific evidence to aid their decision-making. It leads to poor use of expert evidence and errors in judging how much weight a piece testimony deserves. Chapter 2 will elucidate the issues and misconceptions that plague the law-science relationship.
- (ii) *Lack of conceptual clarity around the role and scope of the factual causation requirement in negligence:* This will be examined in Chapter 3. The judicial tendency to see causation as synonymous with liability has led to an excessive infusion of normative considerations into the factual inquiry. Judicial anxiety to retain discretion over the outcome of the causation analysis seems to prevent the causal enquiry from



being carried out with any rationality or objectivity. This thesis strongly disputes the idea that causation is a normative issue.

Causation analysis should be accorded a narrower place in the overall liability analysis but should be carried out with due regard for the best available empirical evidence about factual matters.

- (iii) *The rigid and deterministic 'but for' test, that aligns poorly with increasingly probabilistic scientific models of disease causation:* The 'but for' test has proven unsatisfactory in many situations in recent decades, and it is no accident that these have most often occurred in disease disputes. This will be taken up for detailed discussion in Chapters 3 and 4. Disease causation is complex, and most major diseases can be caused through a number of different combinations of events. Thus, science can only provide probabilistic evidence of the likely contribution of a causal factor. However, the 'but for' test is too rigid and deterministic to be able to accommodate this. This leads to courts sometimes formulating odd exceptional causal tests, in a piecemeal fashion, in order to avoid the injustice of the 'but for' test. This thesis contends that a coherent legal approach to disease claims requires a more flexible, principled test for disease causation. This will not only bring the law more in line with modern medicine and science, but will also help courts to better utilise probabilistic evidence from science and epidemiology.

After clarifying these broader areas of misconception, the thesis will then return, in Chapter 5, to the judicial misconceptions about epidemiology outlined in this chapter, and will explain why these are wrong in light of the preceding

discussions about science and disease. Through these discussions, this thesis seeks to demonstrate that epidemiology, used wisely, can contribute valuable evidence about specific causation. It will also acknowledge, however, that a good deal of discrimination and judgment is crucial when using scientific evidence in legal settings.<sup>135</sup> As Feldschreiber, Mulcahy and Day<sup>136</sup> point out, statistical and epidemiological methodologies measuring associations and causal relationships can be highly useful and practical, but the methodology is only as good as the integrity, comprehensiveness and quality of the data used. Databases can be erroneous (deliberately or inadvertently), and this can lead to false assumptions as to causation of injury and adverse events. Applying such evidence to real-world questions thus requires some basic legal felicity with scientific and probabilistic methods. Dawid cautions that '(d)ata, even scientific data, never speak for themselves: before we can sensibly interpret the data it is vital to know and take account of the nature, protocol and properties of the study which the data resulted from.<sup>137</sup> To believe that there are hard and fast rules by which medical science can use biostatistical and epidemiological data to draw definitive conclusions about disease causation is an oversimplification<sup>138</sup> and will lead to wrong conclusions. At the same time, it is crucial not to overstate the differences between law and science. As Soar notes, despite the number of divergences, the law is no stranger to scientific inquiry: science, like the law, has its own philosophy, and lawyers use scientific notions and theories in everyday legal

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<sup>135</sup> P Feldschreiber, L Mulcahy and S Day, 'Biostatistics and Causation in Medicinal Product Liability Suits', in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 182

<sup>136</sup> *ibid* 185-187

<sup>137</sup> A P Dawid, 'The Role of Scientific and Statistical Evidence in Assessing Causality', in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 135.

<sup>138</sup> Feldschreiber et al (n 124) 179

practice.<sup>139</sup> Wilson<sup>140</sup> points out that what a criminal investigator does is not very different to the process of scientific fact-finding: investigators, he observes, form a subjective hypothesis determining how to progress a crime scene, and, like in science, may not know how partial their evidence is, or what evidence is missing. Trials require subjective judgment, and evaluating subjective probability is simply what scientists call the Bayesian approach. At the same time, cautions Wilson, even subjective investigations must be rational, and can be aided by research and inductive logic- all of which can only suggest, but never ensure truth.<sup>141</sup> While experimentation is the scientific ideal, there are many cases where it is simply not available, be it for pragmatic, economic or ethical reasons, and recourse to pure observation becomes a pragmatic necessity.<sup>142</sup> Most knowledge about disease causation rests, therefore, on observational data, as experimentation involving deliberate administration of suspected disease-causing agents to assess causal links is clearly impossible. The question that requires closer examination is not which is the more superior form of evidence, but the oft-repeated claim that epidemiological or general evidence is never sufficient to establish specific causation:<sup>143</sup> for, as authors such as Broadbent ask, does epidemiological evidence have a role to play in the assessment of specific causation when it is the only evidence we have to prove causation?<sup>144</sup>

This thesis will seek to illustrate that it does.

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<sup>139</sup> K Soar, 'A Probabilistic Theory of Causation' (1996) UCL Juris. Review 132, 132

<sup>140</sup> A Wilson, 'Away From the Numbers: Opinion in the Court of Appeal' (2011) 75(6) Journal of Criminal Law 503, 503

<sup>141</sup> *ibid* 503

<sup>142</sup> Dawid (n 126) 136

<sup>143</sup> S Haack, cited from Broadbent 'Epidemiological Evidence' (n 103) 239

<sup>144</sup> Broadbent, 'Epidemiological Evidence...' (n 103) 239

## CHAPTER 2 SCIENCE AND THE LAW: THE LEGAL MYTH OF SCIENTIFIC CERTAINTY

Causal inference about disease is only a special case of the more general process of scientific reasoning.<sup>145</sup> One of the main propositions of this thesis is that the problematic legal approach to medical and epidemiological evidence in disease causation disputes does not occur in a vacuum, but is symptomatic of broader misconceptions the law holds about science. Amongst the most pernicious of these misconceptions for disease litigation, in the view of this author, is the legal myth of scientific certainty. Many lawyers appear to view the ideal science as a deterministic inquiry that must provide certainty to the legal question. The myth of scientific certainty, it is contended, is particularly important in this context because it has caused epidemiology to be devalued in UK law for its perceived epistemic deficiencies, due to its overtly probabilistic evidence about specific causation, as we saw earlier in Chapter 1. Observational epidemiologists, as Rothman et al note,<sup>146</sup> often face the criticism that proof is impossible in epidemiology, and that epidemiologic causal relations are only suggestive. This view seems to reflect an idea that proof is possible in other sciences. Unfortunately, they point out, causal relations derived from other methodologies such as experimentation are also only suggestive, as we shall see later in Chapter 5 (section 2.1).

The law tends to take a deterministic view of the world: it operates under the assumption that there are fixed, universal laws of nature, and that all events have clear, linear causal mechanisms. Thus, in the legal view, ‘good science’ should ideally

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<sup>145</sup> KJ Rothman, S Greenland, C Poole and TL Lash, ‘Causation and Causal Inference’ in KJ Rothman, S Greenland and TL Lash (ed), *Modern Epidemiology* (Lippincott, Williams and Wilkins 2008) 18

<sup>146</sup> *ibid* 24

be able to uncover these fixed laws, and provide a clear, certain answer to questions. In fact, science is a much more probabilistic and uncertain enquiry than lawyers believe. It is vital for lawyers to recognise that all scientific inference is probabilistic, including in disciplines such as physics, medicine and forensic sciences: fields which often tend to find much more favour with the law, due to inflated perceptions about their 'conclusive' nature. This is tragic, because honesty about the uncertainty in one's conclusions may actually be a characteristic of better science, as Berger and Solan point out: 'Junk science has no place in the legal system. But when a scientist says, "I'm not sure, but the data are suggestive," the scientist's words are not necessarily a telltale sign of junk science. On the contrary, they may be a sign that real science is occurring. The Supreme Court's *Daubert* trilogy appears to have allowed the continuation of junk science, while denying individuals their day in court when their proof includes real science at a state of incomplete knowledge. The solution, we believe, must lie in the legal system, judges and lawyers alike, recognising what it means to be a gatekeeper with respect to scientific truth. And that, in turn, requires the legal system to come to understand just what it is that scientists do.'<sup>147</sup>

This chapter is an attempt to help clarify to lawyers what scientists do. It explores the wider issues surrounding the law-science divide as they touch upon the factual causal assessment in disease litigation. It will examine how scientists make inferences; with the ultimate aim of helping lawyers attain a clearer understanding about how to apply scientific evidence in disease causation disputes. The focus of the discussion about science here is therefore on the epistemic and methodological aspects of the scientific

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<sup>147</sup> MA Berger and MA Solan, 'The Uneasy Relationship Between Science and the Law' (2008) 73 Brooklyn Law Review 847, 847

inquiry rather than on the history or metaphysical aspects of science, as much of the judicial confusion around epidemiological evidence in the UK centres on epistemic concerns (as noted earlier in Chapter 1). Scientists use a range of reasoning strategies, but courts have a somewhat hazy understanding of the purpose or strengths and deficiencies of these. Thus the law tends to apply rigid and over-simplified rules to assess scientific evidence, which leads to various legal errors.

Scientific inference is, this chapter argues, ultimately not very different from ordinary, or ‘common-sense’ inference: this is a term that many courts and judges express approval of in explaining their approach to causation, as we shall see later in Chapter 3 (section 1). The law however, usually employs this term in order to justify largely subjective decisions about causation. In science, on the other hand, the term means something different, and is much **more** than ‘common sense’ as judges use the term. As the eminent philosopher of science Karl Popper explains, although scientific knowledge is the result of the growth of common sense knowledge, it is at the same time an enlargement of common sense: it attempts to go beyond, or get rid of, what Popper calls the “pseudo-psychological or subjective method of the new way of ideas.”<sup>148</sup> Science is, as Popper puts it, ‘common sense writ large.’<sup>149</sup>

Section 1 will examine the differences between law and science in their approach to proof, knowledge and certainty: differing perspectives that come even sharply into contrast when science is relied upon to answer factual disputes in litigation.<sup>150</sup>

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<sup>148</sup> KR Popper, *The Logic of Scientific Discovery* (first published 1959) (Routledge 2002) xxv-xxvi

<sup>149</sup> *ibid* xxvi

<sup>150</sup> as, for example, in questions about disease causation, which is the particular focus of this thesis. See D Faigman, PA Dawid, and SE Fienberg, ‘Fitting Science into Legal Contexts: Assessing Effects of Causes or the Causes of Effects?’ (2014) 43 *Sociological Methods and Research* 359 (hereafter, Faigman et al, ‘Fitting Science..’)

Subsection 1 will highlight that these misconceptions have led to failure to correctly assess and apply scientific (particularly epidemiological) evidence to questions of disease causation. The legal view of the certainty as the ideal of knowledge is out of step with current scientific understanding about the world. Such misconceptions have led to differences in legal regard for what the law sometimes calls ‘hard sciences’ and ‘soft sciences’. Hard sciences include disciplines such as physics, chemistry and biology/medicine, which are seen as certain, and therefore as highly valued and epistemically valid. Disciplines such as epidemiology appear to be categorised within UK judicial systems as ‘soft sciences’, which are treated as less valid and valuable. Subsection 1.3 of this chapter will explore ‘hard’ and ‘soft’ sciences, and will find the differences between them are much less marked today than when this terminology first originated around 200 years ago. It will illustrate this by examining two disciplines that are particularly valued in the law for their perceived “hardness”: physics and medicine. Science as a whole, even in disciplines such as physics that were once perceived to be highly deterministic, is moving towards realisation of fundamental, insurmountable uncertainties. It is now imperative for lawyers to grasp how use and apply probabilistic reasoning and evidence.

Section 2 will explore the legal errors that have resulted from this myth of scientific certainty. This thesis argues that this myth of ‘good science as certain’ has led to erroneous applications of epidemiological evidence (Chapters 1 & 5), as well as to the adoption of unrealistic and simplistic tests for factual causation in complex disease litigation (Chapters 3 and 4). This chapter will first examine the impact of this myth about science more broadly in the law. Misconceptions about science have wide legal ramifications, and lead to a range of errors, across different branches of the law, and

across jurisdictions. It has caused courts to discriminate between different scientific disciplines, and to accord different value to different sciences, depending on the extent to which they are seen to provide certainty. Given that all sciences are probabilistic and have potential for error, this inevitably leads to legal errors. Such errors can occur both when scientific evidence is *over-valued*, and becomes the defining factor for a legal decision, as much as when it is *undervalued* and underutilised, leading to unacceptably subjective decision-making, as the discussion of the dichotomy between approaches in civil and criminal law (subsection 2.1) will show.

Section 3 then explains how scientists make inferences. It begins by exploring some philosophies of science, and the role of inductive and deductive reasoning. It concludes that there is no universal philosophy of science, but that one unifying characteristic of science appears to be the probabilistic nature of scientific reasoning. It then examines the reasoning strategies that scientists actually use to make inferences from evidence. In particular, this chapter seeks to demystify the process of scientific reasoning, and illustrates why scientific inference ultimately rests on ‘common-sense reasoning’. However, this term in science has a significance that the law fails to appreciate. Most importantly, scientists proceed on the basis that common sense reasoning is that which is guided by verifiable facts and evidence, subjected to scrutiny, and rigorously justified. The law could usefully learn from this approach. In the absence of such rigorous justification, ‘common sense’ can easily become no more than personal hunches and intuition. There are numerous examples of this occurring in the law. One example is the legal approach to factual causation in complex disease litigation, as we shall see later in Chapters 3 and 4.



In focusing on legal misconceptions about science, this thesis does not, however, suggest that all problems lie entirely on the part of the law. Scientists who provide expert testimony also need to improve their understanding about how legal questions differ from scientific study, and about the differing goals of the law. Further, scientific experts need to be more transparent in courtroom situations about the limitations of their evidence and expertise. These will be explored briefly in section 2 of this chapter, and again in Chapter 5 (section 3.4).

### **SECTION 1: The law-science divide**

The exponential pace of scientific and technological advances means that courts increasingly have to examine scientific evidence to resolve disputes. They are required to grapple with complex, and often contradictory, expert testimony, and have to make decisions about the relative scientific merits and validity of these. There is no denying the scale of the issue, or the growing ‘scientisation’ of the factual inquiry’.<sup>151</sup> As Damaska noted in 1997: “Let there be no mistake. As science continues to change the social world, great transformations of the factual inquiry lie ahead for all justice systems. These transformations of factual inquiry could turn out to be as momentous as those that occurred in the twilight of the Middle Ages, when magical forms of proof retreated before the prototypes of our present evidentiary technology”.<sup>152</sup> Unfortunately, law and science have tended to approach each other in a rather baffled and mistrustful way so far. Their interaction has variously been described as ‘an

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<sup>151</sup> M Damaska, *Evidence Law Adrift* (Yale University Press, 1997) 150

<sup>152</sup> *ibid* 51

uncomfortable alliance<sup>153</sup>, or as an ‘uneasy relationship’,<sup>154</sup> and the erroneous use of scientific evidence in courts raises concerns that the legal system may lack the necessary knowledge to correctly evaluate such evidence.

A recent academic commentary on the law and science points out that:

Law and science are entirely separate institutions, with distinct approaches to identifying what is important to consider or study, to the methodologies brought to bear on those subjects deemed relevant, and to the objectives or goals of the entire enterprise. At the same time, these two venerable institutions intersect in a multitude of ways. When they do meet, however, each does so on its own terms, or... from its own perspective. From the scientist’s standpoint, law very often suggests hypotheses of interest or permits the application of hypotheses to new and salient contexts. From the law’s standpoint, science very often supplies the brute facts integral to legal decision making or policy formation or creates new matters that legal doctrine must address. In short, when law and science intersect, the two institutions continue to maintain their separate methods and objectives, with each eyeing the other for its own purposes.<sup>155</sup>

There are, naturally, many differences between law and science, for they have different purposes and goals. The two disciplines differ considerably in the degree of certainty with which they are required to formulate their conclusions. The search for ‘truth’, it is generally agreed, does not serve the same aims for the two disciplines, and may not be subject to the same constraints and requirements.<sup>156</sup> Haack notes the

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<sup>153</sup> G. Edmond, ‘Science in Court: Negotiating the Meaning of a “Scientific” Experiment During a Murder Trial and Some Limits to Legal Deconstruction for the Public Understanding of Law and Science’ (1998) 20 Sydney Law Review 361, 361

<sup>154</sup> Berger and Solan (n 3) 847

<sup>155</sup> Faigman et al, ‘Fitting Science..’ (n 6) 360

<sup>156</sup> National Research Council, *A Convergence of Science and the Law: A Summary Report of the First Meeting of the Science, Technology and Law Panel* (National Academies Press 2001) 1-2

‘real tensions between the goals and values of scientific enterprise and the culture of the law.’<sup>157</sup> She cites some salient divergences, for example:

- science is investigative while the culture of the legal system is adversarial
- science searches for general principles while the legal focus is on particular cases;
- the scientific enterprise is pervasively ‘fallibilist’ (open to revision in the light of new evidence), while the law aims to arrive at prompt and final resolutions;
- science pushes for innovation while the law focuses on precedent;
- the aspirations of science are essentially theoretical while the law is inevitably oriented to policy;
- the scientific system is informal, problem-oriented and pragmatic, while the legal system relies on formal rules and procedures.

The crucial need is not, of course, for them to be similar (for each discipline has its own unique task to perform). The crucial need is only for both disciplines to be able to identify a realistic approach to their common task in the courtroom, and to develop a shared perspective about achieving this. This reconciliation, however, requires both disciplines to at least develop some basic mutual understanding of each other’s goals and methods, and to modulate their approach when working together. It is imperative for the law to develop a more realistic and sophisticated understanding about science and the scientific method. The law needs a clearer realisation that no claim asserted as ‘scientific’ will have any absolute claim to conclusive certainty, no matter how robust the discipline, or how eminent the expert who asserts it. The use of research can

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<sup>157</sup> S Haack, *Evidence Matters: Science, Truth and Proof in the Law* (Cambridge University Press 2014) 90

usually suggest the truth (to varying degrees of certainty), but cannot ensure truth.<sup>158</sup>

At the same time, more accountability must also be required of scientists who act as expert witnesses. Juries and courts can find science and statistics very persuasive, and this calls for responsible communication on part of scientific experts. It is thus crucially important for scientists to be more transparent about the subjectivity in their testimony where it exists, and to recognise how the objectives that drive the legal enquiry are very different to those that drive scientific enquiry.

### **1.1 Magnified impact on disease litigation**

The differences between science and the law have been particularly prone to cause difficulties in disease litigation.<sup>159</sup> There has been, over the last few decades, an explosion in negligence claims for diseases allegedly caused by toxic substances, and establishing causation under existing legal principles has become a fraught and often incoherent exercise. The thorny issue of disease causation is one that courts can no longer afford to ignore. This frequently requires that scientific (e.g. medical and epidemiological) evidence be adduced to aid the causal assessment.

As Justice Jay notes in a recent extrajudicial lecture:

*Most legal problems touching on the world of science in the civil law tend to arise in connection with medical and related issues, by which I mean issues which touch on the human body and mind. We lawyers are in the business not of proving the theory of evolution...but in arguing about and deciding whether a claimant's personal injuries should be compensated in a court of law. It is in the rough and tumble of this work*

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<sup>158</sup> A Wilson, 'Away From the Numbers: Opinion in the Court of Appeal' (2011) 75(6) Journal of Criminal Law 503, 503

<sup>159</sup> For a discussion of the difficulties that arise due to differing perspectives on medical causation see Faigman et al 'Fitting Science..' (n 6) 360-362 and 366-370

*that the law often has to rely heavily on scientific opinion, and in doing so needs to understand its methodology, the evidence it uses, and the standards it typically applies.*<sup>160</sup>

The specific scientific issues and complexities surrounding disease causation will be explored in greater detail in Chapter 3. However, it is important to note at the outset of the discussion about science that disease causation is even more susceptible to problematic legal applications of science, because disease causation is one of the most uncertain areas of science, and because there is a considerable difference between approaches taken towards causation in medicine and in the law.<sup>161</sup>

## **1.2: Unrealistic legal expectations from science:**

*‘...I would suggest that nothing is regarded as proven in science in the sense in which that term is understood and deployed in the law... science never rests on its laurels; proof in science is elusive as the grapes of Tantalus.’<sup>162</sup>*

*-Justice Jay*

“Mention science to lawyers” one commentator notes, “and their minds become besieged with chaotic images of schoolroom antics in a Chemistry class on a Friday afternoon”.<sup>163</sup> Judges on the whole, Faigman believes, have very little training in, knowledge of, or inclination to learn science.<sup>164</sup> Partly, this divide is a product of the law’s unrealistic expectations about the power of science.

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<sup>160</sup> Sir Robert Jay, ‘The Richard Davies QC Memorial Lecture 2015: “Standards of Proof in Law and Science: Distinctions Without a Difference?”’ (2016) *Journal of Personal Injury Law* 1, 2-3

<sup>161</sup> R Goldberg, ‘Using Scientific Evidence to Resolve Causation problems in Product Liability: UK, US and French Experiences’ in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 150

<sup>162</sup> Sir Robert Jay (n 16) 2

<sup>163</sup> K Soar, ‘A Probabilistic Theory of Legal Causation’ (1996) *UCL Jurisprudence Review* 132, 132

<sup>164</sup> D Faigman, ‘Judges as “Amateur Scientists” ’ (2006) 86 *Boston University Law Review* 1207, 1207

Byers emphasises that the misconceived illusion of absolute certainty in science has had noxious consequences for both societies and individuals.<sup>165</sup> The lay understanding of science, he notes, is too simplistic and inadequate: it is important to develop a more sophisticated understanding of what science is, and what it can and cannot do for us.<sup>166</sup> The law is no exception. In general, lawyers turn to science in the hope that scientists will be able to provide certainty to the legal enquiry, and will be able to answer the very black-and-white (and often simplistic) questions the law asks. This is problematic because science is intrinsically uncertain, and based on research methods that inevitably possess limitations and flaws<sup>167</sup> (anyone who has checked a weather forecast knows this basic lesson, observes Faigman<sup>168</sup>). The singling out of epidemiology for such judicial criticism, on the grounds that it can never conclusively prove causation, suggests a judicial belief that conclusive proof, free of methodological and epistemic caveats, is possible in most other scientific disciplines. There is a yawning disconnect, argue Dawid et al, between how the law defines expert proof and the ability of science validly to supply such proof.<sup>169</sup> Yet the law continues to ‘construct legal doctrines, either with utter disregard for what scientists might be able to say about the facts relevant to those doctrines, or in light of a folk understanding of the science that might bear on those relevant facts.’<sup>170</sup> Courts regularly, and in the face of contrary evidence, prefer the scientifically naïve view that scientific findings are categorical and certain.<sup>171</sup>

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<sup>165</sup>W Byers, *The Blind Spot: Science and the Crisis of Uncertainty* ((Princeton University Press 2011) vii-viii

<sup>166</sup> *ibid*, viii

<sup>167</sup> Faigman ‘Amateur Scientists..’ (n 20) 1225

<sup>168</sup> D Faigman, *Constitutional Fictions: A Unified Theory of Constitutional Facts* (Oxford University Press 2008) 162

<sup>169</sup> Faigman et al, ‘Fitting Science..’ (n 6) 381

<sup>170</sup> *ibid* 381

<sup>171</sup> Faigman *Constitutional Fictions* (n 24) 162

Many academic commentators note that while the sciences are familiar and comfortable with uncertainties and undetermined aspects of evidence, ‘legal systems demonstrate discomfort with uncertainty. The law operates as though there is *one* correct explanation to be discovered.’<sup>172</sup> The idealised legal view of science appears to operate on the ‘myth of scientific certitude’,<sup>173</sup> which refers to the idea that scientific disciplines are defined by laws drawn from simple mathematical relationships with apparently universal applicability. Broadbent attributes this to a phenomenon he terms the ‘long shadow of physics’<sup>174</sup>: the belief that physics has fixed, universal, mathematically precise laws that are capable of explaining and predicting all observed physical events. Unfortunately, as section 1.3 below will discuss, this illusion of certainty and predictability in the laws of physics has also now been shattered by twentieth century discoveries Burk points out: “The law seems to exhibit an odd sort of love-hate relationship with matters of complex science: although the legal community is reluctant to grapple with the substance of such matters, it apparently yearns for the ‘certainty’ that science can provide.”<sup>175</sup> Courtroom situations are particularly designed to exaggerate the mistrust between the two disciplines. A report of the National Research Council in the US observes that science and technology seek knowledge through an open-ended search for expanded understanding, and scientific conclusions are subject to revision, while the law demands definite findings of fact at given points in time. However, as the report points out, ‘when the two disciplines encounter each other in the courtroom, the differences between them are magnified. The legal tradition of adversarial

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<sup>172</sup> C Willmore, ‘Codes of Practice: Communicating Between Science and Law’ in M. Freeman and H. Reece (eds), *Science in Court* (Ashgate 1998) 40

<sup>173</sup> Faigman, *Constitutional Fictions* (n 24) 162

<sup>174</sup> A Broadbent, *Philosophy of Epidemiology* (Palgrave Macmillan 2013) 142-143

<sup>175</sup> DL Burk, ‘DNA Identification: Possibilities and Pitfalls Revisited’ (1990) 31 *Jurimetrics* 53, 67

proceedings necessarily contrasts with the generally more ‘co-operative ethic of science.’<sup>176</sup> Scientists tend to express their conclusions much more tentatively than lawyers want them to. This can cause much mutual frustration in courtrooms: for scientists (when, for example, they are pushed to frame their evidence in ways that they do not fit with the science), and for lawyers (when scientists do not provide the unambiguous, black-and-white answers the law wants, and thinks they can provide).<sup>177</sup> The reasons behind this scientific urge to identify flaws in the data will be re-visited in section 3, when we discuss the philosophy of science, particularly the influence of Popper’s view of ‘falsification’ as the goal of scientific thinking (section 3.2.2).

There are signs that the law may be starting, at long last, to recognise this problem. In his recent thoughtful Richard Davies QC Memorial Lecture about the problems in the legal application of scientific evidence, Justice Robert Jay concedes that the law ‘may be based on a deterministic view of the natural world.’<sup>178</sup> Many members of the judiciary, Justice Jay acknowledges, believe that science deals in the currency of objective, irrefutable proof.<sup>179</sup> This leads to the idealised view of science as a discipline that is able to provide fixed and conclusive findings, and the value attached to different forms of scientific testimony depends on the extent to which they are able to conform to this idealised view.

This has led to false dichotomies about science in legal minds, reflected in a tendency to either over-value or devalue evidence from different disciplines. Some sciences

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<sup>176</sup> National Research Council (n 12) 1

<sup>177</sup> Popper (n 4) 57-72

<sup>178</sup> Jay (n 16) 3

<sup>179</sup> *ibid*



that use laboratory and/or 'gold-standard' randomised control methods (often referred to as 'hard' sciences, such *physics, chemistry, and biology/biomedical research*) are effectively treated as certain and conclusive. In contrast, evidence from other sciences that do not formulate universal laws or use laboratory-based methods has little value in the eyes of the law. UK courts often point to the probabilistic reasoning of epidemiological evidence as proof of its lack of epistemic validity and reliability.<sup>180</sup> The devaluation of epidemiology reflects the judicial belief that proof absolute is possible in other sciences: an idea derived from the perceived determinism of sciences such as physics.

Determinism is founded on the outdated belief that there is, as Professor Hawking puts it, 'a one to one correspondence between initial states and final states'<sup>181</sup>: if you know the state of the universe at some time in the past, you can predict it in the future; and if you know its state in the future, you can calculate what it must have been in the past.' However, as scientific discovery expands, scientists themselves are beginning to question this archaic distinction between hard and soft sciences.<sup>182</sup> As 'hard' sciences become more uncertain about ideas they once thought established beyond doubt, and as 'soft' sciences' get better at using more rigorous scientific methods, all sciences appear to be converging towards probabilistic models of reality. Physicists are encountering evidence suggesting that even events in the physical world cannot be fully measured or predicted due to significant unknown, random, or untestable variables. Newer discoveries, and modern research, rather than removing

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<sup>180</sup> See earlier, Chapter 1 (section 1.2) and Chapter 5 (section 2)

<sup>181</sup> S Hawking, 'Does God Play Dice?' (1999) <<http://www.hawking.org.uk/does-god-play-dice.html>> accessed 15 March 2016

<sup>182</sup> P. Campbell, 'Editorial: In Praise of the Soft Sciences' (Nature, Vol 435, 22 June 2005) <<http://www.nature.com/nature/journal/v435/n7045/full/4351003a.html>> accessed 15 March 2016

uncertainties, appears to be increasing them. Probabilistic reasoning appears to be an increasingly universal aspect of all sciences.

It appears that epidemiology, for UK courts, falls within the second category, with frequent judicial references to its lack of experimental methodology, and the fact that it only studies populations and the mistaken view that it deals only in ‘naked statistics.’<sup>183</sup> But do the ‘hard’ sciences do better at providing the security and certainty that the law appears to yearn for? We explore, as an example, the field of physics. Later, in chapters 3 (section 2) and 5 (section 4.2), we will also examine certainty in the field of medicine (as courts usually tend to view both physics and biomedicine as ‘hard’ sciences), in order to explore this question.

### ***1.3 Hard and soft sciences: the ‘long shadow’<sup>184</sup> of physics***

*God does not play dice (with the universe).*

-Albert Einstein, c. 1944

*Not only does God definitely play dice, but He sometimes confuses us by throwing them where they can't be seen.*<sup>185</sup>

-Stephen Hawking, 1999

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<sup>183</sup> C McIvor, ‘Debunking Some Judicial Myths About Epidemiology and its Relevance to UK Tort Law’ (2013) 21 Medical Law Review 553, 553-554

<sup>184</sup> A Broadbent (n 30) 129

<sup>185</sup> For a discussion of both these scientific viewpoints, and the attachment to myths of scientific determinism, see S. Hawking (n37)

This distinction between hard and soft sciences arose around 200 years ago from the world of science, initially as a way of distinguishing between natural and social sciences. The traditional underlying assumption at the time was that sciences are arranged in a hierarchy, with developed natural sciences such as physics at the top, and social sciences such as sociology at the bottom.<sup>186</sup> The traditional belief is that hard sciences are characterised by rigour and objectivity, in contrast to soft sciences.

There are, of course, differences between the many different disciplines that call themselves ‘science’. Some fields of science are more advanced or “mature,” and others so undeveloped and speculative that one would ‘hesitate to call them sciences’ at all’.<sup>187</sup> Examples of disciplines that are frequently accused of masquerading as ‘sciences’ on the basis of no or unacceptably weak evidence include homeopathy and chiropractic.<sup>188</sup> This thesis does not suggest that all assertions that call themselves ‘scientific’ should be accorded equal weight: quite the contrary. Nuance and a critical approach is crucial when assessing scientific evidence. This author only argues that the assessment of science should be based on more accurate criteria, which is only possible if lawyers have a more accurate understanding of science and its methods. It is dangerous, it is submitted, to value sciences depending on how much they promise certainty: such promises often turn out to be either illusory, or, more rarely, disingenuous.

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<sup>186</sup> S Cole, ‘The Hierarchy of the Sciences?’ (1983) 89 *American Journal of Sociology* 111, 111

<sup>187</sup> S Haack, *Evidence Matters* (n13) 87

<sup>188</sup> See, for example, P. Ingraham, ‘Overview of Chiropractic’ (Science-Based Medicine) <<https://www.sciencebasedmedicine.org/reference/chiropractic>> accessed 24 August 2016; E Ernst, ‘There is no Scientific Case for Homeopathy: The Debate is Over’ (The Guardian, 12 March 2015) <<https://www.theguardian.com/commentisfree/2015/mar/12/no-scientific-case-homeopathy-remedies-pharmacists-placebos>> accessed 03 September 2016

As Haack<sup>189</sup> points out, the core business of science is inquiry: and scientific inquiry is tentative and fallibilist. Its core values are intellectual honesty and willingness to share evidence; and its procedures are problem-oriented and pragmatic. Thus any attempt to assess the extent to which a discipline is ‘scientific’ must, most importantly, examine the extent to which it adheres to these principles. *The short-sighted hierarchy of sciences based on certainty fails to take account of insurmountable differences that arise due to the nature of the subject matter examined by different sciences.* It is time for the law to abandon outdated and superficial criteria when assessing scientific disciplines, such as whether the discipline formulates mathematical laws or whether its conclusions were tested in a laboratory, and to develop a more sophisticated understanding of what constitutes ‘good’ science. There are many complex reasons why experimental, laboratory methods are more appropriate for some sciences (such as physics) than for others (such as epidemiology): reasons that have little to do with lack of objectivity or robustness.

The legal tendency to sometimes devalue evidence from so-called soft sciences is based in poor understanding of their epistemic value as well as of the inherent limitations of different fields of study. *Even* if we were to accept that physics offers universal, irrevocable laws, most sciences apart from physics do not study fixed states of matter in the physical world, and it is thus unfair to measure the worth of sciences which study more diverse and dynamic systems in the currency of ‘certainty’ and ‘determinism’. How, for example, can the incredible variability and complexity of human behavior ever be captured within fixed, predictable laws and proven

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<sup>189</sup> S Haack, *Evidence Matters* (n 13) 90

conclusively in a laboratory in the same way that the velocity of a pebble can be demonstrated to be dependent on its mass? Rosenberg similarly points out that the science of biology studies much more complex, adaptable and diverse living systems, and despite many efforts by biologists in the early days of scientific determinism it has proved impossible to identify universal, irrevocable biological laws similar to Newtonian physical laws.<sup>190</sup>

Further, scientists themselves are increasingly beginning to question this hierarchy, as greater doubt creeps in about the certainty and stability of the 'hard' sciences, and social sciences examine their theories with increasing rigour and better methodologies.<sup>191</sup> The law, however, remains rather spellbound by this hierarchy, and continues to operate on a binary view of the scientific world. Lawyers often tend to view different disciplines as belonging to one camp or the other (soft or hard), rather than on a continuum.<sup>192</sup> American courts indicated in *Frye*,<sup>193</sup> for example, that the admissibility test for scientific evidence only applied to hard sciences (a rule which held until *Daubert v Merrell Dow Pharmaceuticals, Inc*<sup>194</sup> subsequently clarified that the definition of science covers both categories of sciences).

Newton's discovery of laws of motion in 1687, first published in the *Principia Mathematica*, stated that the motion of all objects was governed by a small number of simple, mathematically expressible and perfectly exceptionless laws.<sup>195</sup> These laws were deterministic: given the position of the planets at any one time at all, the

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<sup>190</sup> A Rosenberg, *Philosophy of Science: A Contemporary Introduction* (Routledge, 2005) 8

<sup>191</sup> E.g. P. Campbell, 'Editorial' (n 38)

<sup>192</sup> R Slovenko, *Psychiatry in Law/Law in Psychiatry* (Brunner-Routledge 2002) 46

<sup>193</sup> *Frye v United States* 293 F 1013 (1923)

<sup>194</sup> 509 US 579 (1993)

<sup>195</sup> I Newton, *Principia Mathematica* (cited from Rosenberg, *Philosophy* (n 46) 8-9

physicist could calculate their position at any past time and any future time.<sup>196</sup>

Newton's theory, which viewed that space and time are absolute, laid the foundations for the idea, first articulated by the French scientist Laplace, that science was a fixed, deterministic inquiry: scientific determinism became an achievable goal for a period. This idea, as Professor Stephen Hawking points out, remained the official dogma throughout the 19<sup>th</sup> century.<sup>197</sup>

However, twentieth century developments in physics and the mathematics, such as the discoveries of quantum mechanics, have shaken these Newtonian beliefs. The formulation of Heisenberg's Uncertainty Principle,<sup>198</sup> published in 1926, first pointed out that it is impossible to accurately measure both the position and the speed of a particle: an insurmountable obstacle to being able to fully measure and predict the natural world. If the Uncertainty Principle set limits on how much it was possible for scientists to know even about the present state of the world let alone about the future<sup>199</sup>, this uncertainty was compounded several times by the subsequent, more recent discovery of black holes.<sup>200</sup> Professor Hawking describes the impact of the discovery of black holes on determinism: 'even this limited predictability disappeared, when the effects of black holes were taken into account. The loss of particles and information down black holes meant that the particles that came out were random. One could calculate probabilities, but one could not make any definite predictions.'

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<sup>196</sup> Rosenberg (n 46) 8-9

<sup>197</sup> Hawking 'Dice' (n 37)

<sup>198</sup> W Heisenberg (1926) cited from Hawking (ibid)

<sup>199</sup> Furuta A, 'One Thing is Certain: Heisenberg's Uncertainty Principle is Not Dead' (Scientific American, 8 March 2012) <<http://www.scientificamerican.com/article/heisenbergs-uncertainty-principle-is-not-dead>> accessed 25 April 2016

<sup>200</sup> NI Shakura and RA Sunyaev, 'Black Holes in Binary Systems' (1973) 24 *Astronomy and Astrophysics* 337, 337-355; SW Hawking, 'Particle Creation by Black Holes' (1975) 43 *Communications in Mathematical Physics* 199, 199-220

These twentieth century discoveries now establish that at the level of sub-atomic matter , there are no universal or exceptionless laws. The laws seem to be inescapably indeterministic so that ‘we appear to be fundamentally forbidden from deriving detailed specific predictions about all molecular and supramolecular events.’<sup>201</sup> This indeterminism is not limited just to physics. Byers<sup>202</sup> uses the physiological blind spot from biology as a metaphor to demonstrate how our knowledge is necessarily always incomplete, as all perception is ultimately filtered through human brain and human senses. The blind spot refers to the place in the visual field that corresponds to the lack of light-detecting photoreceptor cells on the optic disc of the retina where the optic nerve passes through it. Since there are no cells to detect light on the optic disc, explains Byers, a part of the field of vision is not normally perceived, but the brain fills in with surrounding detail and with information from the other eye, so that we are unaware of the blind spot: ‘It seems incredible to us that our perception is incomplete in this way, and goes against our conviction that the world we perceive is coherent and complete.’

Rosenberg points out that these recent discoveries suggest a conclusion even beyond simply stating that we cannot things with certainty: discoveries in quantum mechanics are leading physicists to the idea that, ‘at the fundamental level, the principle of same cause, same effect, is invariably violated.’<sup>203</sup> Sir Stephen Hawking sums up the essential uncertainty of the scientific quest, and the futility of the yearning for conclusive proof in science:

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<sup>201</sup> S Kauffmann, *At Home in the Universe* (Oxford University Press 1995) 17

<sup>202</sup> Byers (n 21) 2

<sup>203</sup> Rosenberg (n 46) 8

*'It seems that even God is bound by the Uncertainty Principle, and cannot know both the position, and the speed, of a particle. So God does play dice with the universe. All the evidence points to Him being an inveterate gambler, who throws the dice on every possible occasion... Thus, the future of the universe is not completely determined by the laws of science, and its present state, as Laplace thought. God still has a few tricks up his sleeve.'*<sup>204</sup>

It is time, perhaps, to let the sun set on the 'long shadow' of physics.

#### ***1.4 The implications for disease causation***

Developments in the sciences are often interconnected, and thus events in the world of physics have import for our discussion about scientific evidence about disease causation. Rosenberg points out, for example, that the relatively brief spectre of scientific determinism of Newtonian mechanics for a while raised the specter of determinism in human behavior and biology as well.<sup>205</sup> Biological scientists, during the heydeys of scientific determinism, studied the hypothesis that the brain and humans are no more than complex collections of molecules or matter: aiming to identify how these 'collections' might behave in accordance with self-same laws.<sup>206</sup> However, the realm of the biological proved to be beyond the reach of the physical, and beyond the reach of Newtonian determinism: biological systems display a level of complexity, diversity and adaptation<sup>207</sup> that makes it impossible to identify fixed, universal laws that govern their operation.

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<sup>204</sup> S. Hawking 'Dice' (n 37)

<sup>205</sup> Rosenberg (n 46) 8

<sup>206</sup> *ibid* 8-9

<sup>207</sup> *ibid* 8-9



The judicial reluctance to accept that epidemiological findings can be good, if probabilistic, evidence for specific causation seems rooted in the belief that a ‘good’ science is one that can offer certain, unequivocal answers. However, as the above section has argued, the more science progresses, the further it appears to be moving away from such ideas of certainty and proof: probabilistic models seem here to stay, at least for the conceivable future. This is even more important in disease causation, where the chances of identifying causal relationships that are certain and linear is even more remote than in physics. If this goal of ‘same cause, same effect’ is now no longer even believed to be true in physics, then to expect to find such laws in biological realms, with their incredible ‘complexity, diversity and adaptation’, as noted earlier, is a doomed quest.

***SECTION 2: Legal errors stemming from myths of scientific certainty: extreme attitudes towards scientific evidence***

This thesis argues that the legal failure to recognise the probabilism inherent in almost *all* sciences has led to unfair scepticism towards epidemiological evidence, on the grounds that it cannot provide certainty about specific causation. The attitude towards epidemiology is in stark contrast with the uncritical acceptance accorded to many other forms of scientific testimony in other legal contexts. The use of forensic evidence in criminal law is an example.

The absurd result of this dichotomous judicial attitude is that probabilistic evidence from epidemiology, which is much more transparent about its limitations and error rates, is rejected in civil litigation, which imposes a lower burden of proof (i.e., proof

on the balance of probabilities). On the other hand, subjective forensic evidence such as fingerprint and eyewitness testimony is given an inflated role in decision-making in criminal trials, which impose a higher standard of proof (i.e. beyond reasonable doubt). Judicial attitudes towards science can fluctuate between extremes of excessive deference towards scientific findings or complete dismissal of their validity for legal purposes. Both extremes can lead to errors, and the case of *R v Sally Clark*<sup>208</sup> (discussed in section 2.2 below) illustrates the tragic consequences of this.

The dichotomy towards scientific evidence in UK law is analogous to Haack's analysis of the misconceptions about science. Professor Haack<sup>209</sup> classifies confusions about the capabilities of science into two types, which she terms the 'scientific' and the 'anti-scientific' approaches: the former refers to a tendency to display an 'exaggerated deference towards science' and an 'excessive readiness to accept as authoritative' any claim made by the sciences, while the anti-scientific view is characterized by an exaggerated kind of suspicion of science, 'an excessive readiness to accept every kind of criticism of science and its practitioners as undermining its pretensions to tell us how the world is.'

The contrast between the near-hostile judicial approach towards epidemiology in UK civil law (on the grounds that such evidence is subject to limitations), and the over-valuation of forensic testimony to secure convictions in criminal law (despite its limitations) is visible evidence of this. An associated problem is that courts are often unduly swayed by the perceived status of the 'expert', and the confidence with which

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<sup>208</sup> *R v Sally Clark* (No 2) [2003] EWCA Crim 1020

<sup>209</sup> S Haack, *Defending Science- Within Reason* (Prometheus Books 2007) 17-18

they state their assertions, rather than the scientific merit of their claim.<sup>210</sup> Complete and uncritical acceptance of any evidence convincingly presented by an eminent expert (especially a medical expert)<sup>211</sup> can cause scientific and statistical testimony to be given far too much weight in legal decision-making.

***Section 2.1: The dichotomous attitudes towards scientific evidence in civil and criminal law:***

The problems and errors in the legal applications of forensic scientific testimony are particularly well documented, both in the UK and in other jurisdictions such as the US.<sup>212</sup> The increasing use of scientific evidence in fact-finding poses a challenge to the traditional principles, procedures and evidentiary rules of the criminal trial.<sup>213</sup> Concerns are expressed repeatedly about the miscarriages of justice that may have resulted from the lack of legal concern about the subjectivity inherent in many forms of forensic testimony.<sup>214</sup> The U.S. National Institute of Justice notes that ‘several of the forensic sciences (such as handwriting analysis, fingerprints, firearms identification, bite marks, microscopic hair comparisons, and voiceprints) (have a) purported lack of a convincing scientific foundation...’<sup>215</sup>

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<sup>210</sup> See G Edmond, ‘Legal versus Non-Legal Approaches to Forensic Science Evidence’ (2016) 20 International Journal of Evidence & Proof 3, 12

<sup>211</sup> *ibid* 3-10

<sup>212</sup> outlined in an article by G Edmond, S Cole, E Cunliffe and A Roberts, ‘Admissibility Compared: The Reception of Incriminating Expert Evidence (i.e., Forensic Science) in Four Adversarial Jurisdictions (2013) 3 *University of Denver Criminal Law Review* 31-109

<sup>213</sup> O Sallavaci, *The Impact of Scientific Evidence on the Criminal Trial* (Routledge 2014) 3

<sup>214</sup> E.g. see Edmond, ‘Legal versus Non-Legal..’ (n 66)

<sup>215</sup> D McClure, ‘Focus Group on Scientific and Forensic Evidence in the Courtroom’ (June 25 2007) (National Institute of Justice, Washington DC) 6  
<<https://www.ncjrs.gov/pdffiles1/nij/grants/220692.pdf>> accessed 20 February 2016

However, this message appears to have been slow to have filter through to English law, where latent fingerprint evidence, for example, is routinely presented, against the best scientific advice, as positive identification.<sup>216</sup> Many straightforward accident claims' concurs Justice Jay, 'are resolved solely on the basis of eyewitness evidence, which is almost always flawed because human memory is so unreliable. No one would claim this resolution to be scientific.'<sup>217</sup>

Commentators repeatedly point out the unfair bias towards forensic scientific testimony. Edmond notes, for example, that in contrast to courts' rejection of expert opinion on many other matters such as psychology, they have been highly receptive to the incriminating opinions of forensic analysts.<sup>218</sup> He questions whether conventional admissibility standards, even in conjunction with trial safeguards, provide jurors and judges with the kinds of information required to rationally assess much of the incriminating expert opinion evidence routinely presented in criminal proceedings.<sup>219</sup>

Heffernan and Coen<sup>220</sup> raise concerns that even though scientific evidence is seen as a bulwark against traditional causes of miscarriage of justice such as fabricated testimony and coerced confessions, courts may have failed to spot that it is not infalliable: a failure that has ironically led to its own miscarriages of justice.

Examples abound about the tendency to secure convictions in criminal law, without any recognition of the subjectivity of such evidence. In *R v Dallagher*,<sup>221</sup> for example, an appellant was convicted for murder, a decision that was substantially based on the

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<sup>216</sup> Edmond, 'Legal versus Non-Legal' (n 66) 5

<sup>217</sup> Jay (n 16) 3

<sup>218</sup> Edmond, 'Legal versus Non-Legal' (n 66) 7

<sup>219</sup> *ibid* 3

<sup>220</sup> L Heffernan and M Coen, 'The Reliability of Expert Evidence' (2009) 73 *Journal of Criminal Law* 488, 488

<sup>221</sup> [2002] EWCA Crim 1903

relatively new technique of ear-print evidence. This decision was subsequently quashed because fresh evidence raised doubts about the validity of ear-printing as a forensic scientific technique. Heffernan and Coen<sup>222</sup> cite high-profile cases such as *Sally Clark* (discussed in more detail in section 2.2 below) and *Barry George*<sup>223</sup> as other examples.

There are signs that courts are more recently beginning to pay more heed to this problem (as seen, for example, *R v T*,<sup>224</sup> where the Court of Appeal addressed questions of how forensic scientists and other expert witnesses should present their evidence in court, and what kinds and quality of data experts can properly draw on in formulating their conclusions). Redmayne, Roberts et al.<sup>225</sup> see this judicial willingness to subject the expert evidence adduced in criminal litigation to searching scrutiny as commendable; despite the fact that the Courts' reasoning about the statistical issues was substantively flawed and erroneous (in their view). However, this does not go far enough, and Edmond in a recent article observes that where forensic analysts are believed to possess special skills, aptitude or experience they are almost always allowed to testify.<sup>226</sup>

The case of *R v Sally Clark* (discussed below, section 2.2) is a tragic reminder of the seriousness of the issue, and the severity of the consequences of this 'laissez-faire'<sup>227</sup> approach to scientific evidence in forensic trials.

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<sup>222</sup> Heffernan and Coen (n 76) 488

<sup>223</sup> *R v George (No. 2)* [2007] EWCA Crim 2722

<sup>224</sup> [2010] EWCA Crim 2439

<sup>225</sup> M Redmayne, P Roberts, C Aitken, G Jackson, 'Forensic Science Evidence in Questions' (2011) 5 *Criminal Law Review* 347, 347

<sup>226</sup> G Edmond (n 66) 7

<sup>227</sup> Heffernan and Coen (n 76) 489

## *Section 2.2 Over-valuing or devaluing testimony due to expert 'status'*

Another consequence of the poor understanding about science is that courts can be excessively swayed by the perceived status *and confidence of the expert*. Reliability tends to be inferred from the relatively basic showing by the proponent of the evidence that the witness is a qualified expert.<sup>228</sup> Courts also sometimes fail to differentiate between different scientific fields, and the limits of expert competence. Wilson points out a number of cases where higher academic qualifications in one discipline may permit admission of testimony in another discipline.<sup>229</sup> Cases such as *R v Robb*,<sup>230</sup> where an expert's qualifications in modern languages was seen as sufficient to admit testimony from him in the field of phonetics; and *R v Doheny*,<sup>231</sup> where the prosecution was permitted to admit scientists to address both the biological and mathematical aspects of DNA evidence.

Recognition of a field, Edmond points out, often operates as a proxy for a more informative inquiry into the value of techniques and opinions, a phenomenon which in Edmonds' view, usually favours forensic scientists.<sup>232</sup> The judicial bias towards clinical medicine is another example of this. Testimony from medical practitioners

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<sup>228</sup> *ibid* 489. For admissibility issues more generally, see T. Hodgkinson and M. James, *Expert Evidence: Law and Practice*, (2nd edn, Sweet & Maxwell: London, 2007). Also discussed later at Chapter 5 section 3.1

<sup>229</sup> Adam Wilson, 'Expert Opinion Evidence: the Middle Way' (2009) 73(5) *Journal of Criminal Law* 430, 432

<sup>230</sup> [1991] 93 Cr App R 61 at 165.

<sup>231</sup> [1997] 1 CR App R 369 at 376 and 383.

<sup>232</sup> Edmond (n 66) 8-9

seems to be particularly impressive to courts. This occurs even when practitioners make claims outside their area of expertise or without clarifying how they arrived at their conclusions, as may be recalled from the discussion of *Novartis*<sup>233</sup> in Chapter 1 (section 1), where courts preferred the doctor's testimony over that of the epidemiologist, even though the issue fell directly within the specialism of the latter. The same phenomenon also led to errors in *R v Sally Clark*, discussed below.

*R v Sally Clark*<sup>234</sup> is a disturbing example of the potentially devastating consequences of uncritical over-valuation of scientific evidence presented by 'eminent' experts. The defendant in this case, who had lost two children in early infancy through sudden and unknown means, was accused of having murdered them. The prosecution's case relied significantly (though not exclusively) on the testimony of Professor Roy Meadows, a distinguished paediatrician. Having wrongly dismissed the relevance of genetic and environmental factors to Sudden Infant Death Syndrome (SIDS), he estimated the likelihood of two sudden infant deaths in the same family as one in 73 million, an eventuality likely to occur, he testified, about once in every 100 years and further equated the likelihood to backing a long-odds outsider at the Grand National. Mrs Clark was convicted of murder and imprisoned, partially on the basis of this testimony. However, her conviction was quashed a few years later partly because of new evidence about natural causes that could explain the infants' deaths: evidence that had been withheld from the jury by the expert witness pathologist. Further, the Court of Appeal found the statistical methods and language used by Dr Meadows in his testimony to be questionable (he was, as mentioned, a paediatrician, not a

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<sup>233</sup> [2007] EWCA Civ 1261; [2007] EWCA Civ 1261

<sup>234</sup> *R v Sally Clark* (n 64)

statistician). The Court of Appeal opined that the 1 in 73 million figure was grossly overstated;<sup>235</sup> and further that Professor Meadows' 'graphic reference' to long odds winners at the Grand National was likely to have had a major impact on the minds of the jury. This case is now widely cited as an instance where an expert breached the duties both to give 'objective unbiased opinion' and to testify only in relation to 'matters within his expertise'<sup>236</sup>. The defendant, tragically, never recovered from the experience, developing serious psychiatric problems and alcohol dependency after her release from prison, and died in 2007 from alcohol poisoning.

Heffernan and Coen<sup>237</sup> contrast *Clark* with *R v Cannings*,<sup>238</sup> also involving sudden infant deaths, in which a different kind of flaw rendered the evidence untrustworthy. The decision of the Court of Appeal to quash the appellant's convictions for the murder of her two infant sons was based largely on fresh evidence which suggested that multiple sudden infant deaths within the same family could be caused by genetic factors, a conclusion distinctly at odds with the prevailing expert opinion at trial that the likelihood of murder increased exponentially with the number of deaths within the same family. The Court of Appeal was at pains to stress that the experts had testified in good faith but acknowledged that on the subject of unexplained infant deaths, 'we are still at the frontiers of knowledge'<sup>239</sup> with the consequence that 'what was confidently presented to the jury as virtually overwhelming expert evidence providing

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<sup>235</sup> *ibid* at [178] (Lord Justice Kay)

<sup>236</sup> D Dwyer, 'The Duties of Expert Witnesses of Fact and Opinion' (2003) 7 *International Journal of Evidence and Proof* 264, 267

<sup>237</sup> Heffernan and Coen (n 76) 496

<sup>238</sup> [2004] 1 WLR 2607

<sup>239</sup> *ibid* [178]



the necessary proof that [the infant boys'] deaths resulted from the infliction of deliberate harm, should now be approached with a degree of healthy scepticism.<sup>240</sup>

### ***Section 2.3: Failure to distinguish between fact and opinion testimony***

A further error that frequently arises in legal contexts is the failure to distinguish between factual expert evidence, and more speculative expert opinion. Scientific inference (as section 3, below, will discuss) almost always involves some degree of interpretation from evidence. However, the crucial qualification for an assertion to be called scientific is that it should be based on unbiased empirical evidence: it is this that gives science validity and justificatory power. Unfortunately, scientific expert witnesses (particularly within an adversarial judicial system) can sometimes succumb to pressure to testify to conclusions that are poorly validated by any actual evidence, especially when it comes to 'specific' conclusions about the case at hand. In such circumstances, assertions (for example, about specific causation) become no more than dangerously subjective opinion: dangerous particularly because they masquerade as 'scientific' fact. The Law Commission published a consultation paper on this issue in 2009,<sup>241</sup> recommending an overhaul of the law in this area, and identifying the issue as 'a real, ongoing problem which demands an urgent solution.'<sup>242</sup> The Law Commission Report that followed<sup>243</sup> notes that the common law approach to the

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<sup>240</sup> *ibid* [156]

<sup>241</sup> Law Commission, *The Admissibility of Expert Evidence in Criminal Proceedings in England and Wales: A New Approach to the Determination of Evidentiary Reliability* (Law Com No 190, 2009)

<sup>242</sup> Edmond, 'Legal versus Non-Legal..' (n 66) 4-5

<sup>243</sup> Law Commission, *Expert Evidence in Criminal Proceedings in England and Wales* (Law Com No

admissibility of expert opinion evidence is one of *laissez-faire*,<sup>244</sup> with such evidence being admitted without sufficient regard to whether or not it is sufficiently reliable to be considered by a jury. This, the Law Commission points out, is particularly concerning where the evidence is presented as scientific, and thus more likely to be readily accepted.<sup>245</sup>

The legal demand for ‘individualistic’ evidence causes this problem to arise frequently in disease litigation. The unfortunate judicial tendency to draw watertight distinctions between ‘general’ evidence drawn from populations studies and ‘specific’ evidence about the individual case leads to pressure on experts to pass off as scientific speculative assertions about the specific claimant, even where the only real evidence available is general, as ‘scientific’ testimony. (This is, in fact, one of most significant reasons that epidemiology has been devalued in UK law, as outlined in Chapter 1. The law has tended to discount ‘general’ evidence about causation offered by epidemiology, as it is not specific evidence about the claimant. Courts fail to recognise, however, that assertions about specific causation made by clinicians are often no more than the clinician’s personal opinion, which are (ironically) evidentially backed only by the same ‘*general*’ evidence the court rejected as insufficient when proffered by an epidemiologist. Chapter 5 (section 2.2) will return to this issue of general and specific evidence, and will analyse why this odd legal view that the two forms of evidence are entirely disconnected is scientifically naïve and misconceived.

The lack of ‘fit’ between lawyers’ expectations from science and what science can realistically deliver adds to the danger that courts may, in fact, be privileging the less

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325, 2011)  
<sup>244</sup> *ibid* 1.8  
<sup>245</sup> *ibid* 1.8-1.9

valid and reliable forms of testimony. Haack<sup>246</sup> cautions that the adversarial character of the legal system tends to draw in as witnesses scientists who are ‘marginal’, i.e., more willing than most of their colleagues to give an opinion on the basis of less-than-overwhelming evidence. Moreover, ‘the more often he serves as an expert witness, the more unbudgeably (sic) confident a scientist may become in his opinion.’<sup>247</sup> A legal system that views certainty as a yardstick to measure the probative value of evidence, can very easily end up valuing confidently asserted (albeit less scientifically honest) testimony more than scientifically honest and reliable, but cautiously phrased testimony.

Faigman et al<sup>248</sup> cite the American case of *Zamora v. State*<sup>249</sup> to illustrate misconceptions about science, and this case provides a useful illustration of our point. Here, a defendant under trial for murder attempted to plead the insanity defence, contending that he had killed as a result of ‘involuntary subliminal television intoxication’. This claim was based on the defence argument that television had a noxious effect on sociopathic children. To support this argument, the defence offered two experts. One, a psychologist, wanted to present robust research evidence about the effects of television on adolescents generally, while the other, a psychiatrist, testified that the specific defendant did not know right from wrong when he committed the act. The court preferred the latter psychiatric testimony, but excluded the first psychologist’s testimony, on the ground that this was only ‘general’ evidence, and was not speak about the specific defendant. The defendant was absolved from liability on the basis of the psychiatrist’s testimony. However, the valid general

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<sup>246</sup> S Haack, *Evidence Matters* (n 13) 95

<sup>247</sup> *ibid*

<sup>248</sup> Faigman et al, ‘Fitting Science..’ (n 6) 365

<sup>249</sup> 361 So.2d 776, 779 (Fla. Dist. Ct.App. 1978) (US), cited from Faigman et al, ‘Fitting Science..’ (n 6)

research findings that were being offered by the psychologist constituted, in this case, the scientifically more robust form of evidence than the psychiatrist's personal, subjective opinion. Thus, the more enlightened choice would have been to admit the first testimony for courts to consider as *one* factor in their ultimate decision. To accept as *fact* a piece of subjective testimony just because an expert is more willing to speak to the legal question, even when this involves speculations that have little backing in scientific evidence, can lead to ludicrous and unfair outcomes: especially when this is accompanied by rejection of robust evidence just because the expert is honest about the limits of their data.

This thesis does not intend to deny the utility of expert opinion altogether. Expert opinion, as Cooke stresses, can, under certain circumstances, be a very useful source of data.<sup>250</sup> For example, it is frequently the case that there is no or little available evidence that maps specifically on to the legal question at hand. Where the only option would be subjective decision-making by a court that may have little understanding of related issues, it is far better to seek the assistance of an expert who is at least able to provide an informed opinion,<sup>251</sup> and can extrapolate from what evidence is available (although, of course, this should only be used to inform, rather than decide, the legal outcome). However, it is not the same as expert knowledge: and the consequences of ignoring this distinction may be grave.<sup>252</sup>

Given that opinion evidence may sometimes be vital to the legal inquiry, it is vital to recognise the distinction between fact and opinion, and it assessing how and when it

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<sup>250</sup> RM Cooke, *Experts in Uncertainty: Opinion and Subjective Probability in Science* (Oxford University Press, 1991) 3

<sup>251</sup> This was also reiterated in recent Supreme Court case of *Kennedy v Cordia Services LLP*: [2016] UKSC 6

<sup>252</sup> Cooke (n 106) 3

should be used in decision-making. As Cooke puts it, there is merit in asking if there are ‘better’ or ‘worse’ ways of using expert opinion evidence, rather than allowing great weight to be placed on the uncertain opinion of experts.<sup>253</sup> Expert opinion about the specific case should only be admitted if it can be validly provided.<sup>254</sup>

Further, crucially, opinion testimony must be expressed and understood as being what it is, rather than be falsely presented as an objective demonstrable fact. Transparency on part of experts is vital to the correct use of expert testimony. Courts must be aware that an expert is making a judgment that is based primarily on their own interpretation of previous skill and experience, rather than on the basis of any specific evidence. However, this can only happen when courts cease to devalue probabilistic scientific reasoning, and when they grasp that all scientific conclusions have potential for error. Scientific evidence, as Faigman at all points out, should only be considered as one ‘brick in the wall’<sup>255</sup> of proof: it is not the wall. Scientists must be encouraged, rather than discouraged, to be transparent about the potential for error in their testimony, so that courts can weigh this testimony in a more nuanced fashion. The current legal climate tends to devalue expert witnesses and disciplines (epidemiology is an example) that are transparent about their error rates, and may unconsciously deter experts from being open about the potential for error in their conclusions. Error rates do not negate the utility of scientific evidence, but in fact allow more accurate assessment of how much weight to accord to it. The empirical uncertainties of factual statements, as Faigman asserts, are as important as the statements themselves, and should be part of the legal calculus.<sup>256</sup>

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<sup>253</sup> *ibid*

<sup>254</sup> Faigman, ‘Amateur Scientists’ (n 20) 1207-1208

<sup>255</sup> Faigman et al ‘Fitting Science...’ (n 6) 365

<sup>256</sup> Faigman, *Constitutional Fictions* (n 24) 162

### *Section 2.4: Erroneous legal criteria for reliability of scientific evidence*

Beyond basic studies, Cranor<sup>257</sup> raises the concern that courts may not understand the critical features of scientific reasoning, which causes them to adopt overly simplistic or misconceived indicators of reliability of evidence. This leads to several errors in using scientific evidence. One result of this is the tendency to ‘fetishise evidential hierarchies’.<sup>258</sup> The judicial dismissal of epidemiological evidence about causation on the grounds that much of this derives from observation, rather than experimentation is an example of the law applying over-simplified ‘scientific’ rules to make evaluations of evidence (we will return to discussing the value of epidemiology, and of observational methods in greater detail in Chapter 5, section 2.1).

Case studies is another form of evidence that is often disadvantaged in such an approach, often causing courts to lose out on valuable supporting evidence for or against an inference. As Cranor<sup>259</sup> points out, what can make case studies good evidence about causation is ‘the *analysis* to which they are subjected, and the way *scientists reason about them*. Good case studies rest on a principle of diagnostic or non-deductive reasoning that is essential to all causal judgments.’ The same is true of observational methods, frequently used in epidemiological studies of causation, which have tended to be devalued by UK courts. Chapter 5 (section 2.1) will discuss the

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<sup>257</sup> C Cranor, *Toxic Torts: Science, Law and the Possibility of Justice* (Cambridge University Press 2007) 115

<sup>258</sup> Jay (n 16) 1-3

<sup>259</sup> Cranor, *Toxic Torts* (n 113) 115

utility of observational studies to the study of disease causation, and demonstrate why the UK judicial dismissal of their value is deeply flawed.

This is related to a broader problem, which is that courts often tend to view or make judgments about individual pieces of scientific evidence, rather than viewing it in conjunction with the overall evidence. As an academic article co-authored by a physician and a legal scholar notes,<sup>260</sup> ‘Courts tend to assess separately the reliability of each component of the evidence, rather than assessing the reliability of the “totality of the evidence” including all relevant clinical factors. In doing so, courts fail to take into account the complex inferential process that lies at the heart of clinical medical reasoning.’

This is a frequent problem in jurisdictions such as the US, where courts have greater jurisdiction over admissibility of scientific evidence under the *Daubert* guidelines, and where relevant supporting pieces of evidence may be excluded from consideration altogether, because they were deemed individually inadequate. This violates, as Cranor<sup>261</sup> notes, an important scientific principle, which is to “never throw evidence away” just because it does not provide sufficiently strong support for a theory: put together with other supporting relevant information, it can add significantly to the weight of the best available evidence. This is especially valuable where there would otherwise be no other evidence with which to tackle a question. This points to an ongoing legal failure to fully understand the process of scientific reasoning, which may be even more prevalent in UK courts, with the continued lack of clear guidance or direction to courts about reliability and admissibility issues.

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<sup>260</sup> JP Kassirer and JS Cecil, ‘Inconsistency in Evidentiary Standards for Medical Testimony’ (2002) 28 *Journal of the American Medical Association* 1382, 1384

<sup>261</sup> See Cranor, *Toxic Torts* (n 113) 259-260

### **SECTION 3: How scientists make inferences: scientific reasoning**

The importance of science in today's world, and the regard it holds in modern society, cannot be overstated. Science's centrality to society's welfare marked the twentieth century, both in terms of posing dire threats and promising salvation, and this trend is likely to expand geometrically in the century ahead.<sup>262</sup> To describe any claim as 'scientific' instantly enhances its credibility and respectability. Science is what we use to understand the world and to understand ourselves.<sup>263</sup> Science has come to define, in many people's minds, 'what is real and what is true'.<sup>264</sup> However, it is important to remember that science has no absolute monopoly over 'truth'. As scientists will be first to point out, 'science is not a creed. It was not revealed to man by a superior deity. Science is a product of the human brain, and as such, it is always open to discussion and possible revision... It represents a logical summary of human knowledge, based on human observation and experience, both of which are always of limited range and finite accuracy.'<sup>265</sup>

The belief in scientific certainty, Byers notes, has two aspects: first, the belief that a state of objective certainty exists and second, that scientific activities are methods through which this state can be accessed.<sup>266</sup> The preceding sections of this chapter examined both these ideas, and illustrated that not only is science in its present state

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<sup>262</sup> Faigman, 'Amateur Scientists' (n 20) 1207

<sup>263</sup> Byers (n 21) vii

<sup>264</sup> *ibid* ix

<sup>265</sup> L Brillouin, *Scientific Uncertainty, and Information* (Academic Press 1964) vii

<sup>266</sup> Byers (n 21) vii



unable to access or measure all variables with complete certainty, but also, further, that there are now increasingly fundamental doubts about the existence of absolutely fixed, irrevocable, universal laws of nature.

Although this may sound disappointing to lawyers, this lack of certainty is not a fatal flaw for a scientist. Science is, ultimately, a methodical attempt to understand, quantify, reduce, or manage this uncertainty. Despite uncertainties and fallibility, science and scientific methods have transformed our world in ways that are impossible to enumerate. The assessment of disease causation, and the correct application of epidemiological evidence, would be aided by an accurate judicial grasp of the value of the probabilistic, but empirical, reasoning that science relies on.

### *Section 3.1: What is science*

The UK Science Council defines science as ‘the pursuit of knowledge and understanding of the natural and social world following a systematic methodology based on evidence.’<sup>267</sup> The new Webster’s Dictionary defines ‘scientific’ as ‘pertaining to science, using methods based upon well-established facts, thorough and accurate.’<sup>268</sup> The scientific method, explains Klinkner, observes an empirical problem in a way that will allow inferences to be drawn about that phenomenon using investigative techniques such as observation, experimentation and the formulation and testing of hypotheses.<sup>269</sup>

Bird<sup>270</sup> cites the explanation of science provided by Judge William Overton, in the American case of *McLean v Arkansas Board of Education*, one of many cases where American courts had to decide whether creationism was a science. The court outlined the defining features of science (on the basis of which analysis Judge Overton, incidentally, ultimately rejected the contention that creationism is a “science”). A scientific theory, stated the court, needs to possess the following characteristics:

- It is guided by natural law
- It has to be explanatory by reference to natural law

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<sup>267</sup> Science Council, ‘Our Definition of Science’ (*Science Council*) <<http://sciencecouncil.org/about-us/our-definition-of-science/>> accessed 14 March 2016

<sup>268</sup> R Slovenko, ‘The *Daubert* Sequelae’ (1998) 2 *International Journal of Evidence and Proof* 190, 195

<sup>269</sup> M Klinkner, ‘Forensic Science Expertise for International Criminal Proceedings: an Old Problem, a New Context and a Pragmatic Resolution’ (2009) 13(2) *International Journal of Evidence and Proof* 102,106

<sup>270</sup> A Bird, *Philosophy of Science* (Routledge 1998) 2

- It is testable against the empirical world
- Its conclusions are tentative
- It is falsifiable (Karl Popper<sup>271</sup> viewed this quality of falsifiability as a central feature of science, as we shall see later in section 3.2.2).

Thus, uncertainty and openness to refutation by contradictory evidence, far from being viewed a deficiency, is seen as one of the very defining features of a scientific theory (This is, it must be noted, a crucial point in relation to our discussion about how the legal view of scientific certainty is in stark contrast to the scientist's view of science). Additionally, the explanatory principles of science must be testable.

Anything that cannot be tested, many believe, is simply not within the realm of science.<sup>272</sup> Testability it is also one of the factors that American courts must consider, under Daubert guidelines, when assessing the reliability and admissibility of scientific evidence.<sup>273</sup> The concept of testability is however not the same as asking whether the theory has, in fact, been tested by experimentation. There may sometimes be practical reasons why a hypothesis cannot be tested experimentally, even though it is in theory testable. For example, the effects of a toxin on humans cannot, for obvious ethical reasons, be tested experimentally by administering this to human research subjects. In such situations, the pragmatic scientist will look for evidence through alternative methods such as observation, which even if untested, can still be founded on robust scientific principles. (This will be discussed further in Chapter 5, section 2.2, where we will use this to show that the legal devaluation of epidemiology because it does not use experimental methods to test causal hypotheses is thus unfair. This is not a

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<sup>271</sup> Popper (n 4)

<sup>272</sup> JF Fielder, 'Daubert Can Undermine Psychiatric Testimony' (1997) National Law Journal 8-14.

Cited from R Slovenko, *Psychiatry in Law* (n 48) 46

<sup>273</sup> Slovenko, 'Daubert' (n 124) 193

deficiency of epidemiology, but more commonly of the subject matter it examines in toxic tort and disease litigation).

Over the centuries, philosophers of science have tried to answer questions about what makes scientific findings valid, and to formulate standards that would help identify good scientific explanations. They have considered whether scientific pronouncements ought to be causal, unified, nomological, statistical, deductive, inductive or any combination of these.<sup>274</sup> The following section briefly outlines some schools of thought.

### **Section 3.2: Philosophies of science**

Modern science began to emerge around the 16<sup>th</sup> and 17<sup>th</sup> centuries, when the knowledge demands of emerging technologies (such as artillery and transoceanic navigation) stimulated inquiry into the origins of knowledge.<sup>275</sup> Since then, Kitcher<sup>276</sup> points out that the “scientific faithful” see the sciences as representing the apogee of human achievement, that have, since the seventeenth century, disclosed important truths about the natural world. Scientific ‘truths’ are credited for having enlightened us, by replacing old prejudices and superstitions; and for creating conditions in which people can lead more satisfying lives.<sup>277</sup> Philosophers of science have widely divergent views about how scientific views are shaped, and about the processes by which scientists reason through their conclusions. This section is not an exhaustive or complete account of all the many different philosophies of science (which is outside the scope of this thesis), but is simply a brief description of some influential schools

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<sup>274</sup> Klinkner (n 125) 102

<sup>275</sup> Rothman et al (n 1) 18

<sup>276</sup> P Kitcher, *Science, Truth and Democracy* (Oxford University Press 2001) 3

<sup>277</sup> *ibid* 3

of thought. The purpose of this section is to illustrate the diversity that characterises scientific reasoning, and to identify those aspects that help lawyers better apply scientific evidence. Almost schools of science, despite divergences, appear to agree about the uncertainty in science. Even a conclusion drawn from the most well replicated gold-standard research study cannot provide definitive proof that the conclusion is true. Our capacity to know the ‘truth’ is limited; both by human and natural limitations, and the best that science can do is to find ways of managing and minimizing this uncertainty.

### **3.2.1: Inductivism:**

One of the early explorations of the inductivist scientific method was Francis Bacon’s *Novum Organum*.<sup>278</sup> Induction is the form of reasoning that distinguishes the natural sciences such as chemistry and geology from mathematical subjects such as algebra and geometry, which rely more on deductive reasoning.<sup>279</sup> Inductive reasoning is the process of making generalisations, or inductions, from (specific) observations to general laws of nature.<sup>280</sup> Using inductive reasoning, the scientist reaches a conclusion, or theory, about the subject matter, and the data he or she uses to justify the theory is called the evidence. The strength of justification given by the evidence may vary. It may be very strong, or good, or very weak. Where the evidence is strong, the conclusions have a high degree of probability of being true, and where the evidence is weaker, the conclusions have a lower probability of being true.<sup>281</sup>

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<sup>278</sup> Francis Bacon, *Novum Organum Scientiarum* (1620) cited from Rothman et al (n 1) 18

<sup>279</sup> Bird (n 126) 7

<sup>280</sup> Rothman et al (n 1) 18-19.

<sup>281</sup> Bird (n 126) 7

Deductive reasoning, by contrast, reasons from the general to the particular. Both of these reasoning strategies are used by people in everyday lives, including scientists and by lawyers.

The problem in finding a complete justification for inductive reasoning is that no matter how strongly the evidence is shown to support a hypothesis, the logical, theoretical possibility of the hypothesis being wrong, or superseded by a rival theory with better evidence, can never be ruled out in inductive reasoning. This is because no matter how many times a proposition is tested and shown to be true, it is logically impossible to be absolutely certain we will have the same result next time it is tested.<sup>282</sup> This is called the problem of induction: a problem that has occupied, and continues to occupy, a great many philosophers such as David Hume,<sup>283</sup> John Stuart Mill<sup>284</sup> and Karl Popper<sup>285</sup> since the last two centuries at least.

The 18<sup>th</sup> century Scottish philosopher David Hume pointed out that a causal argument based on previous experience or observation represented nothing more than an assumption that certain events would in the future follow the same pattern as they had in the past. This, Hume pointed out in *A Treatise of Human Nature*,<sup>286</sup> has no logical necessity, and cannot prove causal connections. Causal inference based on a mere association of events constitutes a logical fallacy known as *post hoc ergo propter hoc* (Latin for “after this therefore on account of this”). An example of the erroneous conclusion this could lead to is, for instance, inferring that the crowing of a rooster is

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<sup>282</sup> Also acknowledged by Popper (n 4), a robust supporter of ‘deductivist’ reasoning in science. For a more detailed discussion see Rosenberg (n 46) 121

<sup>283</sup> D Hume, *A Treatise of Human Nature* (first published 1739) (Oxford University Press 2002)

<sup>284</sup> JS Mill, *A System of Logic, Rationative and Inductive* Vol 1 (John W Parker 1843) 437-449

<sup>285</sup> Popper (n 4) 3-7

<sup>286</sup> Hume (n 139)

necessary for the sun to rise because sunrise is always preceded by the crowing.<sup>287</sup>

Observers cannot perceive causal connections, but only a series of events.<sup>288</sup>

Hume's argument has for 250 years been treated as an argument for scepticism about empirical science, for it has been interpreted to mean that all conclusions about scientific laws, and all predictions science makes about future events, are unwarranted, owing to their reliance on induction.<sup>289</sup> However, this, in Rosenberg's<sup>290</sup> view, is a misinterpretation of Hume's point. Hume's own conclusion was different: as a person who acts in the world, he was satisfied that inductive arguments were reasonable. The problem his critique intended to highlight was not that there is no justification for inductive reasoning, but rather we have not yet found the right justification for induction.

Unfortunately, this academic critique of the logical limits of inductive reasoning is something that courts have overstated and misunderstood, when they dismiss epidemiological causal inferences because they are based on observation and inductive reasoning, as we will see later in Chapter 5 (section 2.1). This is ironical, especially given that courts make much of their preference for pragmatic, common-sense approaches to causation. The problem of induction is primarily a problem for scholars of logic, philosophy and epistemology: how to find an appropriate philosophical justification for a reasoning method that we all know to be reasonable, and that works for us in our everyday lives. This does not, however, hinder such knowledge from being invaluable in everyday practical decision-making.

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<sup>287</sup> Rothman et al (n 1) 19

<sup>288</sup> Hume (n 139)

<sup>289</sup> Rosenberg (n 46) 115

<sup>290</sup> *ibid*

As Rothman points out, inductive reasoning, with all its limitations, was a great improvement on prior ways of drawing conclusions about the world, for at least it demanded that a scientist make careful observations of people and nature, rather than appeal to faith, ancient texts, or authorities.<sup>291</sup> Although inductive reasoning can never provide certainty, it can point out possibilities, and further testing of those possibilities constantly leads to great strides in scientific discovery. Edward Jenner's discovery of the smallpox vaccine, which has had such a significant impact on human and public health, is an example of one such discovery made through inductive reasoning.

So far as Hume's assessment of the limits of inductive reasoning is a caution about the fallibility of knowledge, and a general warning against the dangers of too readily accepting any scientific assertion as the 'truth', it is sound criticism. Whilst deductive inferences are deemed true if the premises are accepted as true, inductive inferences are evaluated according to their contextual strengths and are a matter of degree of credibility: they cannot produce certain conclusions.<sup>292</sup>

Conclusions from empirical sciences, unlike in mathematics, can never be proven or disproven with finality. As Bird<sup>293</sup> notes, mathematical sciences do not study the natural world, and rely on deductive, rather than inductive, reasoning. The mathematician's final conclusion (called a theorem) results from a chain of reasoning, each link of which proceeds by logic. This chain of reasoning, or proof of a theorem, does not come by degree: it either establishes the conclusion completely or fails to

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<sup>291</sup> Rothman et al (n 1) 18-19

<sup>292</sup> Klinkner (n 125) 106

<sup>293</sup> Bird (n 126) 7-8



establish it all. Mathematical proof is final, and cannot be refuted by subsequent evidence. As long as the initial premises are true, the theorem is true.<sup>294</sup> Deductive reasoning is not specific to mathematics, however. It simply refers to a form of reasoning in which one begins with general premises and uses these to arrive at a general conclusion.

If all mammals are warm blooded (general premise)

And, horses are mammals (specific premise)

Then it must follow that horses are warm blooded (specific conclusion).

Clinicians regularly employ a combination of inductive and deductive strategies in everyday clinical work, in evidence-based medical models of practice, as do lawyers in the resolution of legal dilemmas. Although inferring specific causation on the basis of general evidence about causation does not represent ‘proof’ in the mathematical sense, because we have no guarantee that the initial premises (i.e. general evidence about causation) are always correct in every instance, they still represent the best evidence we have. As such, they are more likely to be fair than inferences drawn from no evidence at all. Although UK courts have shown awareness that causation cannot be conclusively inferred from associations (as discussed earlier in Chapter 1, section 3.1), they have missed the broader point that almost all scientific facts we accept as given are also ultimately tentative (to a greater or lesser degree).

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<sup>294</sup> *ibid* 7

### 3.2.2: Refutationism

Sir Karl Popper, who was one of the most vocal proponents of ‘refutation’ or ‘falsification’ school, is described as being ‘among the most influential of twentieth-century philosophers of science.’<sup>295</sup> The refutationist philosophy holds that a scientific theory can only be tested by attempting to falsify, or refute, it. Popper’s belief is that scientists should not seek positive evidence in favour of their theory, but should in fact actively seek negative evidence against their scientific hypotheses.<sup>296</sup> Science, he proposes, advances by a process of elimination called “conjecture and refutation.”<sup>297</sup> Scientists form hypotheses based on conjecture and previous experience. Good scientists make predictions from a hypothesis and then compare observations with the predictions. Hypotheses whose predictions agree with the observations are “corroborated” only in the sense that they can continue to be used as explanations of phenomena. At any time, however, they may be refuted by further observations and may be replaced by other hypotheses that are more consistent with other observations.

Popper argues that merely having a good body of empirical data and making generalizations from it can lead to wrong conclusions that can never be tested, and has sometimes led to the growth of pseudo-science (astrology and Freudianism, he believes, are examples of such pseudo-science).<sup>298</sup> The refutationist school sees the

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<sup>295</sup> Rosenberg (n 46) 121

<sup>296</sup> *ibid*

<sup>297</sup> Popper (n 4) 57-72

<sup>298</sup> K Popper, ‘Science: Conjectures and Refutations’ in A Bird and J Ladyman (eds), *Arguing About*

best science as one that constantly and actively seeks possible falsities in the hypothesis under consideration. Criticism of a theory is not seen as a criticism of the person who proposed it<sup>299</sup>, but as the defining characteristic of the scientific approach itself.

Popper's rather extreme stance regarding has generated approval and criticism in equal measure. Rosenberg sums up the rather uninspiring futility of Popper's philosophy thus: 'Good scientific method, and good scientists, should seek only to falsify hypotheses, to find evidence against them, and when they succeed in falsifying, as inevitably they will (until science is "complete"- a state of affairs we wont be able to realise we have attained), scientists should go on to frame new hypotheses and seek their falsification, world without end.'<sup>300</sup> Many scientists point out that taken too far, this can be harmful to science. David Goodstein (Vive provost and distinguished professor of physics at the California Institute of Technology) cautions that The Popperian ideal would also be harmful to science if pursued, and it is crucial for every idea to receive the most vigorous possible advocacy, 'just in case it might be right.'<sup>301</sup> Science, he notes, can be an adversary process, with 'observations and data the tools of combat.'<sup>302</sup> Haack opines that Popperian philosophy- eschewing verifiability, inductive logic, confirmation, supportive evidence, and reliability, and urging scientists to make highly falsifiable conjectures

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*Science* (Routledge 2013) 15

<sup>299</sup> Rothman et al (n 1) 20

<sup>300</sup> Rosenberg (n 46) 121

<sup>301</sup> Goodstein D, 'How Science Works' In Federal Judicial Center' (eds) (2000) 74 Cited from Cranor (n 113) 145

<sup>302</sup> *ibid*

and then test them to destruction- is thoroughly negative, and is a kind of ‘covert scepticism’.<sup>303</sup>

### 3.2.3: Consensus and Naturalism

Some 20<sup>th</sup> century philosophers of science, most notably Thomas Kuhn, dispute the logical empiricist view of science. Kuhn’s ‘historically oriented view of science’ describes science as a rather more creative undertaking, one that changes and develops in significant ways over time.<sup>304</sup> Each natural science begins with a pre-paradigm period, where there is no universally agreed method of research, and then through ‘breakthroughs’ or momentous discoveries, evolves a conceptual paradigm. Kuhn emphasised the significant role played by the scientific community in judging the validity of scientific theories, holding that that the scientific assessment of the evidence may not be as dispassionate as the ideal suggests. When confronted with a refuting observation, Kuhn points out, a scientist faces the choice of rejecting either the validity of the theory being tested or the refutation. Observations that do not fit with a theory may sometimes just be treated as “anomalies” that may eventually be explained. In other instances, however, anomalies may lead to a complete overthrowing of an established scientific doctrine (the overturning of previous ideas in physics due to the discovery of relativity and quantum mechanics, discussed earlier in section 1.3, is an example of this).

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<sup>303</sup> Haack, *Evidence Matters* (n 13) 129

<sup>304</sup> TS Kuhn, *The Structure of Scientific Revolutions* (first published 1962) (4<sup>th</sup> ed University of Chicago Press 2012) 1-9; 10-22; 43-46

The philosophic debate about Kuhn's description has never fully resolved whether Kuhn only meant this as a descriptive account of what has historically happened in the world of science, or whether it was meant to propose what should happen. Thomas Kuhn's basic claim, that theoretical paradigms affect the problems that scientists study and the answers they obtain is accepted by most of the scientific community, even scientific realists.<sup>305</sup> However, Kuhn was criticised for taking this argument too far, because his ideas were sometimes interpreted as a suggestion that science as an irrational process. Others regretfully accept Kuhn's description as a true of much of what passes for scientific activity, but not prescriptive for any good science. Kuhn himself, Rothman notes, was uncertain about how he intended his theory to be read:

Are [my] remarks about scientific development...to be read as descriptions or prescriptions? The answer, of course, is that they should be read in both ways at once. If I have a theory of how and why science works, it must necessarily have implications for the way in which scientists should behave if their work is to flourish.<sup>306</sup>

### 3.2.4: Bayesianism

Reverend Thomas Bayes, in the 18<sup>th</sup> century,<sup>307</sup> put forward a different view of the goal of science. The Bayesian approach represents a constructive attempt to deal with the dilemma that scientific laws and facts should not be treated as known with certainty. The logical problem that the Bayesian school focuses on is that deduction

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<sup>305</sup> DL Faigman, *Constitutional Fictions* (n 24) 24-25

<sup>306</sup> TS Kuhn, 'Reflections on My Critics' in I Lakatos et al (eds) *Criticisms and the Growth of Knowledge* (Cambridge University Press 1970) cited from KJ Rothman, S Greenland, C Poole and TL Lash, 'Causation and Causal Inference' in KJ Rothman, S Greenland and TL Lash (n 1) 21-22

<sup>307</sup> T Bayes 'An Essay Towards Solving a Problem in the Doctrine of Chances' (1764) *Philos Trans R Soc Lond*, 370-418.

has limited scientific validity: following logical rules to make a deductive argument can provide no information about the truth or falsity of a scientific hypothesis unless you can be 100% certain of the truth of the initial premises of the argument. It highlights that scientific conclusions depend heavily on the person who supplied the initial certainties, which may vary across individuals, and thus will always have some degree of subjectivity.<sup>308</sup> The best option therefore is to accept that complete certainty or “truth” is something that we can never find, and even if we find it can never know that we have found it. Therefore, the most rational option in this view is to focus on evaluating knowledge rather than truth; and estimating degrees of certainty or probability that our knowledge may be true. The French mathematician and scientist Pierre Simon de Laplace first give this view an applied statistical format, further refinements of which then culminated in the renowned Bayes theorem.<sup>309</sup>

The Bayesian approach, legal commentators note, can usefully be applied to the assessment of evidence in a trial context. Wilson, for example, draws a number of parallels between Bayesian approaches and forensic investigations.<sup>310</sup> Bayes considers probability as measures of belief based upon knowledge, experience and information regarding the truth of a statement or an event whose truth or falsity is unknown.<sup>311</sup> Probabilities are not states of nature, but states of mind.<sup>312</sup> In assessing which proposition is more probable, scientists attempt to elicit a single figure, the likelihood ratio. Bayes combines prior, or background, information with new information to

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<sup>308</sup> Rothman et al (n 1) 22-24

<sup>309</sup> *ibid*

<sup>310</sup> Wilson (n 14) 504

<sup>311</sup> F Taroni, C Aitken and P Garbolino, ‘DeFinetti’s Subjectivism, the Assessment of Probabilities and the Evaluation of Evidence: A Commentary for Forensic Scientists’ (2001) 41(3) *Science and Justice* 145, 146

<sup>312</sup> *ibid*

provide posterior probabilities. Case-specific facts not only change propositions, but also change the pertinent data.<sup>313</sup>

Not everyone accepts the utility of drawing this distinction between belief and fact, and focusing on belief rather than fact.<sup>314</sup> This philosophy has been criticised for diverting attention away from the classic goals of science, such as the discovery of how the world works, towards states of mind such as probabilities, or degrees of belief.<sup>315</sup> However, Rothman points out that critics fail to recognise the importance of the scientist's state of mind in determining what theories to test and what tests to apply.<sup>316</sup> Most people reason poorly in the face of uncertainty. At the very least, subjective Bayesian philosophy provides a model for reasoning more soundly under uncertainty and, in particular, provides many warnings against being overly certain about one's conclusions (Greenland 1998).<sup>317</sup> The conventional process, Rothman et al contend, is informal, intuitive and ineffable, and therefore not subject to critical scrutiny; 'at its worst, it often amounts to nothing more than the experts announcing that they have seen the evidence and here is how certain they are.'<sup>318</sup> How they have reached these certainties is left unclear, and the process is not transparent: biases and prior prejudices can easily creep into expert judgments. No one, not even an expert, is very good at informally and intuitively formulating certainties that predict facts and future events well. Bayesian methods force the experts to "put their cards on the

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<sup>313</sup> *ibid*, 180

<sup>314</sup> Jay (n 16) 13

<sup>315</sup> Popper (n 4) 133-136; 204-205

<sup>316</sup> Rothman (n 1) 23

<sup>317</sup> S Greenland (2006) 'Bayesian Perspectives for Epidemiological Research I. Foundations and Basic Methods (with comment and reply)' 35 *International Journal of Epidemiology* 765-778

<sup>318</sup> Rothman et al (n 1) 23

table” and specify explicitly the strength of their prior beliefs, why they hold those beliefs, and defend those specifications against arguments and evidence.<sup>319</sup>

### **3.3: Reconciling the philosophies**

As the above discussion shows, there is no universally accepted view of science, nor any universal explanation of how it works. Supporters of the scientific method view science as a continual quest for rationality, through engagement in free inquiry. On an idealised view, scientists engage in a dispassionate, unbiased search for knowledge, show no bias towards their own theory, and resist attempts to hobble investigations for the sake of any moral, political, or religious agenda.<sup>320</sup> However, observers such as Kuhn disagree with this notion of a pure science that stands free of moral, political, and religious values, and contend that there is always some value judgment involved. Further, critics of science argue, there is no objective notion of “the evidence”: decisions about which “scientific conclusions” to accept are always made on the basis of moral or political values.<sup>321</sup> Debates still rage in scientific circles about whether scientific research represents true underlying realities of the natural world or merely represents constructed accounts of observed events. Scientific realism proposes that science represents “truth”: but truth does not imply that scientists can say

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<sup>319</sup> *ibid*

<sup>320</sup> Kitcher (n 132) 3

<sup>321</sup> *ibid* 4



unambiguously or with certainty that the world operates in a particular fashion.<sup>322</sup>

Rather, the world operates in particular ways and science (with greater or lesser precision) endeavours to describe that world. Realists believe that scientific methods provide a lens through which the world can be described, even if only imprecisely.<sup>323</sup>

Byers believes that seeking a single definition or philosophy is a mistake, because this quest assumes that science is monolithic, when in fact it actually carries within it diverse tendencies.<sup>324</sup> Fortunately our task here is much more modest: it is only to identify the fundamental aspects of science that can help us make better use of it in legal settings. The general consensus about science that emerges from all these debates appears to be that the scientific method is simply a set of tools that help us make sense of an uncertain world, by employing reasoning strategies that are not so different from ordinary common-sense reasoning, in addition to, *most centrally*, rigorous verification of empirical facts and evidence. Further, rigid, hierarchical approaches to scientific evidence are flawed: different approaches and forms of evidence work better in different background circumstances. Learning how to use probabilistic evidence is crucial for the proper legal use of science.

#### ***3.4: How scientists make inferences: commonalities between legal and scientific reasoning***

*The whole of science is nothing more than a refinement of everyday thinking. It is for this reason that (the physicist)...cannot proceed without considering critically a much more difficult problem, the problem of analysing the nature of everyday thinking.*

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<sup>322</sup> Faigman, *Constitutional Fictions* (n 24) 23

<sup>323</sup> Faigman, *Constitutional Fictions* (n 24) 24

<sup>324</sup> Byers (n 21) viii

This chapter has highlighted that the legal failure to understand the nature of scientific reasoning has led to many judicial errors in the use of scientific evidence (section 2, above). This section attempts to clarify to lawyers how scientists make inferences. This process is not altogether dissimilar to the methods used by lawyers. Nonetheless, there are also many ways in which the methods of law and science differ significantly, and this author views this as a positive thing: it means that scientific evidence, used appropriately, has something useful to add to the legal inquiry.

Scientific reasoning is not a mystical process. There is no mode of inference, no “scientific method” exclusive to the sciences and guaranteed to produce true or more empirically adequate results.<sup>326</sup> As the Nobel laureate physicist Percy Bridgman put it, “the scientific method, as far as it is a method, is nothing more than doing one’s damndest with one’s mind, no holds barred.”<sup>327</sup> Haack notes that ‘just like anyone seriously trying to figure something out, scientists make informed guesses at the answers, work out the consequences of these informed guesses, seek out evidence to check how well these hold up, and use their judgment about how to proceed from there.’<sup>328</sup> The point at which evidence becomes strong enough for an inference to be accepted, or weak enough to be rejected, is not dictated by hard-and-fast rules or bright-line delineations. It was this very anxiety that rigid rules would become a substitute for judgment based on the totality of the evidence that led the epidemiologist Sir Austin Bradford Hill to strongly emphasise that the Bradford Hill

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<sup>325</sup> A Einstein, *Physics and Reality* (1936). Reprinted in: A Einstein, *Out of My Later Years* (Philosophical Library, 1950)

<sup>326</sup> Haack *Defending Science* (n 65) 24

<sup>327</sup> Percy Bridgman, cited from S Haack (n 65) 93

<sup>328</sup> Haack, *Evidence Matters* (n 13) 85

factors for causation were meant as considerations only, not as criteria (see Chapter 1 earlier, section 1.1). Haack proposes the term Critical Common-Sensism as a more flexible, less formal, but more realistic view of the standards for solid evidence and rigorous inquiry.<sup>329</sup> Scientific inquiry, she asserts, is not categorically different from any other kind of enquiry, only more so:<sup>330</sup>

Scientific inquiry is continuous with the most ordinary of everyday empirical inquiry. There is no... 'scientific method' exclusive to the sciences... it is what the rest of us do when we really want to find something out. Make an informed conjecture...check how it stands to the best evidence we can get, and then use our judgment whether to accept it.<sup>331</sup>

However, while the scientific enquiry is a common-sense enquiry, it is also something more: it is 'common sense writ large', as Popper puts it.<sup>332</sup> Science is always based on evidence that has been subjected to scrutiny. Scientists reason through problems by constantly testing and questioning their findings, so that less reliable inferences can be sifted out. The conclusions are therefore capable of clear justification, and are significantly more likely to be accurate than reasoning that relies on intuitions, hunches, opinion and speculation. It is this quality of science that can make it invaluable to the assessment of factual aspects of the legal inquiry, for science has refined its methods for the empirical scrutiny of facts with more rigour than any other discipline: this is, indeed, the business of science. 'The principal advantage of scientific methods is not that they eliminate researchers' biases, only that they help to control and reveal the biases that do exist.'<sup>333</sup> While biases can, and do, infect the explorations of scientists, scientific methods are designed and employed to limit that

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<sup>329</sup> Haack, *Defending Science* (n 65) 23

<sup>330</sup> Haack (n 65) 24

<sup>331</sup> *ibid*

<sup>332</sup> Popper (n 4) xxvi

<sup>333</sup> Faigman, *Constitutional Fictions* (n 24) 28

bias as much as possible.<sup>334</sup> Scientific methods permit the development of a body of knowledge about the world that does not depend on the cultural background or values of its progenitors.<sup>335</sup>

Justice Robert Jay's recent extrajudicial lecture suggests that some members of the judiciary may be starting to now recognise some sources of the law-science divide, as well as the value of this uncertain science. Proof in science, he points out, does not mean certainty:

...proof (to the extent that this term has any validity in science) means something along the lines of a consensus having emerged in the scientific community that a particular hypothesis or proposition may now be regarded as solidly grounded. That proposition does not have to be true; it is simply the best available explanation, the best that science can do for the time being. Because there is sufficient common ground, the hypothesis becomes transmuted into something more robust: call it a statement of principle, or a theory of general application that has sufficient evidential ballast. Thirdly, science speaks in terms of degrees of confidence, not proof. Put another way, an expression of scientific opinion should be couched in terms of the amount of evidential support for it, rather than—as the law prefers—in any binary fashion.<sup>336</sup>

Scientific inferences evolve through a complex and integrative reasoning process that is best defined as 'inference to the best explanation'<sup>337</sup> Cranor<sup>338</sup> refers to this reasoning as 'the foundation of virtually all scientific inferences', noting that it is widely used by scientific bodies such as the WHO, in healthcare and clinical decision-making, and by methodologists. This requires scientists to carry out a complicated

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<sup>334</sup> *ibid* 25

<sup>335</sup> *ibid* 24

<sup>336</sup> Jay (n 16) 2

<sup>337</sup> Cranor (n 113) 128-136

<sup>338</sup> *ibid* 125-136

and exhaustive inquiry in order to verify their theory and rule out other possible inferences or explanations. Before arriving at an inference, about causation or anything else, scientists must consider a list of plausible explanations for observed events, then rank rival explanations according to their plausibility, discern what other evidence might be available, consider all relevant information bearing on possible explanations, and only then arrive at their own inference which must be justified.<sup>339</sup> Thus, “atomistic”<sup>340</sup> approaches to evidence (referred to in section 2.4), are erroneous. Since scientific consensus is achieved from the totality of the evidence after many repetitions of the same result, even pieces of evidence that are insufficient in themselves can add to or support the totality of the evidence. The law must cease to examine pieces of evidence in isolation from each other. To do so may cause valuable and relevant evidence to be discarded, or the opposite danger that overarching inferences may be drawn from one eminent study that could turn out to be wrong. This is what led to the much-publicised 1998 scandal surrounding the links between the MMR vaccine and autism. Much media publicity was generated by this theory, based on a study by Dr. Andrew Wakefield and colleagues that was published in the prestigious medical journal, the *Lancet*. The study failed to be replicated by later research, and subsequent investigations showed that the research was fraudulent, leading to a retraction of the study.<sup>341</sup> This shows the scientific fallacy of being overly persuaded by very few sources of information. Decisions must be made on the totality of the evidence: factors such as further replication, and a body of supporting evidence are crucial for drawing sounder conclusions. Epidemiologists, as we shall see in Chapter 5 (section 1), also use similar complex processes to make inferences about

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<sup>339</sup> *ibid* 129-132

<sup>340</sup> Haack, *Evidence Matters* (n 13) 235-237

<sup>341</sup> see F Godlee, ‘Wakefield’s Article Linking MMR Vaccine and Autism Was Fraudulent’ (2011) 342 *British Medical Journal* 22

causation: such inferences are not lightly made from a few simple observed ‘correlations’, as some judicial analyses of epidemiology seem to suggest.

#### **Section 4: Science and evidence in the analysis of disease disputes**

Buchanan et al<sup>342</sup> point out that as science, in the 20<sup>th</sup> century, was disabused both practically and theoretically of its purely deterministic notions of the laws of nature (discussed above, section 1.3), traditional concepts of causation have also dissolved in many ways during the last century. In Rosenberg’s view, so fundamental is the indeterminism indicated by recent discoveries in quantum physics, that ‘at the fundamental level, the principle of same cause, same effect, is invariably violated.’<sup>343</sup> Linear causal models relying on notions of single, necessary causes are not substantiated by current evidence. If the principle of ‘same cause, same effect’ is now no longer even believed to be true in physics, then the expectation for this principle to operate in biological realms, with their incredible ‘complexity, diversity and adaptation’<sup>344</sup> is a doomed quest.

The law, however, remains attached to deterministic approaches towards causation, even though these are repeatedly proving inadequate in disease litigation, as we shall

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<sup>342</sup> AV Buchanan, KM Weiss and SM Fullerton, ‘Dissecting Complex Disease: The Quest for the Philosopher’s Stone?’ 35 *International Journal of Epidemiology* 562, 567

<sup>343</sup> Rosenberg (n 46) 8-9

<sup>344</sup> *ibid*

see in Chapters 3 and 4. As the next chapter will illustrate, the majority of diseases do not stem from a single, necessary cause but rather from multiple causes- there is, usually, a constellation of causes that is responsible for a disease.<sup>345</sup> Sufficiency and even necessity in terms of disease causation is generally only partial; and biomedical causation is more commonly probabilistic.<sup>346</sup> These issues, and the lack of fit between concepts of causation in the law and medicine will be discussed at length in Chapter 3. Broadly speaking, scientific models of causality can be classified into deterministic models, probabilistic models and quasi-deterministic models.<sup>347</sup>

#### ***4.1 Deterministic and Probabilistic Models of Causation in Science:***

Deterministic models of causation hold that events have specific causes, and if these causes are present at certain points or time periods then the events will follow.<sup>348</sup> By contrast, the probabilistic model of causation<sup>349</sup> defines a cause as an event A such that it makes the occurrence of another event (B) *more likely* than if A had not occurred.<sup>350</sup> In other words, the event A may be said to be a probabilistic cause of event B, if, given the occurrence of A, the probability of the occurrence of B is higher than the probability of B if A had not occurred.<sup>351</sup> The central idea behind probabilistic theories of causation thus is that causes *change the probability* of their effects; an effect may still occur in the absence of a cause or fail to occur in its

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<sup>345</sup> J A Markum, *An Introductory Philosophy of Medicine: Humanising Modern Medicine* (Springer 2008) 36

<sup>346</sup> *ibid*

<sup>347</sup> SS Coughlin, *Causal Inference and Scientific Paradigms in Epidemiology* (Bentham 2010) 8-10

<sup>348</sup> *ibid* 9

<sup>349</sup> For more on probabilistic causation generally, and how these probabilities are computed/expressed see e.g. WC Salmon, 'Probabilistic Causality' (1980) 61 *Pacific Philosophical Quarterly* 50, 50-74; also S Sloman, *Causal Models: How People Think About the World and its Alternatives* (Oxford University Press 2005) 36-46

<sup>350</sup> M Hulswit, *From Cause to Causation* (Kluwer 2002) 57-59

<sup>351</sup> *ibid* 57-59

presence. Thus smoking is a cause of lung cancer, not because all smokers develop lung cancer, but because smokers are *more likely* to develop lung cancer than non-smokers.

A probabilistic model of causation allows uncertainty or ignorance about when an event will occur. It allows us to reason about events when we are unsure about what has happened, what will or what would happen, and even about how events lead to one another. As Sloman puts it, all we have to know is how likely events are and how likely they are to be caused by another: causes don't always have to produce their effects, they only have to produce them sometimes.<sup>352</sup>

Cartwright proposes that both these models represent only partial reality, contending that reality may be more accurately represented in the model of 'causal pluralism.'<sup>353</sup> This is based on the view that there is no single account of what a cause means, but instead multiple concepts of cause. We are used to thinking of causation as one thing, Cartwright explains, but maybe causation works differently in different systems: "what causes are...can vary from one kind of system of causal relationships to another."<sup>354</sup> Thus, causal pluralism rejects any attempt to define cause in terms of 'necessary and 'sufficient' conditions.<sup>355</sup> It is thus probable, as Coughlin concludes, that both probabilistic and deterministic models of causation have something to offer for thinking about the nature of causality.<sup>356</sup> If we accept that there are different kinds of causation with different features (e.g. probabilistic and deterministic causation),

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<sup>352</sup> Sloman (n 205) 36-37

<sup>353</sup> N Cartwright, *Hunting Causes and Using Them* (Cambridge University Press 2007) 11-22

<sup>354</sup> *ibid*

<sup>355</sup> J Reiss, 'Third Time's a Charm: Causation, Science and Wittgensteinian Pluralism' in P Illari, F Russo, J Williamson (eds.) *Causality in the Sciences* (Oxford University Press 2011) 907-920

<sup>356</sup> Coughlin (n 203) 8-10



then we must consider which methods are appropriate for which kind of causation.<sup>357</sup> Chapter 3 will explore the approach to causation in tort law, and will contrast this with models of causation in medicine. It will cite current evidence about disease to argue that the legal approach to causation seems inappropriate for assessing complex disease claims, at least.

Scientific evidence indicates that it is pointless to look for certainty about disease causation: it does not exist. The only pragmatic, common-sense question to ask of inferences about causation is: is it a reasonable conclusion, based on the best evidence we have? Epidemiology, with its fundamentally probabilistic philosophy and methods, may be best placed to answer probabilistic questions of disease causation (although, as with anything in science, to go to the other extreme and impose a rigid requirement for epidemiological evidence would also be fallacious. Epidemiological evidence can be very valuable and helpful, but is not *necessary* in all disease scenarios.

#### ***4.2: The value of an uncertain science: the rationale for scientific evidence in legal decision-making***

*'...the idea is that we interpret the input from our senses in terms of a model we make of the world. One can not ask whether the model represents reality, only whether it works.'*

-Professor Stephen Hawking<sup>358</sup>

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<sup>357</sup> Cartwright (n 209)

<sup>358</sup> S Hawking, 'The Origin of the Universe' (Hawking, 2005) < <http://www.hawking.org.uk/the-origin-of-the-universe.html> > accessed 15 March 2016

The description of science in this chapter, as a discipline characterised by varying degrees of uncertainty, may sound disappointing to some lawyers who like answers in clear, black-and-white terms. As we saw through the preceding discussion in this chapter, probabilistic reasoning, including the use of general evidence to answer specific questions, is integral to all scientific methodology, despite the fact that this process is neither fail-safe nor foolproof, and does not deliver cast-iron guarantees. No matter which branch of science, and no matter how well-designed and rigorous a scientific research design, conclusions in an empirical study will always have some limitations, flaws, and potential for error. Scientists accept this, and constantly attempt to find best ways of reasoning in the face of uncertainty. Science does not satisfy the innate human desire for the security that certainty can provide. We do not fully yet understand how to fully epistemically and philosophically justify many of the methods used in empirical science. Thus it is important to address the question of why, then, should the law turn to this uncertain science for clarity?

Scientific inferences, as section 3.4 above discussed, are not infallible. Yet they are empirically tested, scrutinised, actively criticised and capable of being falsified. They are therefore capable of clear justification, and are significantly more likely to be accurate than reasoning that relies on intuitions, hunches, opinion and speculation. It is this quality of science that can make it invaluable to the assessment of factual aspects of the legal inquiry, for science has refined its methods for the empirical scrutiny of facts with more rigour than any other discipline.

The epistemic validity of the scientific reasoning can best be proven by applying Professor Hawking's pragmatic yardstick:<sup>359</sup> with all of its limitations and uncertainty, it is impossible to deny this one fact about science: it works. As Einstein once stated: 'If you wish to learn from the theoretical physicist anything about the methods which he uses, I would give you the following piece of advice: Don't listen to his words, examine his achievements.'<sup>360</sup>

Although tentative, uncertain conclusions are the best science can offer, these have transformed our world and day-to-day lives in ways that are impossible to enumerate or quantify. Most medical procedures and treatments that routinely save lives, and most of the technological advances that we rely on several times an hour to carry out our day-to-day routines, begin from the use of probabilistic research methodologies that constantly evolving and being further refined. As Professor Dawid, a statistician at the University of Cambridge, puts it: "Some may think of the clinical trial as a very poor example of 'science'. It appears to be a very blunt instrument, making no enquiry into the pharmacological or psychological processes whereby aspirin might influence the sensation of a headache. It is, effectively, a black-box approach, perhaps more akin to engineering than science. Well, let us not fight over nomenclature. The important thing is that this empirical black-box approach does deliver the desired results, allowing us to predict what might happen in future administrations of aspirin.

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<sup>359</sup> See above, text to n 214

<sup>360</sup> A Einstein 'The Herbert Spencer Memorial Lecture: On the Method of Theoretical Physics' (Clarendon Press, 10 June 1933)

<sup>361</sup> A P Dawid, 'The role of Scientific and Statistical Evidence in Assessing Causality', in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 136-137.

The term “scientific” has become an all-purpose term of epistemic praise, notes Haack, and has come to mean data that is strong, reliable and “good.”<sup>362</sup> Science is not, however, sacred: like all human enterprises, it is fallible, imperfect, uneven in its achievements, often fumbling, sometimes corrupt, and of course incomplete.<sup>363</sup> Respect for the scientific method should not be based on illusory certainty, but on what it has done for us: science is epistemologically distinguished, Haack reiterates, by its achievements.<sup>364</sup> Despite the fact that science is not conclusive, over centuries scientists have developed a vast and powerful array of tools and techniques: including ever more cunning research designs, and ever more sophisticated mathematical and statistical techniques: all of which, despite ‘untidiness’ have made it possible to get better evidence, assess where the evidence leads; and overall to extend and amplify unaided cognitive processes.<sup>365</sup>

The law-science debate has, Professor Haack feels, always tended to prompt complaints both about the ‘venality and dishonesty’ of scientific witnesses, as well as of the scientific ignorance and credulity of lawyers and judges.<sup>366</sup> This author submits that the differences may actually be a lot less marked than evident on the surface, and that much of the mutual mistrust may be attributable to lack of knowledge and understanding, and thus resolvable by appropriate clarification. While there are indeed many differences, there are also fundamental similarities in how the two disciplines look at evidence and reason through data: commonalities that may remain hidden unless one looks under the surface. Despite the seemingly large number of divergences, the law is fundamentally no stranger to scientific inquiry. Science, like

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<sup>362</sup> Haack *Defending Science* (n 65) 18

<sup>363</sup> *ibid* 19

<sup>364</sup> *ibid* 23

<sup>365</sup> Haack, *Evidence Matters* (n 13) 86

<sup>366</sup> *ibid* 79

the law, has its own philosophy, and lawyers also use scientific notions and theories in everyday legal practice.<sup>367</sup> Wilson notes that the processes a criminal investigator uses is not very different to the process of scientific investigation: investigators form a subjective hypothesis determining how to progress a crime scene, and may not know how partial their evidence is, or what evidence is missing.<sup>368</sup> Blackstone's exhortation to the prospective lawyer "to lay the foundations of his future labours in a solid scientific method" illustrates that rationality and empiricism are also prized in the law.<sup>369</sup>

Lawyers need to grasp the practical limitations of scientific discovery, and develop greater ease with probabilistic reasoning from the best available evidence. Correct use of scientific evidence requires lawyers to recognise that scientific evidence is, as Faigman et al put it, a brick in the wall of proof, but it is not the wall: in other words, 'it need only be probative, it need not prove the case.'<sup>370</sup> Expert testimony must be considered in a more nuanced and contextualized way, within the totality of all the available evidence, to decide overall liability. The role for evidence in the law, as statisticians Taroni and Aitken put it, is not to verify which assertion is the ultimate truth, but only to decide whether the evidence points to the claimant's, or the defendant's assertion as being the more probable.<sup>371</sup> It is all too easy to confuse the epistemologically ideal with the best that is practically feasible, but it is necessary to work within the confines of practical constraints that cannot be overcome.<sup>372</sup> The

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<sup>367</sup> Soar (n 19) 132

<sup>368</sup> Wilson (n 14) 503

<sup>369</sup> Soar (n 19) 132

<sup>370</sup> Faigman et al 'Fitting Science...' (n 6) 383

<sup>371</sup> F Taroni and C Aitken, 'Probabilistic Reasoning in the Law. Part 2: Assessment of Probabilities and Explanation of the Value of Trace Evidence other than DNA' (1998) 38(3) Science and Justice 179, 180

<sup>372</sup> Haack (n 13) 10

choice to use the best available scientific evidence in assessing a question is, it is submitted, not really a choice. It is not perfect evidence, but the law can have no justification for trivialising, or altogether discounting, the best available evidence for factual questions about disease causation.

**CHAPTER 3**  
**DISEASE CAUSATION:**  
**CAUSATION IN LAW, CAUSATION IN MEDICINE, AND A CALL FOR**  
**CONGRUENCE**

In 1950, William Prosser described causation as a ‘tangle and a jungle, a palace of mirrors and a maze.’<sup>373</sup> That it remains in the same state today is not to the law’s credit. Causation is a notoriously complicated stage in the analysis of negligence. Along with damage, duty, breach and remoteness, it must also be shown that the conduct in question caused the damage complained of. In recent years the most difficult and controversial cases in personal injury law and in tort law generally, have concerned causation.<sup>374</sup> This thesis will try to demonstrate that a lack of understanding about how to deal with probabilistic evidence has led the law to overcomplicate some causal issues and to create controversial exceptional tests,

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<sup>373</sup> William L Prosser, ‘Proximate Cause in California’ (1950) 38 California Law Review 369, 369

<sup>374</sup> A Burrows, ‘Uncertainty about Uncertainty: Damages for Loss of a Chance’ (2008) 1 Journal of Personal Injury Law 31, 31

that are, it is argued, ultimately unnecessary. Causation analysis has frequently, and justifiably, been accused of being incoherent and confused.<sup>375</sup>

Complex disease litigation is especially prone to bewildering causal dilemmas. Most legal problems touching on the world of science, as Justice Robert Jay observes, tend to arise in connection with medical and related issues.<sup>376</sup> Giesen observes that establishing the 'causal connection between medical negligence and the injury complained of is probably the most difficult task in medical malpractice litigation (as indeed in many negligence actions).<sup>377</sup> This chapter aims to explore those aspects of the law of causation that hinder the effective use of scientific and epidemiological evidence in toxic tort litigation, rather than to provide a complete doctrinal account of the philosophical, metaphysical and legal dilemmas of causation. It explores the contrast between legal and medical views about causation, and focuses on the more specific impact of the law-science divide (explored earlier in Chapter 2) on toxic tort litigation.

The chapter will highlight two main issues related to factual causation that, in the view of this author, prevent appropriate application of scientific evidence to the causation enquiry. The first issue is the significant infusion of normative considerations into the causation analysis, which prevents an objective judicial assessment of factual causation. The second issue (which will be introduced here, and then explored in greater length in Chapter 4) pertains to the rigid

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<sup>375</sup> J Stapleton, 'Cause in Fact and Scope of Liability for Consequences' (2003) 119 LQR 388

<sup>376</sup> Sir Robert Jay, 'The Richard Davies QC Memorial Lecture 2015: "Standards of Proof in Law and Science: Distinctions Without a Difference?"' (2016) 1 Journal of Personal Injury Law 1, 2-3

<sup>377</sup> D Giesen, *International Medical Malpractice Law: A Comparative Study of Civil Liability Arising from Medical Care* (Martinus Nijhoff, 1988) 163

current legal test for causation, which does not fit with the increasingly probabilistic nature of scientific evidence about causation, as explored earlier in Chapter 2. The law tends to take a deterministic view of the world,<sup>378</sup> and of causation, with its emphasis on but-for or necessary/sufficient causes. However, current medical evidence (explored in section 2 of this chapter) indicates that disease causation is multifactorial, complex, and frequently stochastic. This chapter argues that the misalignment between law and science (particularly their deterministic and probabilistic views, respectively, of causation) is magnified in disease litigation, and causes a great deal of incoherence in the law of causation. Most inconsistencies in causation principles have arisen from courts' attempts to deal with situations of evidentiary uncertainty, which occur when traditional legal causal questions cannot be answered by the available evidence. (The word 'normative', it must be clarified, is used here to refer to the recourse to intuitive notions of justice and fairness in the assessment of factual causation. This is similar to Fumerton and Kress'<sup>379</sup> use of the term, where they define 'normative considerations' as the consideration of factors such as good/bad, should/should not, right/wrong, or desirable/undesirable in assessing factual causation. Thus this term as it is used in this thesis is narrower than the term 'policy' reasoning, which is a broader concept that can include considerations such as distributive justice etc.).

Unfortunately, rather than reflecting on why medical science is repeatedly unable to answer *legal* questions about disease (while it satisfactorily uses the

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<sup>378</sup> Jay (n 4) 3

<sup>379</sup> R Fumerton and K Kress, 'Causation and the Law: Preemption, Lawful Sufficiency, and Causal Sufficiency' (2001) 64 *Law and Contemporary Problems* 83, 85



same evidence to achieve great progress in diagnosis and treatment in the real world), or taking the opportunity to examine the ‘fitness’ of archaic legal tests for causation, courts have dealt with the difficulties by formulating haphazard ‘exceptional’ causation principles on a case-by-case basis. The boundaries, justification, and applications of these exceptional tests are often unclear. This creates further confusion in causation principles. Courts appear to have sometimes responded to the dilemmas by throwing their hands up in despair at the very concept of causation, and seem content to apply the rather inverted logic that if the imposition of liability seems fair, then causation is, somehow, thereby established. This thesis submits that to so detach causation from factual objectivity, and to turn it into such a normative and subjective inquiry, damages the law. Such an approach defeats the fundamental corrective justice principles of tort law, and renders the causation analysis a meaningless, opaque exercise.

It is now imperative, it is submitted, for the law to grasp that many of the challenges in establishing factual causation in claims for diseases resulting from toxic exposures do *not* stem from failures of science that will soon be rectified by more evidence. Rather, most difficulties stem from misalignment of deterministic legal models of causation with increasingly probabilistic scientific causal models of disease. The problems are further exacerbated by the fact that the law does not fully recognise the extent of its misconceptions about science. Lord Phillips’ speech in *Sienkiewicz v Greif*<sup>380</sup> illustrates the extent to which the myth of scientific certainty is prevalent within the judiciary:

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<sup>380</sup> [2011] UKSC 10

*When a scientific expert gives an opinion on causation, he is likely to do so in terms of certainty or uncertainty, rather than probability. Either medical science will enable him to postulate with confidence the chain of events that occurred, i.e. the biological cause, or it will not. In the latter case he is unlikely to be of much assistance to the judge who seeks to ascertain what occurred on a balance of probability.*<sup>381</sup>

The reality of how a scientific expert actually thinks about causation, is, of course, very different, as we saw earlier in Chapter 2. However, courts continue to use rigid legal models of causation, seemingly in the belief that the ideal scientific evidence should, and very soon will, provide certainty. This leads to a judicial tendency, particularly within UK tort law, to devalue probabilistic scientific evidence about disease (as seen in sceptical UK judicial approaches towards epidemiological evidence). Unfortunately, the best that science may ever be able to offer, in the absence of miraculous eyewitness testimony about chemical and molecular processes going on inside human organs and cells, is probabilistic evidence, which even if not certain, has empirically proved its value by its achievements. It is difficult to justify the legal disregard for the best *available* empirical evidence in assessing factual causation.

Section 1 outlines the problems in the current approach to causation in tort law. It focuses on two particular aspects that, in the view of this author, hinder the effective legal use of scientific evidence in this area. The first, explored in section 1.1, is the judicial tendency to view causation as a normative issue, and to arrive at causal conclusions depending on where courts feel overall liability should fall.

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<sup>381</sup> *ibid* [9] (Lord Phillips)

This normative, backwards analysis of factual causation stems from two further areas of confusion. Section 1.1(a) discusses the first: the judicial tendency to conflate the two-step analysis of causation ('factual causation' and so-called 'legal causation') into one single, often subjective, analysis. Section 1.1 (b), highlights the second issue: lack of clarity about the role of the causation enquiry. Causation is often seen as synonymous with overall liability. This in turn leads to a judicial reluctance to assess factual causation objectively, as courts appear reluctant to give up their discretion in regard to causal conclusions. To deny due importance to robust assessment of factual connections is to detach tort law from its primary goal of corrective justice, this thesis contends. Section 1.2 then outlines the second problem in the current law of causation: deterministic legal tests for causation that do not fit with probabilistic scientific evidence. The traditional but-for test, it is submitted, is often too rigid and simplistic to assess causation in all but the most straightforward disease claims, due to the multifactorial and variable etiology of many diseases. (This issue will be explored at greater length in Chapter 4). Medicine and the law view causation from different perspectives, which is why medical and epidemiological evidence often do not directly map on to legal questions. Jost notes that medicine is primarily interested in causation in a practical, scientific, sense.<sup>382</sup> Medicine seeks to understand the "cause" of a disease in order to facilitate treatment (or to improve public health and prevent disease). However, because tort and criminal law are concerned with attributing responsibility, they must focus on the particular conduct of particular persons, and judgments of attribution are driven

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<sup>382</sup> T Jost, 'Book Review: *Causation in Law and Medicine* by D Mendelson and I Freckleton (eds)' (2003) 24 *Journal of Legal Medicine* 127, 128

at least as much by policy considerations as by scientific investigation.<sup>383</sup>

However, this thesis argues that the infusion of these issues into the assessment of *causation*, which is a scientifically complex matter already, causes untold confusion in the law of causation. Vital as matters such as responsibility, morality and personal conduct are to the law, these must be separated out from the assessment of factual causation (which requires objective and fact-based analysis), and analysed separately.

Section 2 examines, by way of contrast with the legal approach to disease claims, disease causation from the medical perspective. It aims to illustrate the nature of disease in order to later develop the argument that deterministic legal approaches are a poor fit for assessing causation (in this area of the law, at least). It examines current medical literature in order to demonstrate some fundamental issues about disease that lawyers need to grasp. Through this, it aims to make the argument (which will be more fully developed in chapter 4), that it is vital for the law to take a more informed approach towards the probabilistic scientific evidence in this area. The majority of major diseases, evidence suggests, arise from complex, multifactorial causal events rather than the simplistic single-cause model that current legal principles might have more easily dealt with. Mesothelioma-type diseases (which the law tends to view as exceptional in terms of evidentiary uncertainty about etiologic mechanisms) are not so rare or unusual at all. Many complex diseases are, in fact, even more causally uncertain than mesothelioma, because mesothelioma at least has one clearly identified single, necessary, but-for causal factor (i.e. asbestos exposure).

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<sup>383</sup> *ibid*

This is not the case with many other major complex diseases, which can arise from many different combinations of a set of factors. Thus this thesis thus further contends that the deterministic, necessity-based, 'but for' test repeatedly causes problems in disease litigation because it is fundamentally unsuited to the assessment of disease causation.

Section 3 argues that better integration of legal and scientific approaches in this area is now imperative, and examines the futility of the legal idea that better scientific evidence will very soon often solve all problems by offering conclusive evidence about disease causation. A number of commentators have eagerly anticipated the day when science exposes the presumed deterministic mechanism of toxic causation for all to see.<sup>384</sup> However, this may be misplaced optimism. For example, Professor Steve Gold uses sophisticated genomic and molecular research about disease causation to argue that actually the opposite is true: at the highest magnification, "certainty dissolves into probability."<sup>385</sup> (This could be likened to the epistemic trajectory of physics, where the same surprising lesson seems to be emerging from modern quantum discoveries. It appears that more scientists discover, the less certain they are of their laws, or of the predictability of the universe: as explored earlier in Chapter 2<sup>386</sup>). *The very deterministic questions the law asks regarding disease causation, however, suggests that the current legal approach is simply not flexible enough to cope with these different possibilities suggested by current medical knowledge.*<sup>387</sup> As the

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<sup>384</sup> SC Gold, 'When Certainty Dissolves Into Probability' (2013) 70 Washington and Lee Law Review 237, 265

<sup>385</sup> *ibid* 265-266

<sup>386</sup> Chapter 2 section 1.3

<sup>387</sup> CF Cranor, *Toxic Torts: Science, Law and the Possibility of Justice* (New York, Cambridge 2007)

potential unfairness of the but-for test becomes increasingly apparent in the context of a range of serious and debilitating diseases, it will become harder for courts to justify restricting the exceptional approach to a few selected diseases. It is time for the law to re-evaluate its simplistic 'but for' test for causation, at least so far as disease litigation is concerned. Chapter 4 of this thesis will discuss in detail the problems with the but-for test in this area of the law, and will suggest a more principled approach to causation that better aligns with scientific evidence.

## **SECTION 1: Causation in tort law: obstacles to the proper application of science**

### ***1.1: Normative assessment of factual causation***

It is axiomatic that there must be a causal link between the negligence and the claimant's injury before liability can attach to a defendant.<sup>388</sup> A plethora of legal theories about causation abound in the literature.<sup>389</sup> These range from broad-based views about causation encompassing cause-in-fact as well as proximate-cause issues, which equate causation with legal liability,<sup>390</sup> to 'legal realism'<sup>391</sup>

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129-135

<sup>388</sup> M Lunney and K Oliphant, *Tort Law* (5<sup>th</sup> edn, OUP 2013) 205

<sup>389</sup> See Richard Wright, 'Causation in Tort Law' (1985) 73 *California Law Review* 1735, 1737-1774 for a summary

<sup>390</sup> HLA Hart and A Honoré, *Causation in the Law* (2<sup>nd</sup> edn, OUP 2002) 65-66

(causal minimalist) theories which insist that the only causal issue is the simple factual issue of whether the defendant's conduct actually contributed to the plaintiff's injury, to efficiency theories which view causation requirements as subordinate to the simple efficiency purpose of the law.<sup>392</sup>

Despite its overarching centrality to the doctrine of negligence, the word 'causation' often appears to mean very different things to different legal minds. As Wright puts it: in all of tort law, there is no concept that is as pervasive and yet elusive as the causation requirement, which relieves a defendant of liability if his tortious conduct was not a cause of the plaintiff's injury.<sup>393</sup> Much of the current incoherence in causation arises because there is little clarity or consensus about what the causation analysis should focus on, and the role it should play in the overall negligence enquiry. Courts often tend to equate causation with overall liability, and this has infused an unacceptable degree of normativity into the causation analysis. Partly this is because the principles of causation are now so out of step with causal models of disease, that courts are compelled to twist causation principles in order to arrive at the desired outcome. Causation analysis is often equated with the question of whether the defendant should be fixed with legal liability at all, with courts often basing this analysis on such extensive issues as blame-worthiness, economics, morality and the various undefined issues collectively termed as "policy" (as discussed earlier in the context of Fumerton and Kress' definition of a normative approach to

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<sup>391</sup> William L Prosser, *Handbook of the Law of Torts* (4<sup>th</sup> edn, West Publishing Co. 1971) 236-238

<sup>392</sup> See, e.g., P Burrows, 'A Deferential Role for Efficiency Theory in Analysing Causation-based Tort Law' (1999) 8 *European Journal of Law and Economics* 29

<sup>393</sup> Wright (n 17) 1737

causation,<sup>394</sup> policy is a partly overlapping, but broader concept than the term 'normative' as used in this thesis). Beever explains the distinction between policy and principle:

Principle' refers to the rules and doctrines of the law itself... 'Policy', on the other hand, can be defined only negatively. 'Policy' is everything apart from principle. For example, policy has been held to include issues of distributive justice, social morality, economic efficiency, public opinion and so on. But it is impossible to define the content of these terms exactly, because people disagree on what constitute the rules and doctrines of the law.<sup>395</sup>

Beever strongly proposes that 'policy' reasoning has no role to play in the negligence analysis at all. This thesis, however, does not take a position in this debate about the general role of policy in legal decision-making. It simply contends that these are not matters of factual causation, or indeed, of causation at all. Causation analysis, which can only have any useful purpose as a tool to *guide* the court in arriving at decisions, turns (under normative approaches) into an enquiry where the answers are formulated *depending* on what decisions courts have already arrived at. This can turn causation into a redundant and meaningless element of the negligence enquiry. Courts appear to be resorting to a retrospective analysis of causation, and incoherent principles are an inevitable result, as exemplified in *Fairchild v Glenhaven Funeral Services Ltd*,<sup>396</sup> discussed in more detail in Chapter 4.

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<sup>394</sup> See above, text to n 7

<sup>395</sup> A Beever, *Rediscovering the Law of Negligence* (Hart Publishing 2009) 3

<sup>396</sup> [2002] UKHL 22



This thesis does not assert that normativity has no role in the administration of justice, only that these are not matters of causation. As Beever<sup>397</sup> points out, the causation requirement in negligence has a unique purpose to fulfill: while other negligence matters such as standard of care, duty, remoteness and defences fit together in a unified normative enquiry, causation is different. Causation is not an artificially created or legal concept. It is morally arbitrary. To say that causation is in any way “a normative enquiry or a policy matter is to imply that our judgments or preferences for deciding liability determine the fundamental nature of the universe; as if, were human beings not to exist, or were the law to be abolished, the fundamental nature of the universe would change”.<sup>398</sup> As Prosser puts it:

Whether proper construction of a building would have withstood an earthquake, whether reasonable police precautions would have prevented a boy from shooting the plaintiff in the eye with an air gun, whether a broken flange would have made an electric car leave the rails in the absence of excessive speed... cannot be decided as a matter of law.<sup>399</sup>

The insistence on a causal connection has, notes Honoré, a crucial justificatory purpose for tort law, as a causal connection between the conduct and harm ‘ensures that in general we impose liability only on those who, by intervening in the world, have changed the course of events for the worse’.<sup>400</sup> Professor Honoré emphasises repeatedly across a series of distinguished works on causation that it is crucial to distinguish between the concepts of causation, responsibility, and

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<sup>397</sup> Beever (n 23) 413-414

<sup>398</sup> *ibid* 413

<sup>399</sup> Prosser, ‘Proximate Cause in California’ (n 1) 382-383

<sup>400</sup> A Honoré, ‘Necessary and Sufficient Conditions in Tort Law’ in D Owen (ed), *Philosophical Foundations of Tort Law* (Clarendon Press 1995) 385

legal liability. Being held responsible for the consequences of our actions encourages us to act so as to do good and to avoid doing harm, and also allows us to assume a character and identity for which we can, or must, take responsibility.<sup>401</sup> An effort to ensure that people are only penalised for the wrongs they have caused is thus the basic foundation of a just system. Wright similarly argues for a non-normative concept of causation, articulated in famous NESS test for factual causation.<sup>402</sup> This thesis submits that the extensive infusion of normative considerations into the factual causation inquiry is the result of two deeper problems in the law, outlined below.

### **1.1.1 Conflation of factual causation and legal causation**

Traditional academic conceptualisations see causation as being a two-step enquiry. The first step, factual causation, requires the claimant to establish a historical connection between the defendant's act and the ultimate injury. This is normally decided using the but-for or sine qua non test which asks: would the injury have happened but for the defendant's negligence?<sup>403</sup> This deceptively simple question, with its emphasis on necessity, can present insurmountable problems in some situations, where rigid applications of this test can be absurd or patently unfair to the claimant.<sup>404</sup> Problems also occur in evidentiary gap scenarios, and here courts occasionally apply exceptional alternative tests to the but-for test, which will be discussed at greater length in Chapter 4. Thus factual

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<sup>401</sup> A Honoré, 'Principles and Values Underlying the Concept of Causation in Law', in D Mendelson and I Freckleton (eds.), *Causation in Law and Medicine* (Ashgate 2002) 3-4

<sup>402</sup> Wright (n 17) 1735

<sup>403</sup> A principle established in *Barnett v Chelsea and Kensington Hospital Management Committee* [1969] 1 QB 428 (QBD)

<sup>404</sup> As in, for example, *Fitzgerald v Lane*: [1989] 1 AC 328

causation, as Weinrib notes, is a historical inquiry: it is 'a factual inquiry which is resolved by the production of evidence and the drawing of inferences from that evidence.'<sup>405</sup>

Once factual causation has been established, the claimant must then demonstrate 'legal causation': that is, they must establish that the defendant should be legally responsible for the tort. This involves asking various questions such as whether there were any intervening events, or whether the defendant's act was too remote, or not proximate enough, for liability to be imposed.

This, however, is merely the textbook view of causation. In practice, the analysis of causation appears to be a much more haphazard process than the academic view above suggests. Lord Hoffmann, for example, writing extra-judicially, concedes that judges rarely tend to apply the two-stage test of causation: 'Why do they (judges) obstinately refuse to apply the 'two-stage test' and distinguish between their findings of 'cause in fact' and their subsequent decision about 'legal causation'? None of these concepts appears to have attracted any judicial interest.'<sup>406</sup> His Lordship then goes on to state that in his view it is entirely appropriate for judges to avoid too much 'hand-wringing' about factual causation: causal rules are 'creatures of the law and nothing more', Lord Hoffmann emphasises, before concluding that: '(t)he concept of cause in fact seems to me to add nothing of value to the discussion of this question.'<sup>407</sup>

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<sup>405</sup> EJ Weinrib, 'A Step Forward in Factual Causation' (1975) 38 MLR 518, 518

<sup>406</sup> Rt Hon Lord Hoffmann, 'Causation' in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 3

<sup>407</sup> *ibid* 9

‘The failure to distinguish between cause and responsibility is a fundamental one,’ stated Prosser in 1951, ‘and any attempt to state one in terms of the other has led and can only lead to the most hopeless confusion.’<sup>408</sup> Subsequent developments in the law since then bear out the prescience of this statement. Much of the current confusion in the law of causation arises because of this failure to distinguish between factual and legal causation. It is mystifying that the second step (“legal causation”) is called a causation issue at all, because what it is really involves, in practice, is the arrival of the court at a decision about liability, using a variety of different criteria such as remoteness, foreseeability, intervening acts, and the eggshell skull rule. However, these do not assess causation. They examine primarily whether the degree of fault is proportionate to the consequences of legal liability.

The proposition that ‘legal’ or ‘proximate’ causation is not really a causation issue, and that factual causation must be objectively established before considering legal responsibility, has found strong support over the years from a plethora of distinguished academic voices. Professor Glanville Williams asserted (albeit in the context of criminal law) that, ‘when one has settled the question of but-for causation, the further test...in order to qualify it for legal recognition is not a test of causation but a moral reaction.’<sup>409</sup> Further, Professor Williams notes elsewhere, ‘If a fact is not the scientific cause of an event, it cannot be the proximate cause’.<sup>410</sup> Prosser points out that terminology such as “proximate cause” covers a ‘multitude of sins’. It is, he points out, a complex term of highly

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<sup>408</sup> Prosser, ‘Proximate Cause in California’ (n 1) 375

<sup>409</sup> Glanville Williams, *Textbook on Criminal Law* (Sweet and Maxwell 2012) 381, cited from N Padfield, *Criminal Law* (9<sup>th</sup> edn, OUP 2014) 34

<sup>410</sup> Glanville Williams, *Joint Torts and Contributory Negligence* (Stevens & Sons Ltd, 1951), 240

uncertain meaning under which other rules, doctrines and reasons lie buried, and that at least in many cases there is no real question of causation at all.<sup>411</sup> “It (causation) is a question of fact,”<sup>412</sup> Prosser emphasises, going on to point out that establishment of this fact does is not, of itself, sufficient for liability. There is a practical necessity for legal responsibility to be limited to only some factual causes, but that limitation is not ‘in any sense one of causation: it is one of rules and policies which deny liability for what has clearly been caused. Stapleton robustly argues that the term ‘legal causation’ is a misnomer, and *proposes that it should actually be termed ‘scope of liability’ for consequences, as* “a bare reliance on undefined causal terms can lead courts to fail to distinguish the *factual* issue of historical involvement from the *normative* judgment of which consequences of the tort fall within the appropriate scope of liability.”<sup>413</sup>

Lord Hoffmann’s approach to causation appears instead to support a wholly normative analysis, where factual and ‘legal’ causation are combined into a mostly subjective inquiry. With due respect to Lord Hoffmann, while such an approach may make the task of courts easier, it does great disservice to both the interests of justice as well as to the reputation of the law. To advocate that legal analyses of causation can be so detached from factual concerns is also to detach tort law from its basic theoretical foundations of corrective justice, which is the cornerstone of UK tort law. Causation is the central feature of corrective-justice based interpersonal responsibility since it is the causal relationship that connects the claimant and defendant as parties to an interaction, notes

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<sup>411</sup> Prosser, ‘Proximate Cause in California’ (n 1) 374

<sup>412</sup> *ibid* 375

<sup>413</sup> Stapleton (n 3) 392-393

Turton.<sup>414</sup> Stressing that the causal inquiry is of utmost importance in the negligence analysis, Moore further explains that: '[C]orrective-justice...demands a robustly metaphysical interpretation of cause. For legal liability tracks moral responsibility on this view, and moral responsibility is for those harms we cause. 'Cause' has to mean what we mean when we assign moral responsibility for some harm, and what we mean in morality is to name a causal relation that is natural and not of the law's creation.'<sup>415</sup>

The call to clarify legal terminology in this area is not, it is submitted, a quibble about mere semantics. It is an issue that deserves serious reevaluation because of its impact: this misplaced terminology has resulted in blurred boundaries between the analysis of factual causation and legal liability itself. It has damaged the clarity and coherence of the law by turning the causation analysis into a haphazard and occasionally incomprehensible exercise. Courts have gradually come to see the analysis of factual causation as an enquiry whose outcome must be shaped by where they wish liability to fall, rather than an objective evaluation of causation.

### ***1.1.2: Inflated role of causation in the liability analysis***

A further barrier to the willingness of the judiciary to examine factual causation objectively, it is submitted, is that the scope of the causation has now become excessively broad. Causation is seen as synonymous with overall liability, which

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<sup>414</sup> G Turton, *Evidential Uncertainty in Causation in Negligence* (Hart Publishing, 2016) 20

<sup>415</sup> MS Moore, *Causation and Responsibility: An Essay in Law, Morals, and Metaphysics* (Oxford University Press 2009) 83, 95

appears to lead to judicial anxiety to retain discretion over the outcome of the causation analysis. Ironically, the fact that courts give so large a role to causation (in theory) is what appears to make them reluctant (in practice) to conduct a genuine, objective enquiry about whether factual causation has been established on the balance of probabilities. Since causation answers are seen as the defining basis for liability, courts often carry out a retrospective analysis of causation, because they appear to believe that causal conclusions that are incompatible with the overall decision can lead to unfair overall outcomes.

In *Fairchild*, Lord Hoffmann stated:

*Everyone agrees that there is no scientific or philosophical touchstone for determining the relevant causal connection in any particular case. The relevance of a causal connection depends upon the purpose of the inquiry.*<sup>416</sup>

Lord Hoffmann again elaborated on this point in *Kuwait Airways Corporation v Iraqi Airways Co*:<sup>417</sup>

*There is therefore no uniform causal requirement for liability in tort. Instead, there are varying causal requirements, depending on the basis and purpose of liability. One cannot separate questions of liability from questions of causation. They are inextricably connected.*

This statement typifies the inverted logic that now governs the causation analysis. To say that rules about causation can be framed depending upon the

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<sup>416</sup>*Fairchild* (n 24) [48-49]

<sup>417</sup>[2002] UKHL 19 [128] (Lord Hoffmann)

basis and purpose of liability is to render the causation analysis a biased and meaningless exercise. Further, Lord Hoffmann offers no clarity about what, in this view, the purpose of liability is. His approach towards exceptional rules appears to subscribe to the functionalist view of causation: that is, that the main purpose of liability in negligence is to compensate the claimant. However, this vague analysis leaves many important questions hanging.

A more coherent approach to causation requires courts to recognise that causation is but one strand in the negligence enquiry. Giving causation a narrower place in the overall analysis would help courts assess factual issues with much more objectivity and rationality. As Prosser puts it, the answer to the factual question is not determinative: it is, 'at most, a rule of exclusion.'<sup>418</sup> This is a point that Weinrib also emphasises: 'The finding that the defendant did in fact cause the injury does not automatically mean that he is liable since the court still must assess whether the factual causation should have legal consequences. Since cause in fact can determine innocence but not liability, it functions as a test of exclusion, allowing a court to weed out defendants without having to decide whether their conduct was legally culpable.'<sup>419</sup>

This also significantly contributes to UK judicial reluctance to give due weight to epidemiological evidence, because of a misconceived belief that if epidemiological evidence is accepted, it will dictate the outcome of the claim. McIvor presents this belief as a function of the law's approach to statistics in

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<sup>418</sup> Prosser, 'Proximate Cause in California' (n 1) 376-377

<sup>419</sup> Weinrib (n 33) 518-519



general, and posits that it results from courts ‘conflating the substantive and evidential aspects of the test for factual causation and the reducing that conflation into a nonsense numerical test.’<sup>420</sup> This reflects the legal tendency to reduce scientific methodologies into somewhat ‘pat’ and simplistic rules and hierarchies. Chapter 5<sup>421</sup> will later explore these legal hierarchies of scientific evidence further, and will demonstrate why these are flawed.

### ***1.1.3: Reducing the role for normativity in factual causation:***

Courts frequently justify their haphazard approaches to causation on the grounds that causation is a normative enquiry, and that it is less important to accurately assess causation than to achieve fair overall legal outcomes. However, for this thesis to contend that the causation element of the negligence analysis must briefly set aside normative concerns is not to negate the ultimately normative nature of the legal analysis. The proposal that factual causation must be examined objectively, not normatively, only emphasises that different strands of the negligence enquiry may require the exercising of different kinds of reasoning and cognitive processes. Every step involved in carrying out an ultimately normative enquiry does not need to be decided *primarily* by normative considerations, because that makes the individual elements of the negligence enquiry pointless. If I bake a Spiderman cake for a birthday party,

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<sup>420</sup> C McIvor, ‘Debunking Some Judicial Myths About Epidemiology and its Relevance to UK Tort Law’ (2013) 21 Medical Law Review 553, 577 The ‘nonsense’ numerical test refers to the ‘doubling of the risk’ test, that is based on a relative risk value greater than 2, as discussed earlier in Chapter 1 (section 3.3).

<sup>421</sup> Section 2.1

then the stage at which I sculpt Spiderman out of the icing sugar would ostensibly follow none of the processes seen as fundamental to cake-baking: I am not, at this stage, whipping flour and eggs and sugar into a fluffy mass ready for the oven. Nonetheless, I am still involved in the ultimate activity of baking what will hopefully be a very fine cake.

Although legal decision-making may indeed require consideration of many other factors such as values, justice, and 'policy,' these must be separated from the question of fact. Fumerton and Kress address the role for normative considerations in the legal analysis: 'Which of the indefinitely many causally relevant factors constitute proximate causes or the causes with which we are concerned in a legal context may well involve normative elements that cannot be eliminated. Traditionally, it is said that the proximate cause concept selects from among all actual causes the legally responsible causes by invoking policy considerations. But whether something is a causally relevant factor in producing some outcome may be non-normative through and through. One should not succumb to the temptation to suppose that relevant questions of causation are permeated by the normative even if the decision to focus only on certain causal factors is.'<sup>422</sup>

Although causation may sometimes, as Lord Hoffmann notes, need to be differently assessed in different systems,<sup>423</sup> this cannot justify a cavalier approach to facts and proof. A flexible approach to assessing causation is

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<sup>422</sup> Fumerton and Kress (n 7) 88

<sup>423</sup> Lord Hoffmann (n 34) 9

different from an arbitrary and entirely subjective approach. This thesis agrees that courts need to be pragmatic, and cannot get bogged down by philosophical and metaphysical debates about causation in every dilemma they face. However, the law must at least have clarity about the basic purpose of causation in tort law, and strive to formulate principles that have a reasonable degree of clarity and coherence.

This thesis does not advocate that scientific or epidemiological evidence should be decisive in legal decision-making. On the contrary, it argues that inflated perceptions of the certainty and authority of any form of scientific evidence is wrong and will lead to injustice all around (see Chapter 2, section 2).

Nonetheless, scientific evidence, for all its fallibility, is amongst the most valid and well-tested forms of evidence we have. Used judiciously as a tool to help guide the factual causation analysis, rather than as determinative of the outcome, epidemiological evidence can help enhance the clarity of legal decision-making (especially where no other specific evidence is available: as is frequently the case in disease litigation). Questions of causation not only *can*, but also *should*, be separated from questions of ultimate liability.

### ***1.2: The problem with necessity-based tests for disease causation***

On the one hand, courts display a distinct fondness for the notion of a ‘common-sense’ approach to causation, expressing the view that too much ‘hand-wringing’

(as Lord Hoffman puts it)<sup>424</sup> about rigid boundaries is unnecessary. Judicial pronouncements avowing a preference for ‘ordinary, everyday’ concepts of causation abound in case law. Lord Salmon stated in *Alphacell v Woodward* that:

*The nature of causation has been discussed by many eminent philosophers and also by a number of learned judges in the past. I consider, however, that what or who has caused a certain event to occur is essentially a practical question of fact which can best be answered by ordinary common sense rather than abstract metaphysical theory.*<sup>425</sup>

Or, as Lord Reid put it in *McGhee v National Coal Board*,

*It has often been said that the legal concept of causation is not based on logic or philosophy. It is based on the practical way in which the ordinary man's mind works in the everyday affairs of life.*<sup>426</sup>

Hart and Honoré<sup>427</sup> similarly emphasise that “it is the plain man’s notions of causation (and not the philosopher’s or the scientist’s) with which the law is concerned.” However, as Williams points out, Hart and Honoré’s description may be partly true of the legal notion of proximity, but it is not true of the lawyer’s use of the notion of but-for causation.<sup>428</sup> The rigid and deterministic but-for test for factual causation, with all the attendant requirements it imposes on the claimant to prove that the injury was *necessary* for the harm, does not appear to tie with this common-sense approach, certainly not in disease litigation.

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<sup>424</sup> See text to n 35

<sup>425</sup> *Alphacell v Woodward* [1972] UKHL 4; [1972] A.C. 824 [847]

<sup>426</sup> [1973] 1 WLR 1 at [5] (Lord Reid)

<sup>427</sup> Hart and Honoré (n18) 1

<sup>428</sup> Glanville Williams, ‘Causation in the Law’ (1961) 19 Cambridge Law Journal 62, 65

Unfortunately, the most current medical evidence (explored in Section 2 below) suggests that the but-for test is often neither practical, nor fair, nor a common-sense approach, for the purpose of assessing disease causation, at least. Notes Professor Steve Gold, a legal scholar and biology graduate, proof of causation in toxic torts has presented persistent problems for the legal system, because scientific probabilities fit poorly with the demands for particularistic proof imposed by the law's deterministic model of causation.<sup>429</sup> This, Gold points out, is because toxic injuries almost never involve an observable chain of physical events allowing easy inference of a causal relation between a particular defendant's conduct and a particular plaintiff's harm. Courts turn to science to replace causal intuition, but a disjunction remains between the probabilities that science can know and the determined result that the law wants proven; leading to led to numerous calls for doctrinal reforms in recognition of the difficulties that toxic causation presents.<sup>430</sup> The but-for test of factual causation has repeatedly caused problems, particularly when applied to disease disputes.<sup>431</sup> This has resulted in courts fashioning a number of modified tests to help claimants who are unable to establish factual causation due to evidentiary uncertainties. Many complex variations of such situations can arise, and courts have dealt with the dilemmas on a case-by-case basis, formulating a variety of 'exceptional'<sup>432</sup> principles to establish factual causation.

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<sup>429</sup> Gold (n12) 237

<sup>430</sup> *ibid*

<sup>431</sup> Explored later in chapter 4

<sup>432</sup> Discussed further in Chapter 4

Prosser summarises the but-for or *sine qua non* rule as follows: 'The defendant's conduct is not the cause of the event, if the same event would have occurred without it.'<sup>433</sup> The problem with complex disease, as section 2 will demonstrate, is that the same disease can be caused in so many different ways, through so many different combinations of factors, that experts can almost never say with certainty that *this* disease would never have happened but for *that* part of the event. How, then, in such a scenario, is but-for causation to be established? As Justice Jay extrajudicially noted in a recent article:

Practical problems naturally arise in the law where a fact in issue cannot be proved by direct evidence but by inference. **The law may be based on a deterministic view of the natural world.** Causes may in theory be observed as they occur, but they will not have been if the past event has come and gone. The drawing of an inference is the only available pathway to proof.<sup>434</sup>

Chapter 4 will discuss the problems with the but-for test, and will outline the cases where courts have departed from this test. It will also attempt to frame some suggestions for alternatives to the but-for test that fit better with medical evidence about diseases resulting from exposure to harmful substances. First, however, we will explore current medical evidence, in order to better understand why a departure from deterministic causal models is imperative in this area of litigation.

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<sup>433</sup> Prosser, 'Proximate Cause in California' (n 1) 377

<sup>434</sup> Jay (n 4) 3 (emphasis added)

## **SECTION 2: Medical evidence about disease causation: the multifactorial, stochastic nature of disease**

*“Medicine is a science of uncertainty and an art of probability.”*

-Sir William Osler<sup>435</sup>

This section examines the medical issues surrounding disease causation, in order to demonstrate why it is crucial for the law to develop felicity with probabilistic reasoning in the assessment of disease causation. The law’s rather simplistic and deterministic conception of causality has led lawyers to believe that all events have clear, consistent, identifiable causes. The court’s only job, in this view, is to decide in regard of an alleged tort whether such a factor represented a breach of duty, and whether the factor ‘swung the outcome’: in other words, whether the event would not have happened without the tort.

Unfortunately, when applied to claims for negligently caused disease, this approach to causation has repeatedly fallen apart, as shown across a string of toxic tort case law.<sup>436</sup> The legal desire to maintain the traditional deterministic reasoning around causation has necessitated a number of contorted exceptional tests for causation: the scope, justification and boundaries of which become ever more confused.<sup>437</sup>

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<sup>435</sup> Sir William Osler (1849-1919). Cited from P Elliott and W McKenna, ‘The Science of Uncertainty and the Art of Probability: Syncope and its Consequences in Hypertrophic Cardiomyopathy’ (2009) 119 *Circulation* 1697, 1697

<sup>436</sup> As occurred, for example, in cases such as *Fairchild* (n 24)

<sup>437</sup> the inadequacies of the but for test will be discussed in Chapter 4

Courts appear to evading the problem by clinging to the belief that all the problems are due to scientific inadequacies, and that soon better medical evidence will turn up, offering better proof of but for causation. This hope is deeply illusory, at least as far a disease causation is concerned. Increasingly sophisticated research methodologies suggest that most major diseases result from a complex interplay of multiple factors, which have different effects on different people due to biological variations.<sup>438</sup> Multiple studies of toxic susceptibility are unlikely to give identical results because of the influence of other factors and of random chance.<sup>439</sup> 'Variability' stated Sir William Osler, the eminent Canadian physician who is viewed as the founding father of modern medicine,<sup>440</sup> is the law of life, and as no two faces are the same, so no two bodies are alike, and no two individuals react alike and behave alike under the abnormal conditions which we know as disease.'<sup>441</sup> In spite of massive investments in research time and funding, the etiologic mechanisms of many major complex diseases such as cardiovascular disease, cancers, psychiatric illness, asthma, diabetes and Alzheimer's Disease, etc. are still largely unknown or, at best 'vaguely known.'<sup>442</sup>

All of these issues constitute a challenge that the law will undoubtedly have to confront, as courts begin to run out of reasons to limit exceptional approaches to causation to a few select diseases. The sub-sections below outline the common

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<sup>438</sup> See Chapter 2, section 1.4

<sup>439</sup> Cited from Gold (n 12) 277

<sup>440</sup> Michael Bliss, *William Osler: A Life in Medicine* (University of Toronto Press 2002) 480

<sup>441</sup> William Osler, 'On the Educational Value of the Medical Society' (April 1903) IX *Yale Medical Journal* 325

<sup>442</sup> AV Buchanan, KM Weiss and SM Fullerton, 'Dissecting Complex Disease: The Quest for the Philosopher's Stone?' (2006) 35 *International Journal of Epidemiology* 562, 562



problems that arise when attempting to fit evidence about disease into current legal models.

### ***2.1: General difficulties in studying the effects of toxins***

Injuries from toxic substances are unlike typical harms that tort law has generally addressed, such as road accidents, trespasses, and property damage.<sup>443</sup> As Richard Wright notes,<sup>444</sup> the chemical and biological processes that result in disease are poorly understood: in marked contrast to traumatic injury cases, the disease process is unobserved and unobservable as it occurs. Gold<sup>445</sup> defines a toxic tort plaintiff as one who claims that exposure to some chemical, radiological, or biological agent caused a disease. But as Gold further explains, a fundamental difficulty in proving such a claim is that exposure and disease usually do not correlate perfectly: some people get sick without exposure, and some people receive exposure without getting sick. It is a biological reality that disease causation is almost always multifactorial and complex. Added to this is infinite inter-individual biological diversity that biologists have to contend with in attempting to research disease. Thus, notes Cranor, establishing causation in toxic tort claims requires the claimant to rule out other possible contributory causes, which can include evidence from epidemiological studies, animal studies, or other toxicological studies.<sup>446</sup>

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<sup>443</sup> CF Cranor, *Toxic Torts* (n 15) 94

<sup>444</sup> R Wright, 'Probability Versus Belief' in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011), 205-206

<sup>445</sup> Gold (n 12) 244

<sup>446</sup> Cranor *Toxic Torts* (n15) 38

The idea that causation can be complex, and there may be multiple causes that can individually or together produce an outcome is not new or exclusive to disease research. Hart and Honoré note that John Stuart Mill was amongst the first philosophers of causation to provide an account of causal complexity.<sup>447</sup> There are, however, few areas of litigation where the limitations of causal knowledge are more apparent than in disease litigation. Although there are some single-cause infectious diseases that have necessary, sine qua non causes that might fit more easily within legal thinking of causation such as the but-for test (e.g. cholera, tuberculosis, influenza, etc.), this is not typical of a vast majority of the most major diseases. When it comes to the study of disease, it is very seldom that all the relevant causal mechanisms are understood.<sup>448</sup> Due to a variety of reasons, Cranor points out, scientists at present almost certainly do not yet know all the disease processes that early exposure to toxicants can initiate, promote, accelerate, catalyse, potentiate, facilitate or exacerbate. They may not know the lowest exposures during development that can contribute to risks, either immediately or later in life.<sup>449</sup> *The difficulties are compounded by the fact that different diseases can vary in their onset and progression e.g. disease may have a long induction period or a long latency period, which makes it harder to use legal yardsticks such as proximity in order to assess causal connections between tort and injury.*

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<sup>447</sup> Cited from Hart and Honoré, *Causation in the Law* (2nd ed., Clarendon Press 1985) 19

<sup>448</sup> KJ Rothman, S Greenland, C Poole and TL Lash, 'Causation and Causal Inference' in KJ Rothman, S Greenland and TL Lash (ed), *Modern Epidemiology* (Lippincott, Williams and Wilkins 2008) 27

<sup>449</sup> CF Cranor 'The Challenge of Developing Science for the Law of Torts' in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 270-71.

Further, chemical substances have special properties that make the identification of their causal properties difficult.<sup>450</sup> They can have long latency periods, and can operate through unknown, complex, molecular mechanisms that harm humans in ways that can remain hidden for years.<sup>451</sup> Carcinogens, reproductive toxicants, and neurotoxicants, for example, are ‘invisible and undetectable intruders.’<sup>452</sup> Further, many genes and epigenetic factors may affect toxic susceptibility, toxins may affect people in many ways, and many effects may result from more than one toxin.<sup>453</sup> So, for example, even though a particular genotype of the NAT2 gene makes it much more likely that a woman smoker will develop breast cancer, not all women of that genotype who smoke end up with breast cancer; some women who smoke develop breast cancer even though they do not have that genotype; some women develop breast cancer even though they neither smoke nor have that genotype.<sup>454</sup>

## ***2.2: Variable induction and latency periods***

The induction period of a disease is the period of time from causal action until disease initiation, while its latency period is the time from the disease occurrence to disease detection.<sup>455</sup> Although the latency period can be decreased by improved methods of disease detection, the induction period cannot be shortened by early detection of the disease.<sup>456</sup> Both the induction and the latency

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<sup>450</sup> Cranor, *Toxic Torts* (n 15) 92

<sup>451</sup> *ibid* 93

<sup>452</sup> *ibid* 93

<sup>453</sup> Gold (n 12) 275

<sup>454</sup> *ibid* (n12) 275-276

<sup>455</sup> Rothman, Greenland, Poole and Lash (n 76) 15-16

<sup>456</sup> *ibid* 16

periods can vary enormously across different diseases. Type 1 diabetes, for example, is a multifactorial, chronic disease with a long induction period, and little is known about its etiology.<sup>457</sup> Several cancers are also characterised by long induction periods.<sup>458</sup> Mesothelioma is a well-known example that lawyers are well acquainted with. However this, too, like anything in medicine, is highly variable. For some cancers, there will be some causal factors in the long chain of component causes that very quickly then lead to the disease onset, provided all the earlier triggering factors have been activated earlier.

A disease can also have variable latency periods: once initiated, many diseases are not apparent or detected straightaway.<sup>459</sup> For example, studies of Parkinson's disease reveal that early life exposure to some toxicants can lead to a long period of 'silent toxicity' usually lasting decades, in which certain parts of the brain operate at a suboptimal level before the disease manifests at a clinically recognisable level.<sup>460</sup> Similarly, toxicants may add to the adverse effects resulting from exposure to other toxic or naturally occurring substances. Thus, two or more toxicants may add to produce a disease, or a toxicant plus a naturally occurring chemical may together contribute to disease, accelerate and illness or worsen it.<sup>461</sup> The factors earlier in the causal chain may well have made a far more significant contribution to the ultimate outcome than the factors that were

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<sup>457</sup> LC Stene and M Rewers, 'Defining Causal Relationships Between Viral Infections and Human Diabetes' in K Taylor, H Hyoty, A Toniolo, A Zuckerman (eds), *Diabetes and Virus* (Springer 2013) 233

<sup>458</sup> Rothman, Greenland, Poole and Lash (n 76) 16

<sup>459</sup> *ibid*

<sup>460</sup> Cranor, 'The Challenge of Developing Science for the Law of Torts' (n 77) 270-71

<sup>461</sup> *ibid* 274

more proximate in time, and once the earlier contributory events have occurred, it may require a relatively small event to tip the scales and trigger disease. This makes it much harder to assess causation in the context of disease claims. An example of the legal dilemmas this can give rise to is the “Trigger” litigation,<sup>462</sup> which was a joint appeal brought by nine insurance companies regarding the extent to which the employers’ insurance policies covered them against negligence claims for mesothelioma (a disease, as mentioned earlier, with a long latency period: the disease typically manifests around 30 years after exposure). The insurers argued that the policies only responded where the mesothelioma had manifested during the insurance period: Lord Mance called this contention as the manifestation or occurrence basis of insurance liability.<sup>463</sup> The employers and employees representatives, on the other hand, argued that the policies should be triggered if the exposure allegedly happened during the insurance period: the exposure or causation basis. A particular difficulty was that the long gap between exposure and manifestation of disease meant that many employers had had multiple insurance policies in the intervening period. Further, it was difficult to know when exactly the disease was triggered. This, as Lord Mance noted, meant that significant gaps could arise in regard of the employers cover under the manifestation basis.<sup>464</sup> This gap, he noted, would also be open to deliberate misuse by insurers, who could escape liability by simply not renewing employers’ insurance policies.<sup>465</sup> The Supreme Court thus dismissed the insurers’ appeal, and held that the exposure or causation basis of liability was the correct one. However, the claim led to much debate, and Lord

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<sup>462</sup> *Durham v BAI (Run Off) Ltd.* [2012] UKSC 14, [2012] 1 WLR 867

<sup>463</sup> *ibid* [3]

<sup>464</sup> *Durham v BAI* (n 90) [21]-[24]

<sup>465</sup> *ibid* [25]

Phillips dissented with the decision. In a perplexing speech, Lord Phillips appeared to disagree with Fairchild, and to change his mind about how Fairchild should have been decided.<sup>466</sup> This case illustrates the many layers of legal dilemmas that can arise from the complexities of disease causation.

### ***Section 2.3: Single-cause versus multifactorial models of disease causation***

There is little doubt in current medical thinking that a large number of diseases that rank highest among the causes of death and disability today- including hypertension, coronary heart disease, respiratory disease, and cancer- are multifactorial in origin. Buchanan et al.<sup>467</sup> draw a contrast between complex diseases, and disorders resulting from simple Mendelian inheritance, infection, or environmental toxins, for which it is possible to identify a single causal agent that is both specific and highly predictive. Although a few diseases may be the result of a single sufficient and necessary cause, the majority of diseases are generally not the result of any single cause but rather of multiple causes- there is, usually, a constellation of causes that is responsible for a disease.<sup>468</sup>

When a cause of disease is both necessary and sufficient, or nearly so, it should be easier to spot and confirm.<sup>469</sup> However, in medicine, as Markum points out, the causal relationship is generally not a simple linear relationship between

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<sup>466</sup> See N McBride and S Steel, 'Case Comment: the "Trigger" Litigation' (2012) 28 Professional Negligence 285, 289-290

<sup>467</sup> Buchanan et al (n 70) 562-565

<sup>468</sup> J A Markum, *An Introductory Philosophy of Medicine: Humanising Modern Medicine* (Springer 2008) 36

<sup>469</sup> Buchanan et al (n 70)

cause and effect- the relationship is more likely to be complex and multifaceted. Asking such questions as whether a factor was 'sufficient' or 'necessary' for the disease only leads to controversy that often has no clear resolution. Because many complex diseases are caused by complex interactions between genetic factors (polygenic basis) and various environmental factors,<sup>470</sup> it is extremely difficult to unravel and isolate the individual factors and *precisely* assess their relative contributory role in the disease.<sup>471</sup> Thus, biomedical causation, notes Markum, is seldom strictly deterministic, but more often probabilistic.<sup>472</sup> However, most causes, whether sufficient or necessary, can be assigned a percentage in terms of their likely contributory role in a disease, and it may be much more worthwhile for the law to instead assess such questions in disease litigation. Chapter 4 (section 3) will suggest such an alternative approach to causation in disease claims.

Further, even single-cause models of disease causation are no longer viewed as being linear and simplistic. As research becomes more sophisticated, it appears that there may be many more contributory factors than had earlier even been conceptualized under the traditional biomedical model, which was founded on the now-overturned idea that every disease has a specific pathogenic origin.<sup>473</sup> The relatively newer area of medical sociology highlights that causality even in single-cause diseases may be more complex, and that social factors (such as social class, poverty, stress, lifestyle, unpleasant living conditions, socioeconomic disadvantage etc.) may play a significant contributory role in the development of

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<sup>470</sup> W Kirch, *Encyclopedia of Public Health* Vol 1 (Springer 2008) 970

<sup>471</sup> SL Mera, *Understanding Disease: Pathology and Prevention* (Stanley Thornes 1997) 3

<sup>472</sup> Markum (n 96) 36

<sup>473</sup> W Cockerham, *Social Causes of Health and Disease* (Polity Press, 2013) 6

*all* disease. There is now increasing evidence that social context can shape the disease's incidence, course and outcome- regardless of whether it is infectious, genetic, metabolic, malignant, or degenerative.<sup>474</sup>

All of this adds a further layer to the complexity of disease causation, and illustrates the difficulties in isolating 'necessary' or but-for causes of diseases. Increasing medical research appears to be adding to, rather than reducing, the list of factors that interact in various different ways to produce disease, and it is now vital for the law to recognise the importance of probabilistic reasoning in assessing the causal role an alleged toxin may have played in the outcome.

#### ***2.4: The under-appreciated role of stochastic factors in disease causation***

Courts appear to have long avoided confronting the repeated challenges posed by disease causation, by simply attributing these to inadequate science and deficiencies of existing research. This indicates an underlying judicial hope that as science advances, the problems will automatically be resolved, and scientists will sooner or later be able to provide much more conclusive proof of 'individualised', but-for causation in disease disputes. Many people seem to share this faith in the powers of science to eventually resolve all uncertainties. Cranor, for example, following much distinguished legal scholarship on the complexities of disease causation, states hopefully that 'as research tools are refined, and as more studies are conducted, causes for adverse outcomes may

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<sup>474</sup> *ibid* 1



well be more definitely identified, and more certain conclusions may transform how scientists think about, analyse and assign causal responsibility for various diseases.<sup>475</sup>

Sadly, this optimism about a future characterised by more or less complete causal certainty about disease may be misplaced. As Gold<sup>476</sup> argues, the opposite is true. Some scholars, he notes, have hoped that genomic and molecular information will at last provide scientific certainty — definitive, individualized proof of toxic causation. However, increasingly sophisticated scientific research into the interaction between human genes and environmental exposures suggests future extension, rather than resolution, of the problem of causal indeterminacy in toxic torts.<sup>477</sup> He cites genomic research to contend that actually, ‘at the highest magnifications, certainty will dissolve into probability.’ Better scientific evidence will, he believes, only strengthen the argument for doctrinal reforms in toxic tort litigation, and for legal approaches that can take account of probabilistic evidence about toxic substances.<sup>478</sup>

Coggon and Martyn<sup>479</sup> (professors of medicine and epidemiology, respectively) explain why such causal completeness and certainty may be a long time coming, given the seemingly infinite number of variables that often interact in infinite numbers of ways to produce disease. They describe the insurmountable problems with the expectation of complete answers from medical sciences, given

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<sup>475</sup> Cranor, ‘The Challenge of Developing Science for the Law of Torts’ (n 77) 262

<sup>476</sup> Gold (n 12) 237

<sup>477</sup> *ibid*

<sup>478</sup> Gold (n12) 237-238

<sup>479</sup> DIW Coggon and CN Martyn, ‘Time and Chance: The Stochastic Nature of Disease Causation’ (2005) 365 *Lancet* 1434-1437

the stochastic nature of disease causation: 'if they become ill, they demand a reason. If such a reason is not forthcoming, they imagine that this is because medical knowledge is still incomplete, and that further research will soon reveal the answer...Obviously, a patient with a disease must have been exposed to a combination of causes sufficient to induce that disease, but it does not necessarily follow that these causes are measurable or even identifiable. An analogy can be drawn with the throw of a die. The fact that a six is rolled does not mean that the die was heavily exposed to any risk factors for that outcome. Instead, this result is due to the operation of a complex set of circumstances (dimensions, shape, and weight of the die; the exact position of its centre of gravity; forces acting on it and its height above the ground when thrown; viscosity of the air; contour and elastic properties of the surface on which it landed, and so on), none of which could be shown to have a consistent effect on its own on whether the die rolls six.' Coggon and Martyn conclude, after reviewing the nature of disease causation, that the role of stochastic processes is underappreciated.<sup>480</sup> The enormous number of possible combinations of potentially interacting causal factors—genes, epigenetics, other individual characteristics, and exposures— makes it extraordinarily unlikely that complete risk characterization will ever be possible at an individual level.<sup>481</sup> This means that reliance on statistical probability computations may be the only way, within current scientific knowledge, of explaining and predicting disease occurrence.

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<sup>480</sup> *ibid*

<sup>481</sup> Gold (n 12) 278-279

### **SECTION 3: *Greater integration between legal and medical causation: how the law gets the science wrong***

This chapter has attempted to outline the current medical evidence about causation, and the problematic rigid legal approach to causation, in order to illustrate the extent of the divide between law and science in this area. Although lawyers have pinned their hopes on the idea that ‘better’ science will resolve all uncertainties about causation, increasing research into the causes of disease shows that ‘more’ science may increase, rather than narrow, this divide. Legal reform is imperative in the approach to factual causation.

The relationship between medicine and the law, point out Spicker *et al*,<sup>482</sup> has not always been characterised by mutual respect. An unhealthy antagonism between these professions can lead to isolation of each from each other, and discourages a collaborative interdisciplinary approach to problems of mutual concern. This chapter has attempted to contrast deterministic legal approaches to causation, and probabilistic scientific models of disease causation, to make the point that the lack of alignment between law and medicine is becoming an urgent and pressing problem in disease and toxic tort litigation. If lawyers are to understand and properly engage with probabilistic scientific reasoning, any residual disciplinary antipathy must be overcome. Training for both lawyers

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<sup>482</sup> SF Spicker, JM Healey and HT Engelhardt (eds.) *The Law-Medicine Relation: A Philosophical Exploration* (D Reidel, 1981) xiii-xxvii, xv

working in this area and for scientific expert witnesses could be one helpful strategy to bring about better understanding.

Undoubtedly, legal questions are different from scientific questions. Thus they can rarely be answered by a simplistic application of scientific studies. For example, medical research is generally focused on prospective forecasting of the effect of a particular agent on an outcome. The law, on the other hand, looks backwards to see what agent might have caused the outcome: the outcome is already known, and the analysis of causation requires a retrospective assessment of the clinical and biological evidence.<sup>483</sup> As Faigman puts it, "[w]hile science attempts to discover the universals hiding among the particulars, trial courts attempt to discover the particulars hiding among the universals."<sup>484</sup>

Dawid, a professor of statistics at the University of Cambridge, uses the terms 'effects of causes' (EoC) and 'causes of effects' (CoE) to define this distinction. The central question in the scientific inquiry usually pertains to the effects of causes, as future causal predictions are being attempted (e.g. 'will taking this aspirin cause my headache to disappear?'). Lawyers, on the other hand, are primarily interested in the 'causes of effects, which is a backward-looking question: e.g. 'did smoking cause my cancer?' This distinction, Dawid feels, is important because the mismatch requires a need to be cautious when we try to bring scientific evidence and reasoning to bear on questions of legal causality.<sup>485</sup>

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<sup>483</sup> Peter Feldschreiber, Leigh-Ann Mulcahy and Simon Day, 'Biostatistics and Causation in Medicinal Product Liability Suits', in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011), 189-190

<sup>484</sup> DL Faigman, *Legal Alchemy: The Use and Misuse of Science in the Law* (Freeman & Co 1999) 69

<sup>485</sup> AP Dawid, 'The Role of Scientific and Statistical Evidence in Assessing Causality', in Richard

However, perceptions about the differences can sometimes be exaggerated.

Faigman feels, for example, that science and medicine have spent relatively little time on etiological questions. The primary concern in clinical medicine, he opines, is on diagnosis and treatment, not on ascertaining etiology or causation: an oncologist might be curious about what caused the patient's leukemia, but his or her first task is to identify and treat the condition, not to determine whether a toxic substance, electromagnetic fields or genetic predisposition caused it.<sup>486</sup>

However, this view may need to be qualified. While it is true that lawyers are only interested in causes and etiology rather than diagnosis and treatment, both etiology and treatment are important in medical research. The two issues are not as disconnected as such commentators such as Faigman suggest. Despite the greater practical, pressing concerns about diagnosis and treatment, understanding causation is also important to medicine, because this can provide important diagnostic and treatment cues. As Markum notes, identifying the disease's causes is the first step often towards the possibility of treating the person's illness.<sup>487</sup> Thus a great deal of biomedical and epidemiological research remains devoted to causation.

The perceived chasm between law and science may be further exaggerated by the fact that courts apply artificial concepts and terminology about disease that bear little connection with any known medical or empirical evidence; and then tend to seek testimony about these as though they are empirical facts. An

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Goldberg (ed), *Perspectives on Causation* (Hart 2011) 134

<sup>486</sup> D Faigman, 'Judges as "Amateur Scientists"' (2006) 86 *Boston University Law Review* 1207, 1222

<sup>487</sup> Markum (n 96) 33

example of this is the legal distinction between ‘divisible’ and ‘indivisible’ disease, which is a concept invented by lawyers, but largely unknown to scientific experts who are often asked to give their opinion on the issue. The concept of an indivisible injury appears to have first been formulated, interestingly, in a defamation claim (*Associated Newspapers v Dingle*).<sup>488</sup> The term was actually used by Devlin LJ in *Dingle* to decide apportionment for different forms of damage that the claimant had suffered due to the libel, rather than in relation to disease: ‘The damage due to mental distress and to widespread repetition constitute, it may be said, an indivisible injury for which the damages cannot be separately assessed as between different publications of the same libel, and therefore each wrongdoer whose act is a substantial cause of the injury must pay for the whole.’<sup>489</sup> Indivisible injuries would entail joint and several liability, stated Devlin LJ, while divisible injuries would attract several liability.<sup>490</sup>

Since then, however, this artificial concept has become firmly enshrined in disease and toxic tort litigation through its application in *Fairchild*, which defined divisible disease as an injury that can be divided into component parts resulting from different causes. An indivisible injury, on the other hand, is defined in *Fairchild* as a more all-or-nothing concept, in the sense that the attribution of different parts to different causes is not possible.<sup>491</sup> The court in *Fairchild* drew a distinction between asbestosis (deemed to be divisible) versus mesothelioma (deemed to be indivisible).

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<sup>488</sup> [1961] 2 QB 162

<sup>489</sup> *ibid* [187]

<sup>490</sup> *ibid* [188]-[189]

<sup>491</sup> *Fairchild* (n 24) [21]-[27]

Feldschreiber et al take the view that distinction between divisible and indivisible diseases<sup>492</sup> was a consequence of the prevalent view about the etiology of mesothelioma at the time *Fairchild* was decided (i.e. the single-fibre theory of mesothelioma). However, the single fibre theory is now discredited,<sup>493</sup> and evidence suggests something more akin to a dose-response relationship for cases of indivisible injury such as mesothelioma: consequently, the theoretical distinction between divisible and indivisible injuries to begin to break down.<sup>494</sup> Further, the distinction is also likely to have been formulated partly to address judicial concerns about injustice to claimants due to the particular circumstances in *Fairchild*. The case (discussed in detail later in Chapter 4) involved multiple defendant employers who had exposed the claimant to asbestos, leading to mesothelioma (a disease with an especially long latency period, typically 30-40 years). This meant that the evidentiary hurdles faced by the claimants were particularly huge. Typically, mesothelioma claims also involve further difficulties because some of the defendants will be insolvent or will have simply stopped trading by the time the disease manifests. The judicial effort to protect claimants from risk of insolvency required the imposition of joint and several liability, and the causal questions appears to have simply been approached through backwards reasoning, so as to achieve the desired outcome. Regardless, the concept of divisible and indivisible disease appears to have taken on the status of empirical scientific fact for courts.

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<sup>492</sup> Feldschreiber et al (n 111) 193-194

<sup>493</sup> See *Sienkiewicz v Greif* (n 8) [101]; *Durham v BAI (Run Off) Ltd* [2012] UKSC 14 [31]

<sup>494</sup> Feldschreiber et al (n 111) 193-194

As this chapter has attempted to argue earlier, if the exigencies of a case require courts to take into account normative considerations in decision-making, it would be better for the clarity of the law to be transparent about this. Choosing, as they currently do, to twist causation reasoning in enigmatic ways in order to maintain the illusion that the decision is following some coherent legal principle does no service to the coherence of the law. Further, to view factual causation as an enquiry that can be shaped by what “feels” right renders causation meaningless. Such an approach also goes against the foundations of UK tort law: a causal link, as Professor Honoré notes, is necessary to both corrective justice and tort liability.<sup>495</sup>

The law, it is submitted, has avoided confronting the challenges of disease litigation for too long by believing that mesothelioma and asbestos-related injuries belong to some special category of diseases that occur only rarely. Courts have therefore convinced themselves that formulating an exceptional approach to asbestos cases (and those other cases that can bring themselves within the narrow criteria for exceptional tests for causation) is sufficient to do justice in the majority of claims. Unfortunately, the available evidence does not support this legal illusion. A review of the case law shows that despite legal reluctance, the exceptional tests are increasingly being extended to a growing number of disease scenarios that do not fit the conventional criteria for the ‘material contribution’ tests (such as most recently in cases such as *Heneghan v Manchester Dry Docks Ltd & Others*<sup>496</sup> and the Privy Council decision in *Williams v*

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<sup>495</sup> A Honoré, *Responsibility and Fault* (Hart Publishing 1999) 76

<sup>496</sup> [2016] EWCA Civ 86, [2016] 1 WLR 2036. This case will be discussed in greater detail in Chapter 4 (section 2.2)



*Bermuda Hospitals Board*<sup>497</sup>). Such is the complex nature of disease in general, that questions of 'but-for' causation for some of the most disabling illnesses is likely to tie scientists and courts in knots for the foreseeable future. Rising numbers of claims for diseases allegedly arising from toxic exposures ensures that ever-new dilemmas will be placed before courts. The urge to do justice within existing inadequate and ill-fitting causation principles such will lead to increasing confusion in the law of causation. It is urgent for courts to pay regard to the best available empirical evidence about disease and modify the approach towards disease causation. Chapter 4 examines the string of toxic tort litigation where the but-for test has repeatedly proven impossible for claimants to satisfy.

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<sup>497</sup> [2016] UKPC 4

## **CHAPTER 4:**

### **THE BUT-FOR AND EXCEPTIONAL TESTS FOR CAUSATION**

Chapter 3 highlighted the complexities of disease causation. It argued that the legal demand for science to provide precise answers to questions about the etiology of a specific claimant's disease, and about whether the tortious factor was necessary for the disease to occur, is a futile expectation- at least for now, and for the foreseeable future. Most major diseases can be caused in different ways, through interactions between many different factors. In that sense, complex disease litigation can be viewed as an area of the law where over-determined causation is the norm, rather than the exception. In addition, causal factors in disease litigation act on dynamic, infinitely variable biological systems that adapt and respond differently to different substances, rather than on inanimate objects with fixed, determinate properties. This makes it much more difficult to evaluate 'necessary' causal factors for disease, than, for example, a negligently caused fire in a building. Tort law, this thesis asserts, has completely ignored this problem in its rigid approach to causation.

While courts have tended to see the problems raised by disease litigation as arising primarily from deficiencies in current scientific knowledge, this author contends that the greater difficulty arises from the law's simplistic legal test for factual causation: the 'but-for' test, and its inability to take account of the multifactorial and stochastic nature of disease causation. This chapter will outline the problematic application of the but-for test in disease disputes, and

the situations where proven impossible to apply. This appears to have nearly always occurred in the context of complex disease claims. A central argument of this thesis is that it is no accident or coincidence that most of these dilemmas have arisen in disease litigation: this is simply further proof of the complexities of disease highlighted earlier in Chapter 3. Jones<sup>498</sup> notes that while causation is relatively straightforward in most cases, most difficult cases tend to arise in claims for occupational disease and some claims for medical negligence: difficulties which usually stem from scientific uncertainty.

Unfortunately, as this chapter will aim to show, situations where 'but-for' causes of a disease are impossible to identify are *not* exceptional, contrary to judicial beliefs. Although courts hold on to the idea that evidentiary uncertainties arise only in a narrow range of conditions (such as asbestos-related disease), the reality is that most major diseases have similarly uncertain causation. A cancer caused by smoking looks no different on a scan from a cancer caused largely by genetic factors. Hazy judicial recognition of the gross injustice of the 'but for' test in some scenarios has led to the development of more relaxed exceptional tests for factual causation, with poorly defined boundaries, on a case-by-case basis (most of these scenarios occurring in disease litigation). However, it is clear that courts experience much conflict in this regard. Legal outcomes in this area, as Jones points out, are more tied up in policy judgments about when claimants ought to succeed, and a judicial reluctance to translate policy judgements from

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<sup>498</sup> MA Jones, 'Proving Causation- Beyond the "But-for" Test' (2006) 22 Professional Negligence 251, 251

one type of claim to ‘with the result that outcomes on causation can seem to depend on the specific factual context rather than general principle.’<sup>499</sup>

This thesis contends that such an approach is bound to fail, as the slow extension of the exceptional tests to various different factual scenarios (especially in some very recent cases) clearly demonstrates. As the injustice of making such arbitrary distinctions between different diseases becomes more and more apparent, judges appear to have felt compelled to twist and contort causation reasoning in order to avoid injustice to deserving claimants facing insurmountable evidentiary gaps, while at the same time somehow still trying to maintain the illusion that the existent legal principles are still being applied.

This chapter proposes that such contortion of legal principles is both unnecessary and damaging. The need of the hour is more practical, evidence-based legal principles in disease litigation that better align with probabilistic scientific evidence about disease. Causal factors in medicine are usually assessed in terms of their likely role in the onset of disease (on their own, or synergistically), rather than in terms of the *necessity* of each individual factor to the illness. An example of this conceptualisation is the “causal pie” (or sufficient/component cause) model (Rothman et al 2008),<sup>500</sup> which explains disease as a product of several component causes, which *together* comprise a sufficient cause.<sup>501</sup> Thus causal tests in this area need to move away from a focus

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<sup>499</sup> *ibid*

<sup>500</sup> KJ Rothman, S Greenland, C Poole and TL Lash, ‘Causation and Causal Inference’ in KJ Rothman, S Greenland and TL Lash (ed), *Modern Epidemiology* (Lippincott, Williams and Wilkins 2008) 6-9

<sup>501</sup> *ibid*

on the *necessity* of the tortious factor, to an assessment of its likely contributory role in the disease.

Currently, courts prefer to apply the 'but for' test for causation as far as possible. The exceptional tests are only intended for very narrow circumstances, and courts have devised a variety of restricting criteria for their use (criteria which, as later discussions of recent caselaw will show, are proving unfeasible in practice, as there is no real rationale for many of these restrictive criteria). This thesis contends that the 'material contribution' tests, although poorly justified and arbitrarily applied, are at least a better fit in all but the most straightforward toxic tort cases than the but-for test, as they seem more able to take account of probabilistic and multifactorial causation. The haphazard nature of their use is problematic, however. In keeping with existent medical evidence, this thesis proposes that a more scientifically plausible test of general applicability to disease claims, such as a test for causation based on 'significant contribution to disease', on the balance of probabilities, may be a more semantically appropriate in this area of the law. Such a test would avoid many of the impossible-to-resolve controversies around whether liability is being imposed for injury or risk of injury. Further, importantly, it would align better with scientific models of disease and would thus allow more appropriate application of probabilistic scientific and epidemiological evidence to the causal assessment.

Most crucially, such an approach of general applicability to disease claims will not make arbitrary distinctions between chance factual circumstances.

Currently, courts decide whether or not to use more relaxed tests depending on

such factors such as whether the disease was divisible or indivisible (a distinction of the law's own invention, and one that Chapter 3 argued should be abolished); whether the different factors were similar or dissimilar; or whether the exposures happened concurrently or consecutively. This thesis contends that it is both unjust and unnecessarily confusing to make legal outcomes hinge on such yardsticks when, in fact, the same evidentiary uncertainties characterise most complex diseases. The current approach makes legal outcomes a matter of luck rather than justice, and the justification for the relevance of distinctions in legal analysis is too weak or absent altogether

While the probabilistic causal assessment suggested in this chapter lacks absolute certainty, the preceding discussions in chapters 2 and 3 have already highlighted that the quest for certainty is an illusory and misleading goal in science, particularly when the question pertains to the etiology of diseases. The role for evidence (including scientific evidence) in the law, it must be remembered, is not to verify which assertion is the ultimate truth, but only to decide whether the evidence points to the claimant's, or the defendant's assertion as being the more probable.<sup>502</sup> It will be recalled that Justice Jay recently acknowledged that:

Practical problems naturally arise in the law where a fact in issue cannot be proved by direct evidence but by inference. **The law may be based on a deterministic view of the natural world.** Causes may in theory be observed as they occur, but they will not have been if the past event has

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<sup>502</sup> F Taroni and C Aitken, 'Probabilistic Reasoning in the Law. Part 2: Assessment of Probabilities and Explanation of the Value of Trace Evidence other than DNA' (1998) 38(3) Science and Justice 179, 180

come and gone. The drawing of an inference is the only available pathway to proof.<sup>503</sup>

Further, this thesis asserts that if 'contribution to the injury' forms the basis of recovery in disease litigation, this can only be justified if compensation is proportionate to the extent of contribution as indicated by available evidence. The proposed new test only assesses the likelihood that a factor played a contributory role in the disease, and thus acknowledges that other factors would have played a part too. Thus, imposing liability for the whole injury on a defendant who may have only added to a part, or even none, of the harm would be disproportionate and excessive. Also, because most diseases can happen in many different (and often unknowable) ways, it shows due consideration of the fact that it is often impossible to be sure the tortious factor swung the outcome. Although a 'perfect' solution still remains elusive, the law should aim to minimise injustice as far as possible. Thus liability must be proportionate to the probable contribution of the factor to harm. Further, epidemiological methods can make a valuable contribution to such as assessment of likely contribution, even for what the law currently sees as 'indivisible' diseases. However, its potential has so far been under-utilised due to rigid legal adherence to naïve and unscientific causal concepts.

Section 1 of this chapter explores the traditional 'but for' test, and its use in disease claims. This test has been useful as a test for factual causation in some situations, while in other scenarios it has proved less useful. Disease claims, this

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<sup>503</sup> Sir Robert Jay, 'The Richard Davies QC Memorial Lecture 2015: "Standards of Proof in Law and Science: Distinctions without a Difference?"' (2016) *Journal of Personal Injury Law* 1, 3 (emphasis is added)

section argues, clearly belong in the latter category. Section 2 outlines the development of the 'exceptional' approaches, the narrow range of circumstances they were originally designed for, and the very slow (but steady) erosion of these restrictive criteria in recent cases. The most recent cases show that these exceptional tests are being applied to many diseases that do not fit the original criteria they were designed for, but in a confused and haphazard way. Section 2.1 will examine the 'material contribution to injury' (or *Bonnington*)<sup>504</sup> principle, section 2.2 will examine the 'material contribution to risk' (or *Fairchild*)<sup>505</sup> principle, and section 2.3 will examine the 'doubles the risk' test. Section 2.4 will outline the main issues about disease causation that are highlighted by the preceding discussion about the exceptional tests. Section 3 will suggest an alternative approach to causation that is more principled and justifiable, as well as more congruent with current medical and scientific understanding of disease. Epidemiology can play a valuable role in such a probabilistic assessment of causation.

## **SECTION 1: The traditional approach to factual causation**

Chapter 3 (at section 2) earlier discussed the biological reasons for the unsuitability of a deterministic approach to the causes of disease. Prosser summarises the but-for or *sine qua non* rule as follows: 'The defendant's conduct is not the cause of the event, if the same event would have occurred without it.'<sup>506</sup> Thus, necessity is a central component of the but-for test. The problem with

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<sup>504</sup> *Bonnington Castings Ltd v Wardlaw* [1956] AC 613

<sup>505</sup> *Fairchild v Glenhaven Funeral Services Ltd* [2002] UKHL 22

<sup>506</sup> WL Prosser, 'Proximate Cause in California' (1950) 38 California Law Review 369, 377



complex disease, as Chapter 3 demonstrated, is that the same disease can be caused in so many different ways, through so many different combinations of factors, that experts can almost never be sure that *this* disease would never have happened but for *that* part of the event. How, then, in such a scenario, is but-for causation to be established?

The normal rule for factual causation is the but-for test, which requires the claimant to establish that injury would not have occurred but for the defendant's negligence. This must be proven on the balance of probabilities: if it is more likely than not that the event was the cause, it is treated as if it was the cause.<sup>507</sup> Thus, as Steel and Ibbetson note, the normal rule of causation, which applies in almost all situations, has two aspects: the evidential and the conceptual. In order to establish liability, P has to show on balance of probabilities (evidential) that but for D's wrongful conduct the injury would not have occurred (conceptual).<sup>508</sup>

One major advantage of the but-for test is its simplicity, assuming that a definitive answer can be given to the question.<sup>509</sup> The test is thus well suited to answer straightforward causal questions. If, however, there is not a straightforward answer to the conceptual question, or we do not have sufficient evidence, the test can fail completely, or yield illogical outcomes. Even outside of the disease context, it is generally accepted that one situation where the but-for test is comes under stress in situations of over-determination,<sup>510</sup> that is, when

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<sup>507</sup> Lunney M and Oliphant K, *Tort Law* (5<sup>th</sup> edn, OUP 2013) 205-206

<sup>508</sup> S Steel and D Ibbetson, 'More Grief on Uncertain Causation in Tort' (2011) 70 *Cambridge Law Journal* 451, 452

<sup>509</sup> Lunney and Oliphant (n 10) 216

<sup>510</sup> *ibid*

there are multiple causal mechanisms that could result in the same effect. Staunch supporters of the but-for test such as Green also acknowledge that it is unable to deal with 'over-determined' cause scenarios.<sup>511</sup> Green, however, continues to advocate the but-for test despite its inadequacy in dealing with overdetermined causes, on the grounds this is, in her view, mostly just a theoretical rather than a practical problem. Firstly, contends Green, '...there are relatively few cases of this nature which actually trouble the courts,' and secondly, in Professor Green's opinion, 'judicial pragmatism' has usually been sufficient to prevent injustice.<sup>512</sup> This thesis, with respect, disputes both these contentions, at least as far as disease litigation is concerned. It argues that in disease litigation, over-determined cause scenarios are the exception rather than the norm. As a result of the frequency with which such situations arise in this area of the law, high levels of recourse to 'judicial pragmatism' and creativity have led to unacceptable levels of confusion in the law of causation. The extent of current incoherence in tort law as a result of the haphazard application of the exceptional tests for causation suggests the danger of 'judicial pragmatism' becoming simply another term for a mostly subjective approach to factual causation.

Members of the judiciary have on a number of occasions also expressed more general and wider concerns about the 'but for' test. This demonstrates that a rigid, simplistic, one-size-fits-all concept of causation may be insufficient to take account of, and deliver justice in, many situations. As the Canadian Supreme

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<sup>511</sup> Such as, for example, S Green, *Causation in Negligence* (Hart 2015) 9

<sup>512</sup> *ibid* 8-11

Court judge, McLachlin J, extra-judicially pointed out<sup>513</sup>:

*Why are courts asking questions that for decades, indeed centuries, did not pose themselves, or if they did, were of no great urgency? I would suggest that is because all too often the traditional but-for, all-or-nothing, test denies recovery where our instinctive sense of justice- of what is the right result for the situation- tells us the victim should obtain some compensation.*

(McLachlin J's reference to the all-or-nothing test, it must be noted, also raises important questions about the fairness of joint and several liability in complex disease cases, where multiple factors are often involved, and where evidentiary uncertainties are the norm. The principle of joint and several liability is currently applied in mesothelioma cases due to section 3 of the Compensation Act 2006, but it can also be extended to other diseases deemed 'indivisible'. McLachlin J's concerns about the need for victims to receive some compensation suggests a belief that outcomes might be fairer, and reasoning more principled, if more moderate approaches such as proportionate liability are the norm. Issues of apportionment will also be briefly discussed in Section 3 later in this chapter).

Lord Bingham noted, in *Fairchild v Glenhaven Funeral Services Ltd*,<sup>514</sup> some of the problems with the test, and its limited utility:

*The but-for test gives rise to a well-known difficulty in cases where there are two or more acts or events which would each be sufficient to bring about the plaintiff's injury...the test, applied as an exclusive criterion of causation, yields unacceptable results and the results which it yields must be tempered*

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<sup>513</sup>CJ McLachlin, 'Proving the Connection' in N Mullany & A Linden (eds), *Torts Tomorrow: A Tribute to John Fleming* (1998 LBC Information Services) 16 (quoted from *Fairchild* (n 8) at [11] (Lord Bingham))

<sup>514</sup> *Fairchild* (n 8)

*by the making of value judgments and the infusion of policy considerations.*<sup>515</sup>

Lord Bingham appeared to take the view that the only course left open to courts was to ‘temper’ the rigidity of the but-for test by deciding factual causation on the basis of normative considerations. However, this thesis has already made the case (in Chapter 3 section 1) that assessing factual causation on the basis of value judgments is damaging to the ultimate aims of tort law (which is not to deny a role for normative considerations in other strands of the negligence enquiry such as remoteness, duty of care etc.). To decide on factual matters without giving primacy to objective evidence would be to make the factual assessment a hollow exercise.

The Court of Appeal recognised some significant difficulties that beset disease causation in its recent decision in *Heneghan v Manchester Dry Docks Ltd*<sup>516</sup> This involved a claim where the biological evidence could not establish which (if any) of the exposures triggered the cell changes in the claimant’s body; but where epidemiological and statistical evidence was able to establish how much the exposure attributable to each defendant increased the risk that he would contract the disease (the case will be discussed later in section 2.2 below). The Court of Appeal noted<sup>517</sup> the submission of the defendant’s expert, Dr Rudd, who made the point about the problematic legal approach to disease causation clearly during his re-examination: ***“Where we have a process which is essentially random, a series of accidents, a stochastic process, it is never going to be***

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<sup>515</sup> *Fairchild* (n 8) at [10]- [12]

<sup>516</sup> [2014] EWHC 4190 (QB)

<sup>517</sup> *ibid* at [31] (emphasis added)

*appropriate to have a deterministic model of causation. You are never going to say this employer contributed to this fibre which had that effect on that cell.”*

The bewildering range of arguments that claimants have made for an extension of exceptional tests to several diseases other than the restricted disease scenarios they were intended for (as we shall examine at length in section 2 below) demonstrates this problem. Disease causation is prone to inherent evidentiary uncertainties and as well as to over-determined causation. Both these factors in combination make the but-for test fundamentally unsuited to this area of the law, and this has ensured that the most difficult causal dilemmas arise with unfailing regularity in complex disease claims. The section below will outline the development and further applications of the exceptional tests for factual causation. It will highlight the incremental extensions of these tests to a growing range of different diseases in recent case law: a development that, this thesis argues, is inevitable because of the nature of disease.

## **SECTION 2: The ‘exceptional’ tests for factual causation**

Over the years, courts have created a number of exceptions to the traditional but-for test, which have evolved on a case-by-case basis. The need for the exceptional tests has arisen due to evidentiary difficulties that make it impossible for a claimant to satisfy the but-for test. An evidentiary gap scenario occurs when insufficient scientific knowledge or evidence make it impossible for the claimant to prove on the balance of probabilities that their injury or disease

would not have occurred without the involvement of the allegedly tortious factor.<sup>518</sup>

The most well-known of these exceptional tests for factual causation, which are almost always invoked in complex disease claims, are the ‘material contribution to injury’ (or *Bonnington*) test and the ‘material contribution to risk’ test (otherwise called the *Fairchild* test). Although the *Fairchild* test, in particular, has sparked tremendous controversy that continues even today, a string of prior cases had already paved the way for the development of the *Fairchild* principle. The section below will trace the development of the material contribution tests, as well as their ongoing applications, which are often problematic. In addition, it will also outline the third exceptional approach to disease causation: the ‘doubling of the risk’ (DOR) test, which is based on a somewhat peculiar judicial understanding of epidemiological evidence, and which is usually called into play when courts encounter scenarios which do not fit the criteria for the material contribution tests.

## ***2.1: The ‘material contribution to injury’ (Bonnington) test***

### ***2.1.1: Initial formulation***

The first clear relaxation of the standard but-for approach to causation occurred in the case of *Bonnington Castings Ltd v Wardlaw*.<sup>519</sup> The claimant here was an

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<sup>518</sup> See G Turton, *Evidential Uncertainty in Causation in Negligence* (Hart Publishing 2016) 81-86

<sup>519</sup> (n 7)

employee who had contracted pneumoconiosis following exposure to air containing particles of silica during his employment. While some of this exposure occurred without fault on part of the defendant (through “innocent” exposure), the quantity of noxious dust was increased due to faultily maintained swing grinders (the negligent exposure). Thus, some part of the exposure was due to tortious factors, while some of it arose due to non-tortious factors, and both these exposures occurred simultaneously. Due to evidentiary uncertainties, the claimant was unable to establish that he would have suffered the same outcome ‘but for’ the tort: pneumoconiosis is caused by gradual build-up of such toxins in the lungs. There was at the time no known way of preventing some part of the exposure. Thus the dispute was only in regard of the additional exposure due to the employer’s negligence. However, the question of whether the disease would have happened anyway even without negligence was impossible to answer on available medical evidence due to the multiple sources of exposure. The House of Lords held that in this instance it was sufficient for the claimant to establish that the breach had made a material contribution to his injury, and on these grounds, ruled in the claimant’s favour. Waller LJ referred to causation being established:

*...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.<sup>520</sup>*

This test is now commonly referred to as the material contribution to injury, or *Bonnington* test, and has been applied across a string of subsequent caselaw.

Anderson<sup>521</sup> notes that what this case did not decide (because it was never argued) was the extent of the defenders' liability. It is unclear, notes Anderson,

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<sup>520</sup> *ibid* [46] (Waller LJ)

whether the defendants would have only been liable for the aggravation of the disease which the negligent dust caused, had medical evidence been presented to argue that the dust from the “non negligent” source had been the main cause of the disease. But the House did not consider the issue of divisibility, so pneumoconiosis was treated as an indivisible disease, and full liability was imposed on the defendant.

The Court of Appeal did, however, consider the point in the later case of *Holtby v Brigham and Cowan (Hull) Ltd.*<sup>522</sup> The plaintiff had worked as a marine fitter for nearly 40 years, and during the course of his employment he was exposed to asbestos for significant periods. He had worked with the defendants for nearly 12 years during this time. The claimant developed asbestosis and sued the employer who had exposed him to asbestos for the longest period. Stuart-Smith LJ held that the evidence indicated that asbestosis had resulted from and been aggravated by cumulative exposure to asbestos. Thus the defendant was only held liable for the part of the damage they had caused. Although the court found quantification difficult, Stuart-Smith LJ emphasised that the best that could be done was only to minimise injustice to either party as far as possible: “The court must do the best it can to achieve justice, not only to the claimant but the defendant, and among defendants.”<sup>523</sup>

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<sup>521</sup> GM Anderson, ‘Disease Causation and the Extent of Material Contribution’ (2006) 15 Scots Law Times 87, 93-94

<sup>522</sup> [2000] 3 All ER 421 CA

<sup>523</sup> *ibid* at [20]



## ***2.1.2: Inconsistencies, further applications and extensions of Bonnington***

The *Bonnington* principle left many questions unanswered about its scope and applicability: what exactly are the situations in which this test will and will not apply? Despite many judicial inconsistencies in its actual use, the *Bonnington* test is conventionally understood in academic analysis to be intended for restricted and very specific factual scenarios. For example, Green interprets it as applicable where there are multiple causal factors<sup>524</sup> that operate concurrently rather than consecutively,<sup>525</sup> and where the factors cumulatively contribute to the harm.<sup>526</sup> McIvor further cites cases such as *Thompson v Smiths Shiprepairers (North Shields) Ltd*<sup>527</sup> and *Holtby v Brigham and Cowan (Hull) Ltd*<sup>528</sup> to note that the *Bonnington* principle is usually applicable to claims for divisible diseases.<sup>529</sup> However, as McIvor<sup>530</sup> further notes, there have been many contrary judicial interpretations, as indicated by Lord Phillips' obiter approval of its application to lung cancer, which is indivisible.<sup>531</sup> This is consistent with many recent cases where courts have interpreted the rule to be applicable to many situations that fall outside the restricted criteria. The 'material contribution to injury' test has, since its original formulation, been applied to a number of different variations of multiple-potential cause situations.

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<sup>524</sup> Green, *Causation in Negligence* (n 14) 94

<sup>525</sup> S Green, 'Case Comment: When is a Material Contribution Not a Material Contribution?' (2016) 32 Professional Negligence 169, 170 (hereafter, 'Case Comment: Material Contribution..')

<sup>526</sup> *ibid*

<sup>527</sup> *Thompson* [1984] QB 405

<sup>528</sup> *Holtby* (n 25)

<sup>529</sup> C McIvor, 'The 'Doubles the Risk' Test for Causation and Other Related Judicial Misconceptions About Epidemiology' in *Tort Law: Challenging Orthodoxy*, S Pitel, JW Neyers and E Chamberlain (eds) (Hart 2013) 228

<sup>530</sup> *ibid*

<sup>531</sup> *Sienkiewicz v Greif* [2011] UKSC 10 at [75] (Lord Phillips)

Steel<sup>532</sup> is of the opinion that the law is 'in a mess' as far as the material contribution test is concerned: 'Material contribution has at least two meanings. They are not properly distinguished. They have nothing in common. In one meaning, found in *Bonnington*, negligence will be said to have "materially contributed" to the claimant's damage where, but for the defendant's negligence, *some part* (of indeterminate size) of the claimant's damage would not have occurred.'<sup>533</sup> But, Steel further notes, '*material contribution*' also has another meaning, found in *Bailey v Ministry of Defence*.<sup>534</sup> Here a defendant's negligence is said to have contributed to the claimant's injury if it played a significant role in the physical process by which that injury was produced.'<sup>535</sup>

In *Bailey v Ministry of Defence*,<sup>536</sup> the Court of Appeal ruled in favour of a claimant who had suffered brain damage due to severe weakness that could have resulted from either the defendant's negligence, or her own physiological problems (pancreatitis), or both. The claimant argued that the weakness resulted from a combination of the two, and thus that the *Bonnington* principle of material contribution to injury should apply. The defence disputed this point. However, the Court of Appeal in *Bailey* held that the negligence had made a material contribution to the injury and that the two sources of 'weakness' had cumulatively the ultimate asphyxiation. The court thus ruled that factual causation was established. This decision has resulted in much debate about the

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<sup>532</sup> S Steel, 'On When *Fairchild* Applies' (2015) 131 LQR 363

<sup>533</sup> *ibid* 364

<sup>534</sup> [2008] EWCA Civ 883; [2009] 1 WLR 1052

<sup>535</sup> Steel, 'On When *Fairchild* Applies' (n 35) 364

<sup>536</sup> *Bailey* (n 37)

boundaries of the material contribution test, and come in for a fair share of criticism. Bailey, for example, argues that the decision in *Bailey* is wrong, and that it should not constitute authority for the expanded 'material contribution' concept.<sup>537</sup>

Bailey also further notes another fundamental ambiguity at the heart of *Bonnington Castings Ltd v Wardlaw*: it is unclear whether the 'material contribution' rule involves an *application* of the but-for principle or an *exception* to that rule'.<sup>538</sup> There is a lack of clear consensus on this issue in academic commentary. Bailey, for instance, is of the opinion that the material contribution test is an application of the orthodox but-for rule, and not an exception to it.<sup>539</sup> Stapleton and Steel, on the other hand, opine that the principle 'must be understood as employing a causal concept which is broader than but-for causation.'<sup>540</sup>

Additional inconsistencies arise in relation to the question of whether the test applies only when the two factors operate concurrently rather than consecutively. Green<sup>541</sup> takes the view that the simultaneous exposure in *Bonnington* was central to the need for the material contribution to injury test, as there was no way that the court could divide up the claimant's injury into discrete sections to correspond to the two different sources of toxin. In Green's<sup>542</sup> assessment, had the two sources of exposure occurred consecutively

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<sup>537</sup> SH Bailey, 'Causation in Negligence: What is a Material Contribution?' (2010) 30 Legal Studies 167, 184-185

<sup>538</sup> *ibid* 174

<sup>539</sup> Bailey (n 40) 167

<sup>540</sup> J Stapleton and S Steel, 'Case Comment: Causes and Contributions' (2016) 132 LQR 363, 365

<sup>541</sup> Green, 'Case Comment: Material Contribution...' (n 28) 170

<sup>542</sup> Green (n 28) 170

as opposed to concurrently, the problem would not have arisen: *Bonnington* could have been decided on straightforward causal principles, obviating the need for a material contribution analysis. However, the Privy Council in the recent case of *Williams v Bermuda Hospitals Board*<sup>543</sup> used the material contribution to injury test to uphold a claim for conditions that involve multiple causes that did not operate concurrently but consecutively. In *Williams*, the claimant suffered sepsis due to a combination of two factors: surgery due to appendicitis as well as negligent delay on part of the hospital which, he alleged, caused post surgery complications. The resultant complications were held to be due to a combination of both factors, but the claimant struggled to bring his claim within the *Bonnington* exception due to the fact that both factors did not act concurrently, but consecutively. Nonetheless, the Privy Council chose to extend *Bonnington* to also include situations where the causes are concurrent.

Further, previous caselaw had indicated that the *Bonnington* test only applies when multiple similar factors cumulatively result in the injury. Thus, in *Hotson v East Berkshire HA*,<sup>544</sup> the lack of evidence about cumulative operation of the two factors resulted in the court refusing to apply the material contribution to injury test, despite the negligence increasing the likelihood of the injury. The claimant in *Hotson* fell while climbing a tree and suffered ruptured blood vessels, but the hospital failed to diagnose this until five days later, leading to avascular necrosis

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<sup>543</sup> *Williams* [2016] UKPC 4 (Privy Council)

<sup>544</sup> [1987] AC 750 (HL)

of the epiphysis. Evidence indicated that both the fall, as well as the hospital's failure to diagnose/intervene on time, could have contributed to the injury, independently or cumulatively. Lord Bridge stated that unless the claimant proved on a balance of probabilities that the delay in treatment was at least a contributory cause of the avascular necrosis, he failed on causation, and that the evidence amounted to a finding of fact that the fall was the sole cause of the avascular necrosis.<sup>545</sup> *Hotson* was distinguished from *Bonnington* partly on the grounds that the injury in *Hotson* was not *cumulatively* caused by a combination of factors, but by one or the other of different potential causal factors (i.e. the fall itself or the negligent delay).<sup>546</sup> Similarly, in *Wilsher v Essex Health Authority*,<sup>547</sup> medical evidence indicated that there were four separate potential causes for the claimant's blindness in addition to the tortious cause, any of which could have independently caused the condition. The claim failed for lack of but-for causation on the balance of probabilities due to fact that the different potential causes were not similar to each other. The House of Lords in *Wilsher* distinguished *Bonnington*, where the injury was caused by a single known process (the inhalation of dust), and refused to apply the *Bonnington* test for causation.

On the other hand, the outcome was very different in the very recent case of *John v Central Manchester and Manchester Children's University Hospitals NHS Foundation Trust*,<sup>548</sup> which was factually substantially similar to *Hotson*<sup>549</sup> and

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<sup>545</sup> *ibid* [782] (Lord Bridge)

<sup>546</sup> For a further analysis, see J Stapleton, 'Unnecessary Causes' (2013) 129 LQR 39, 49-50

<sup>547</sup> [1988] AC 1074

<sup>548</sup> *John* [2016] EWHC 407 (QB)

<sup>549</sup> *Hotson* (n 47)

*Wilsher*,<sup>550</sup> in that the situation involved multiple different potential factors that could have independently or cumulatively caused the injury. The court in *John* applied the material contribution to injury test to a claim where the claimant was left with long-term cognitive damage following a combination of an initial head injury and subsequent hospital delay in providing appropriate investigation and treatment. The claimant alleged that there was a negligent delay in the performance of the CT brain scan, and in not requesting an ambulance sooner. Thus the claimant's argument was that but for the defendant's negligence, he would have avoided some or all of the dangerously raised inter-cranial pressure that materially contributed to the brain damage that he had sustained from the initial head injury.<sup>551</sup> Expert evidence indicated that both the initial head injury as well as the raised intracranial pressure following the negligent delay could have caused or contributed to the brain damage,<sup>552</sup> thus but-for causation was impossible to establish. Picken J held that the 'material contribution to injury' approach applies in both single agency and multiple factor cases, awarding the claimant the entirety of damages. The court in *John* discussed *Bonnington* at length,<sup>553</sup> and cited Lord Reid as having established that the material contribution test was of general application. The court also referred to Waller LJ in *Bonnington* who saw causation being established:

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<sup>550</sup> *Wilsher* (n 50)

<sup>551</sup> *John* (n 51) at [45]

<sup>552</sup> *John* (n 51) [103]

<sup>553</sup> *John* (n 51) [84]

*...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.*<sup>554</sup>

Professor Green rues the extensions of *Bonnington* to cases such as *Williams* as “unfortunate,”<sup>555</sup> because: ‘making a material contribution to damage which is already bound to happen is not something which should attract liability in negligence.’<sup>556</sup>

However, in the view of this author, the judicial approach of cases such as *Williams* and *John* is surely the correct one (at least in terms of its final outcome, which is consistent with the argument of this thesis: that there is no justification for arbitrary distinctions between diseases in whether more flexible tests for causation should apply). Nevertheless, the significance of these recent decisions for present purposes is that it demonstrates ongoing disputes amongst experts about the remit of the *Bonnington* test.

## **2.2 The ‘material contribution to risk’(Fairchild) test**

### **2.2.1: Initial formulation**

The antecedents for the *Fairchild* principle were laid in the much earlier case of *McGhee v National Coal Board*.<sup>557</sup> The scenario in *McGhee* was more complicated than *Bonnington*, and resulted in a far more significant relaxation of the but-for

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<sup>554</sup> *Bonnington* (n 7) at [46]

<sup>555</sup> S Green, ‘Case Comment: Material Contribution...’ (n 28) 171-172

<sup>556</sup> *ibid* (n 28) 172

<sup>557</sup> [1973] 1 WLR 1

rule than the material contribution to injury test. *McGhee* involved a claimant employee who developed dermatitis following exposure to brick dust during his period of employment in a brick kiln. While some of this exposure was inevitable due to the nature of the work, it was compounded by the employers' failure to provide adequate washing facilities in the workplace. Doctors could only say that the effect of the two factors was cumulative, but could not be sure that the disease would not have occurred but for the failure to provide washing facilities, or even that this had made a material contribution to the injury. The House of Lords ruled that in the absence of complete medical knowledge of all the material factors relating to the disease (a statement that would, as discussed in Chapter 3, accurately describe the situation regarding most major diseases), materially increasing the *risk* of injury could also be sufficient to establish factual causation. The claim succeeded. *McGhee* thus represented a further dilution of the test for factual causation that *Bonnington*, and laid the foundation for the even broader 'material contribution to risk' test.

The *McGhee* test fell into disuse for some decades subsequently, but was resurrected in the landmark ruling in *Fairchild v Glenhaven Funeral Services*.<sup>558</sup> *Fairchild* concerned three conjoined appeals where the claimants had all contracted mesothelioma after having been exposed to asbestos during separate periods of employment with several different employers. They were unable to establish factual causation due to two problems: (i) gaps in medical knowledge about mesothelioma and its etiological mechanisms and (ii) the fact that the claimant had worked for multiple defendants, each responsible of whom were

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<sup>558</sup> *Fairchild* (n 8)



responsible for a substantial degree of exposure.<sup>559</sup> Due to the long and variable latency period of mesothelioma, and due to the single-fibre theory of mesothelioma prevalent at the time, it was impossible to determine which individual employer's exposure had materially contributed to the disease.

The *Fairchild* test was formulated to apply to 'evidential gap' cases involving multiple exposures to a single causal agent. Lord Bingham referred to the "rock of uncertainty":<sup>560</sup> the fundamental problem that it was impossible for the claimant to prove their claim if the conventional rules of causation (including the *Bonnington* 'material contribution to injury' test) were applied, even though the defendant had significantly increased the risk that the claimant would suffer injury. Under the *Fairchild* principle, claimants can establish factual causation simply by showing that the defendant's negligence has materially contributed to the *risk* of the relevant harm being sustained. This was intended as a policy-based response to the specific causal difficulties encountered by mesothelioma victims who had been negligently exposed to asbestos by multiple consecutive employers. However, the House of Lords did not specifically limit the material contribution to the risk of harm principle to this particular context, as McIvor notes.<sup>561</sup> Mesothelioma was also deemed to be an 'indivisible' disease, a legal term that is applied to diseases where it is believed that once triggered, further exposure to the triggering factor (here, asbestos) will not further worsen or aggravate the disease. This led to full liability being imposed on the defendant.

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<sup>559</sup> C McIvor, 'Debunking Some Judicial Myths About Epidemiology and its Relevance to UK Tort Law' (2013) 21 *Medical Law Review* 553, 556-557

<sup>560</sup> *Fairchild* (n 8) at [7] (Lord Bingham)

<sup>561</sup> McIvor, 'Debunking...' (n 62) 556

Lord Nicholls in *Fairchild* felt it necessary to clarify that in the unusual approach taken by the House towards causation:

*..the court is not... concluding that the ordinary 'but for' standard of causation is satisfied. Instead, the court is applying a different and less stringent test. It were best if this were recognised openly.*<sup>562</sup>

### **2.2.2: Inconsistencies, further applications and extensions of *Fairchild***

A similar scenario to *Fairchild* arose again a few years later in *Barker v Corus*.<sup>563</sup> This involved a claim for mesothelioma by an employee who could not pinpoint which employer's exposure to asbestos had triggered his mesothelioma. He had been negligently exposed to asbestos by a number of different employers, and an additional complicating factor was that he had also faced work-related asbestos exposure during a period of self-employment. Although the House of Lords allowed the claimant to succeed by applying the 'material contribution to risk' test, the court in *Barker* also recognized the potential injustice of imposing full liability on a defendant who may have only imposed part (or possibly, none) of the ultimate harm. *Barker* therefore attempted to ameliorate the harshness of the *Fairchild* rule by imposing proportionate liability on the defendant employer (proportionate to the time the claimant spent in his employment). However, the proportionate recovery ruling of *Baker* led to a public outcry, and Parliament

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<sup>562</sup> *Fairchild* (n 8) at [45] (Lord Nicholls)

<sup>563</sup> *Barker v Corus (UK) plc* [2006] UKHL 20, [2006] 2 AC 572

intervened to ensure that restricted recovery under the *Fairchild* rule is now no longer possible in regard of mesothelioma claims. The Compensation Act 2006<sup>564</sup> now rules out proportionate liability in claims for mesothelioma linked to negligent asbestos exposure.

The *Fairchild* 'material contribution to risk' test, points out McIvor,<sup>565</sup> is quite remarkable for several reasons. It does not require proof of the existence of any actual causal connection between the relevant breach of duty and the actionable damage. Further, it demands very little by way of proof of risk creation. Added to this is the fact that mesothelioma is held to be an indivisible disease, thus the defendant will be held to be liable for the entirety of the harm: even if there is a good chance that their tort may not have made *any* contribution at all to it.

Clearly, this is problematic for many reasons. For one, the potential for injustice to a defendant is obvious. A further problem with the *Fairchild* test is the poor reasoning and articulation of its rationale (apart from the catch-all reference to 'policy'). Neither is there clarity about the circumstances in which these policy justifications will apply. After having powerfully advocated wide leeway for courts to decide causation answers as they saw fit (discussed extensively earlier in Chapter 3 section 1), Lord Hoffmann was anxious to reiterate that courts follow principled and transparent lines of reasoning. Discussing *McGhee*, Lord Hoffmann distanced himself from its 'legal fictions':

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<sup>564</sup> Compensation Act 2006, s 3

<sup>565</sup> McIvor, 'Debunking...' (n 62) 558

*When some members of the House said that... there was no distinction between materially increasing the risk of disease and materially contributing to the disease, what I think they meant was that, in the particular circumstances, a breach of duty which materially increased the risk should be treated as if it had materially contributed to the disease. I would respectfully prefer not to resort to legal fictions and to say that the House treated a material increase in risk as sufficient in the circumstances to satisfy the causal requirements for liability.*<sup>566</sup>

However, by repeatedly dismissing the need for consistency in causation principles in practice as Lord Hoffmann does, for example, in an extra-judicial article <sup>567</sup> (also referred to earlier in Chapter 3), whilst continuing to maintain in theory that coherent, principled reasoning is being followed, Lord Hoffmann is, it is submitted, resorting to an even bigger legal fiction.

The *Fairchild* approach has been received with very little warmth by the judiciary. At the same time, however, as we shall see further in this section, the *Fairchild* test is also being extended to a broader range of situations that were not originally envisaged as being within the scope of this test. The Supreme Court described a recent case centering on the *Fairchild* principles (*International Energy Group Ltd. v Zurich Insurance plc*<sup>568</sup>) as “yet another demanding chapter in [that] difficult series of decisions”<sup>569</sup> that has unleashed “a sort of juridical version of chaos theory.”<sup>570</sup> Lord Sumption in the same case also pointed out that *Fairchild* had already “cruelly exposed the problem of dealing with complex

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<sup>566</sup> *Fairchild* (n 8) at [65] (Lord Hoffmann)

<sup>567</sup> Rt Hon Lord Hoffmann, ‘Causation’ in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 8-9

<sup>568</sup> [2015] UKSC 33; [2015] 2 W.L.R. 1471 (“*Zurich*”)

<sup>569</sup> *ibid* at [189] (Lord Neuberger and Lord Reed)

<sup>570</sup> *ibid* at [191] (Lord Neuberger and Lord Reed)

and interrelated issues piecemeal.”<sup>571</sup> Baroness Hale<sup>572</sup> in *Sienkiewicz* acknowledged the difficulties created by the *Fairchild* approach, which she felt had “kicked open the hornets' nest”:

*I find it hard to believe that their Lordships there foresaw the logical consequence of abandoning the “but for” test: that an employer or occupier whose wrongful exposure might or might not have led to the disease would be liable in full for the consequences even if it was more likely than not that some other cause was to blame (let alone that it was more likely than not that he was to blame). But...that is the logical consequence of Fairchild and there is nothing we can do about it without reversing Fairchild. Even if we thought it right to do this, Parliament would soon reverse us, and it is easy to understand why.*

Questions about the scope and applicability of the ‘material contribution to risk’ test continue to plague tort law. The situations in which courts will depart from the ‘but-for’ test remain enigmatic. Is the test restricted to mesothelioma, or is applicable to other diseases? Both courts and legal commentators are divided on this issue. *Fairchild* did not explicitly limit itself to mesothelioma as we noted earlier,<sup>573</sup> but there have been subsequent pronouncements in the highest courts that have attempted to limit it in this way. In *Sienkiewicz*,<sup>574</sup> Lord Brown expressed unhappiness about this exceptional principle, but felt that mesothelioma was in a ‘category of its own’<sup>575</sup> and cautioned that: ‘(s)ave only for mesothelioma cases, claimants should henceforth expect little flexibility from the courts in their approach to causation.’<sup>576</sup> The scope to extend *Fairchild* to

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<sup>571</sup> *ibid* at [128] (Lord Sumption)

<sup>572</sup> *Sienkiewicz* (n 34) at [167]

<sup>573</sup> E.g. see above, text to (n 64)

<sup>574</sup> *Sienkiewicz* (n 34)

<sup>575</sup> *ibid* at [174] (Lord Bridge)

<sup>576</sup> *Sienkiewicz* (n 34) at [187] (Lord Bridge)

other conditions has been left open to courts. Steel points out: “But has the extension of *Fairchild* not subsequently been ruled out by the Supreme Court decisions in *Sienkiewicz v Greif...* and *Durham v BAI (Run Off) Ltd...*? No. Of the seven judges in *Sienkiewicz*, only Lord Brown suggested that *Fairchild* should be taken to apply only to mesothelioma cases (at [174]). This obiter suggestion can hardly defeat the point that *Fairchild* was founded on *McGhee*. In any event, Lord Phillips in *Sienkiewicz* explicitly envisaged an extension of the principle (at [105]) whose justification cannot be limited to mesothelioma cases.”<sup>577</sup>

However, the very recent case of *Heneghan v Manchester Dry Docks Ltd & Others*<sup>578</sup> has now extended the *Fairchild* test to a claim for lung cancer where there were multiple different potential causal factors, thus clarifying that the rule is not restricted to mesothelioma. As Allan notes, the Court of Appeal in *Heneghan* also opened *Fairchild* to even further extension, stating that there is ‘no reason why it should not apply to other diseases or injuries if similar factors which gave rise to *Fairchild* should apply. ***The most important of those factors was the uncertainty surrounding the physiological mechanisms which resulted in the disease.***’<sup>579</sup>

If uncertainty about the causal mechanisms of a disease is the criterion that will now be used to decide whether the exceptional ‘material contribution to risk’ test can be applied, per *Heneghan*, this leads to the result that a probabilistic assessment of causation should be of general application in disease claims. As

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<sup>577</sup> S Steel, ‘On When Fairchild Applies’ (2015) 131 LQR 363, 365

<sup>578</sup> *Heneghan* [2014] EWHC 4190 (QB); [2016] EWCA Civ 86, [2016] 1 WLR 2036 (CA)

<sup>579</sup> D Allan, ‘The Extension of Fairchild to Lung Cancer’ (2016) 2 JPIL 61, 61 (emphasis added)

this author has discussed extensively throughout the thesis, uncertainty about causal mechanisms is inherent in disease scenarios. The outcome of *Heneghan* thus supports the argument that this thesis has made, that all disease situations where evidentiary uncertainty is a significant problem (i.e. most complex disease scenarios) should be assessed by more probabilistic causal tests. Further, *Heneghan* not only involved lung cancer, but also involved two entirely different causal agents, which also goes against the original *Fairchild* principle that this reasoning only applies to single causal agent scenarios. This also opens the door for further extensions that are possibly less controversial as the Compensation Act does not apply to non-mesothelioma situations, as outlined in section 2.2 above. Thus proportionate liability could apply in such an approach.

The Court of Appeal in *Heneghan* also cited<sup>580</sup> the ‘Phurnacite’<sup>581</sup> litigation in order to support its decision to extend a more relaxed test to a scenario involving multiple potential causal factors. The ‘Phurnacite’ case concerned eight claimants who had all worked at a plant that produced a smokeless fuel with the trade name Phurnacite. They claimed damages for a range of conditions including respiratory illnesses (including pneumoconiosis), lung cancer and bladder cancer, alleging that these had been caused by exposure to two different carcinogen substances at the plant. The three claimants for lung cancer were exposed to carcinogens both from their occupational exposure at the Phurnacite Plant, as well as due to their smoking, as well as to environmental carcinogens. Lady Justice Swift acknowledged<sup>582</sup> that all these factors were likely to have

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<sup>580</sup> *Heneghan* (n 81) at [38]-[39]

<sup>581</sup> *Jones v Secretary of State for Energy and Climate Change* [2012] EWHC 2936 (QB) (‘Phurnacite’)

<sup>582</sup> *ibid* at [644]

played some part in the carcinogenic processes going on in the claimants' bodies, processes that were, by their nature, random. Regarding the medical evidence, LJ Swift stated that it was “not possible to say, in relation to any individual cancer, which factor or factors have caused or contributed to its development.”<sup>583</sup> Lady Justice Swift also took the view that the *Fairchild* test could not be applied outside of the mesothelioma context<sup>584</sup> (as we discussed earlier in this section, members of the judiciary seem to take widely varying views on this issue), so that only either the *Bonnington* or ‘doubles the risk’ (DOR) test could be applied to these claims. The *Bonnington* test, LJ Swift further held, only applied to divisible diseases (as we discussed in section 2.1.2 earlier, members of the judiciary also take widely varying views on this issue). Thus, while the *Bonnington* test was applied to the claims for respiratory illness (which fell into the divisible category), Lady Justice Swift concluded that the claims for lung cancer and bladder cancer could not qualify for applying the *Bonnington Castings* principle. Thus the obvious alternative<sup>585</sup> for these claims, concluded LJ Swift, was the ‘doubling of the risk’ test.<sup>586</sup> This test, a relatively recent alternative approach to disease causation, is explored below.

### ***2.3: ‘Doubling of the Risk’ (DOR) test***

Chapter 1 (at section 3.3) introduced the problems with the legal misconception that epidemiology infers causation solely from the risk ratio or relative risk (RR)

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<sup>583</sup> *ibid* at [646]

<sup>584</sup> *ibid* at [540]; [543]

<sup>585</sup> *ibid* [655]-[656]

<sup>586</sup> See also McIvor, ‘The ‘Doubles the Risk’ Test..’ (n 32) 226-228 for a further analysis of how this approach relates to confusion around the scope of *Bonnington*



value, and the idea that epidemiologists apply a rigid threshold value of  $RR > 2$  in order to conclude causation. This  $RR$  value has been interpreted, in legal contexts, into the ‘doubling of the risk’ (DOR) test, which has emerged in recent case law as a means of establishing causation in cases involving indivisible damage where there are competing sources of risk and the etiology of the disease is not sufficiently well understood to pinpoint the causes without recourse to probabilistic evidence.<sup>587</sup>

The law appears to have now concluded that an  $RR > 2$  equates to proof of causation on the balance of probabilities. This, in turn, appears to arise from the judicial translation of the ‘balance of probabilities’ test into a percentage figure (i.e. greater than 50% likelihood of a causal connection). Lady Justice Smith stated in *Novartis Grimsby v Cookson*<sup>588</sup> that ‘if occupational exposure more than doubles the risk due to smoking, it must as a matter of logic be probable that the disease was caused by the former.’<sup>589</sup> *Novartis*, it may be recalled from an earlier discussion (in Chapter 1 section 2) involved a claimant who developed bladder cancer following exposure to carcinogenic agents at work, as well as through his cigarette smoking. The dispute centred on the relative contributions of the two sources of exposure to the bladder cancer, and both medical and epidemiological expert evidence was adduced before the Court of Appeal. *Jones v Secretary of State for Energy and Climate Change*<sup>590</sup> (discussed in the previous section) chose to apply the DOR test to some of the claims (i.e. only to those that were

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<sup>587</sup> G Turton, *Evidential Uncertainty in Causation in Negligence* (Hart Publishing, 2016) 82

<sup>588</sup> *Novartis* [2007] EWCA Civ 1261

<sup>589</sup> *ibid* at [34]

<sup>590</sup> *Jones* (n 84)

indivisible) in a joint appeal involving various different diseases, while the Bonnington test was applied to divisible diseases.

This legal interpretation of the RR value is erroneous for several reasons. It is based on a fundamental misconception about epidemiology, and also errs in its simplistic translation of an RR value into proof on the balance of probabilities. As McIvor<sup>591</sup> points out, at a theoretical level, the DOR test belies a series of misconceptions about the science of epidemiology and its potential relevance to tort law. In addition, at a practical level, the seemingly scientific manner in which courts apply the test is unscientific and internally inconsistent.<sup>592</sup> *Further, this view also becomes another reason to undermine epidemiology, because courts assume that epidemiologists infer causation from no more than an RR value. Lord Phillips stated in Sienkiewicz v Greif that 'if statistical evidence indicates that the intervention of the wrongdoer more than doubled the risk that the victim would suffer the injury, then it follows that it is more likely than not that the wrongdoer caused the injury.'*<sup>593</sup> *After his analysis of RR values and the DOR test, Lord Phillips concluded that 'an 'RR>2 is a tenuous basis for concluding that the statistical cause of a disease was also the probable biological cause, or cause in fact.'*<sup>594</sup> McIvor notes that both of these mistaken judicial ideas about epidemiology: i.e. that it makes inferences from 'naked' statistics; and that epidemiologists make

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<sup>591</sup> McIvor, 'The 'Doubles the Risk' Test..' (n 32) 215

<sup>592</sup> *ibid*

<sup>593</sup> *Sienkiewicz* (n 34) at [72] (Lord Phillips)

<sup>594</sup> *ibid* (n 34) at [84] (Lord Phillips)

sweeping causal inferences simply based on an  $RR > 2$  'serves to cement the judicial view of epidemiology as a pseudo-science.'<sup>595</sup>

In fact, epidemiological inferences are far more complex than this judicial description suggests. Epidemiologists use a variety of other measures (including other statistical measures such as the attributable fraction, discussed earlier in Chapter 1 section 3.3; as well as non-statistical measures) to assess this issue, not just RR alone. Moreover, contrary to the impression given by the courts,  $RR > 2$  holds no intrinsic significance or value in epidemiology.<sup>596</sup> As we shall see later in Chapter 5 (section 1 and section 2.3), epidemiological inferences involve the use of many different techniques to assess the reliability of causal inferences. Further, Chapter 5 (section 3) will also discuss why it is erroneous to base legal outcomes directly on numerical or statistical figures, in general. In science, the totality of the evidence is all-important, and numbers in themselves can be meaningless and misleading, *unless* considered and interpreted in context. 'More probable than not' does not, epidemiologists Sander Greenland and James Robins stress, mean the same as an RR of more than 2.<sup>597</sup> Numerical data can be valuably used to *inform* decision-making, but should not be *determinative* of the legal outcome.

#### ***2.4: Problems in disease litigation highlighted by the exceptional tests***

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<sup>595</sup> McIvor, 'Debunking..' (n 62) 568

<sup>596</sup> McIvor, 'Debunking..' (n 62) 573

<sup>597</sup> S Greenland and JM Robins 'Epidemiology, Justice, and the Probability of Causation' (2000) 40 *Jurimetrics* 321, 323

Diverse and occasionally chaotic in application as the alternative tests discussed above are, it is important to begin this section by noting one important unifying quality of all of these exceptional approaches employed in disease litigation: they are all more suited to probabilistic causal reasoning than the deterministic but-for test. The other common feature all the exceptional approaches to factual causation share is that they have all been formulated to respond to urgent causal dilemmas and the apparent inadequacies of the traditional causation test in *disease* litigation. Further, all the arbitrary (and unjustifiable on principle) distinctions that were formulated to restrict their use are starting to break down, as the exceptional tests are being applied to an increasing number of diseases. Thus, they clearly highlight one central argument of this thesis: the pressing imperative for a legal approach to disease causation, at least, that can take account of probabilistic evidence.

Oddly, despite the judicial and academic criticism of the ‘material contribution’ tests, courts seem to be resorting to variations of these lines of reasoning in an ever-broadening range of disease-related claims. Despite many attempts to draw it back, the controversial *Fairchild* exception has not remained restricted to mesothelioma cases. It has been applied to, for example, Vibration White Finger condition<sup>598</sup> and, in *Heneghan v Manchester Dry Docks Ltd*,<sup>599</sup> to adenocarcinoma of the lung caused by asbestos. However, the applications of the test outside of mesothelioma situations are infrequent, and often require judicial creativity,

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<sup>598</sup> *Brown v Corus (UK) Ltd* [2004] EWCA Civ. 374; [2004] P.I.Q.R. P30

<sup>599</sup> *Heneghan* (n 81)

such as in *Heneghan*, where the court had to resort to somewhat perplexing lines of reasoning to justify its use. The preceding discussion of the exceptional tests appears to highlight a few issues pertinent to disease causation that will be outlined below. Similarly, as the discussion of the *Bonnington* test in section 2.1 shows, the ‘material contribution to injury’ test, originally circumscribed to situations where similar causal factors acting concurrently make a cumulative contribution to the harm, has now been extended to situations where the multiple *different* factors may have independently been sufficient to cause the harm (as in *John*<sup>600</sup>) or where they occurred concurrently (as in *Williams*<sup>601</sup>). There appears to be no current agreement amongst the judiciary as to their scope and remit. This is, this thesis contends, because restricting criteria for the use of probabilistic tests in disease litigation is fundamentally unjust due to the nature of biomedical causation: the very recent extensions of the tests are simply a sign that this injustice is becoming more apparent. The following subsections outline a few key aspects about disease claims that the preceding discussion of the exceptional approaches highlights.

#### ***2.4.1 The inadequacy of deterministic approaches***

All of these exceptional tests for factual causation in this area of the law reflect that it is futile to ask scientists for evidence that a certain factor was causally *necessary* in a claimant’s disease. This question will repeatedly present the danger of potential injustice, as medical science can rarely answer questions

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<sup>600</sup> *John* (n 51)

<sup>601</sup> *Williams* (n 46)

about what would have happened if the tortious factor had not been present. Even if courts only seek an answer to this question on the balance of probabilities, the fundamental *conceptual* question that courts ask science to answer is still about the *necessity* of the causal factor. As case after case shows, this question is unsuited to assess a disease process which can happen in many different ways, and is fundamentally unobservable as it happens.

The case of *Bailey v Ministry of Defence*<sup>602</sup> serves as a good example of the impossibility of satisfying but-for questions in the assessment of disease causation. Weakness resulting from medical negligence does not look any different from weakness caused by, as in this case, pancreatitis. Nor is it possible for an expert (no matter how skilled) to measure the quantity of 'weakness' that pancreatitis and subsequent surgery results in; or to say how much weakness would have been needed to have caused the claimant to asphyxiate. Such a number, even if there were such a thing as units of 'weakness', would have no validity due to the incredible biological variability of different human bodies, in terms of resilience. To a medical expert, the existence of a potentially causal factor and the ultimate injury are events they can only infer the *likelihood* of causation from. Until human bodies come equipped with internal CCTV cameras that videotape toxins entering a claimant's bloodstream and beginning microscopic processes of cellular mutation, it may remain impossible to satisfy current legal questions about factual causation for most diseases.

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<sup>602</sup> *Bailey* (n 37)

The whole of the *Fairchild* approach seems to be founded on the idea that mesothelioma is somehow an ‘exceptional’ disease in terms of the uncertainties about its onset, etiologic mechanisms, and the many unobservable and synergistic mechanisms through which it occurs. The recent expansions of *Fairchild* to diseases beyond mesothelioma, and to causal scenarios beyond those involving similar causal agents (such as in *Heneghan*), indicates that courts are realising that these arbitrary distinctions between factual scenarios in disease claims run counter to the principles of justice. *Most* diseases, as we saw in Chapter 3, have evidentiary gaps that can make but-for assessments of causation unworkable: mesothelioma is not extraordinary in its long latency period or somewhat mysterious onset and progression. In fact, if anything, it would be possible to argue that mesothelioma may actually be a *simpler* disease for courts to deal with, because it is a ‘signature’ disease that has one causal factor (inhalation of asbestos fibres) which is almost always linked to the outcome.<sup>603</sup> Further, there are no controversies at least around ‘general’ causation aspects.<sup>604</sup> On the other hand, conditions such as cardiovascular disease and most other forms of cancer have no single necessary cause, but can be caused by many different combinations of various risk factors such as stress, genetics, poor diet, unhealthy lifestyle, smoking, etc.

A medical expert who is forced to testify about whether any one of those was **necessary** for a specific claimant’s heart attack or stroke will be forced to rely on mostly subjective opinion and educated guesswork. There would be high

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<sup>603</sup> J Sanders, ‘Risky Business: Causation in Asbestos Cases (and Beyond?)’ in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 14

<sup>604</sup> *ibid*

variation between the opinions of different experts on this issue: a problem that can be, and often is, exploited in an adversarial judicial system, where each side brings its own experts who are influenced by conscious or unconscious conflicts of interest to give very different opinions. It could be argued that this played a part in the outcome of *McTear*, where even extremely high 'general' causal links- indicating a causal likelihood of around 90%- between smoking and lung cancer was disputed by the defendant's expert, and the court found this persuasive enough to rule that even general causation was not established despite the overwhelming epidemiological evidence.<sup>605</sup> The law urgently needs to revise this scientifically naïve view of disease, and to develop principles that can deal effectively with probabilistic causation.

#### ***2.4.2: Overdetermination as the norm in disease litigation***

Despite all the dilemmas the but-for test has raised in disease litigation, the judicial attachment to the traditional approach to factual causation has led to courts striving hard to formulate a variety of strict criteria to limit the use of the exceptional tests, pertaining to qualities of the disease, causal agents or timing of exposure. It appears, however, that this restrictive approach may have been easier to formulate than to implement. Despite much judicial effort to limit their use, courts are increasingly being presented with much sound scientific evidence to argue that they should be expanded to a growing range of diseases (since, as discussed earlier, inherent causal uncertainties characterise most complex diseases). As the previous subsections above demonstrate, all of these arbitrary

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<sup>605</sup> See Chapter 1 section 2.1.1



distinctions between disease and timing of exposure are already incrementally starting to break down in any case, especially in a number of very recent cases. Despite judicial reluctance, the *Bonnington* and *Fairchild* principles have recently been extended to more diseases and variant causal scenarios, even when these do not meet the original specified criteria.

Most diseases are caused in many complex and stochastic ways, and can often arise from various different combinations of multiple different factors. Thus, this thesis contends that over-determined causation may be the ***norm*** rather than the ***exception*** in disease litigation. It is no accident that the most difficult dilemmas around causation arise in the context of disease claims: this is proof of the special complexities that beset this area. Given this reality, it is difficult to justify why this exceptional approach should be restricted to select diseases when similar uncertainties apply equally, if not more, to many other diseases.

This thesis agrees in principle with the outcome of the recent cases, which widens the more flexible assessments of causation in disease involving toxic exposures, since it is a central contention of this thesis that probabilistic causal assessments must be the norm rather than the exception in disease litigation. However, this author disagrees with the form these extensions have taken. The extensions of the exceptional tests have so far been reluctant and haphazard, leaving a raft of questions hanging about when and where exceptional tests will apply. A fairer approach to such claims must not (and further, need not) occur at the cost of the coherence and clarity of the law. There is no need, this author argues, for recourse to effortful judicial creativity and 'policy' justifications in

order to assess disease causation differently: in this area, at least, it is possible to identify a more principled justification for such a departure from tradition (as will be discussed in section 2.4.3 below). It would do the law far more credit, this thesis asserts, to identify an alternative approach to causal analysis in disease claims generally, that is able to take account of probabilistic causation.

A decade ago, Plowden and Volpe queried whether the exceptional rules applying to mesothelioma could be applied to hospital-acquired MRSA that might have been contracted due to hospital negligence.<sup>606</sup> The condition, they pointed out, can potentially satisfy all of the *Fairchild* criteria:<sup>607</sup> the claimant may well have been close to sources of MRSA as a result of both innocent and negligent means; and it is scientifically impossible to prove, merely by virtue of the fact of the infection, the moment or means of contamination. So could the more relaxed test of causation sanctioned by the House of Lords in the mesothelioma cases (such as *Fairchild* and *Barker*), queried the authors, allow the MRSA claimant to overcome the usual evidential impossibility of proving the means of contamination? A decade later, it appears that courts are starting to find such arguments persuasive, as indicated by cases such as *Heneghan*<sup>608</sup> and *Williams*.<sup>609</sup>

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<sup>606</sup> S Plowden & H Volpe, 'Fairchild and Barker in MRSA cases: Do Fairchild and Barker Provide an Argument for a Relaxation of Causation Principles in Claims for Hospital Acquired MRSA?' (2006) *JPI Law* 259

<sup>607</sup> *ibid* 260

<sup>608</sup> *Heneghan* (n 81)

<sup>609</sup> *Williams* (n 46)

The exceptional *Fairchild* principle enabled the claim for lung cancer in *Heneghan* to succeed, although the court ruled that damages should be apportioned according to each defendant's contribution to the deceased's risk of contracting lung cancer (despite the fact that lung cancer was held to be an indivisible disease). Lord Dyson drew support for the extension of the *Fairchild* principle to this scenario from the earlier Supreme Court decision in *International Energy Group Ltd v Zurich Insurance Plc UK*:<sup>610</sup>

*Thus, Lords Neuberger and Reed said at para 191 that the Fairchild exception is "applicable to any disease which has the unusual features of mesothelioma." The possibility of its application in cases concerning other injuries or diseases was also expressly contemplated by Lord Hodge (para 109) and Lord Sumption (para 127).<sup>611</sup>*

Steel<sup>612</sup> is critical of the extension of the *Fairchild* principle to *Heneghan*, citing two reasons for doubting it: "The first is the "single agent requirement" for the application of *Fairchild*: in order for the rule to apply, all of the potential causative agents for the disease must operate in substantially similar ways. Mr Heneghan was a smoker and this smoking *might* have contributed to his lung cancer. Smoking operates in a different way to asbestos dust in causing lung cancer... (Justice Jay's) interpretation of the single agent rule is, with respect, unappealing. The view that the rule does not apply (or is satisfied when the claimant has been exposed to the same kind of agents by multiple defendants is overinclusive...'<sup>613</sup> The second reason for doubting the decision, opines Steel, is that no claim was brought against the claimant's earlier employer, who had

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<sup>610</sup> *Zurich* (n 71)

<sup>611</sup> *Heneghan* (n 81) at [49]

<sup>612</sup> Steel, 'On When Fairchild Applies' (n 80) 366

<sup>613</sup> Steel (ibid)

contributed 56% of the total risk (greater than the aggregate risk contributed by the defendants).<sup>614</sup>

However, the case of *Heneghan* again serves to strengthen the argument being advanced in this thesis. Any attempt to restrict, in principle, the more flexible and probabilistic tests causation to a few select diseases will inevitably run into problems, because most complex diseases share the quality of etiological uncertainties. Such distinctions as single/multiple agents, divisible/indivisible disease, or alternative/cumulative causes are illusory as far as decisions about causal contributions or ultimate liability are concerned.

### ***2.4.3: Dangers of excessive judicial 'pragmatism': identifying a more principled approach***

The existent problems with disease claims are increasingly leading to courts having to make the effort to find “ways around” existing legal principles, to avoid injustice to claimants for other diseases that do not fall within current exceptions. Despite its ambiguity and vagueness, the ‘material contribution’ test is at least better able to handle its encounters with probabilistic evidence, and thus it is more suitable than the ‘but for’ test to toxic tort litigation. However, the problem with this test lies in the hazy judicial explanation of its rationale. Courts explain the ‘material contribution’ tests as being a special dispensation on ‘normative’ grounds, and this is cited as a reason why the circumstances in which this test will and will not apply have been left open. Stapleton expresses

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<sup>614</sup> Steel (n 80) 366-367

approval of this degree of 'normativity' in factual causation, so that claimants can 'leap that gap'.<sup>615</sup>

This thesis, however, submits that an exceptional causation approach in disease claims does not require hazy 'normative' grounds to justify it. However, although this author strongly advocates a more probabilistic approach to factual causation in toxic tort litigation, at the same time this must be distinguished from subjective decision-making that has no evidence at its base. There is no need for contorted legal reasoning in order to accommodate to the realities of disease and medicine. The but-for test makes a number of presumptions about causality that are detached from the real world, and from all available scientific evidence, when it comes to disease causation. For the law to continue with such unclear and uncertain assessment of causation carries a real risk that the factual causation element of the negligence analysis will eventually become entirely meaningless.

*Further, courts often resort to convoluted lines of reasoning in order to maintain the illusion that principled lines of reasoning are being followed, and to justify these extensions of the exceptional tests.* Lord Nicholls, whilst agreeing with the majority ruling in *Fairchild* recognised the problems in the reasoning:

*I have no hesitation in agreeing with all your Lordships that these appeals should be allowed. Any other outcome would be deeply offensive to instinctive notions of what justice requires and fairness demands. The real difficulty lies in elucidating in sufficiently specific terms the principle being applied in reaching this conclusion. To be acceptable the law must be coherent. It must be principled. The basis on which one case, or one type of*

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<sup>615</sup> J Stapleton, 'Cause in Fact and Scope of Liability for Consequences' (2003) 119 Law Quarterly Review 388, 388

*case, is distinguished from another should be transparent and capable of identification. When a decision departs from principles normally applied, the basis for doing so must be rational and justifiable if the decision is to avoid the reproach that hard cases make bad law.*<sup>616</sup>

McIvor<sup>617</sup> notes that much of the incoherence arises because courts have not always been consistent in their application of these principles in difficult probabilistic causation cases: they have failed to clearly articulate, or carefully circumscribe the exceptional policy considerations to justify a departure from orthodox factual causation principles. This thesis, however, contends that a departure from the but-for test in disease claims, at least, does not require policy justification. In this area of the law, the justification for a probabilistic approach to causation can be identified on more principled grounds: that is, the vast body of sophisticated modern biomedical research. It is imperative for the law to recognise that the growing mismatch between legal and medical models of causation. It would do the law far more credit to overhaul the legal principles that govern disease litigation, and acknowledge their inadequacy. Making impossibly fine distinctions between diseases cannot justify the approach that only a few select diseases will merit compensation, while other claimants who also suffer horrific harms (albeit with a different label) due to negligence will go entirely uncompensated. Thus this thesis contends that more probabilistic tests for causation should be of **general** application in disease claims.

Not everyone agrees, however, with the more radical shift from orthodoxy suggested in this thesis. Sanders notes, for example, that “calls for altering the

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<sup>616</sup> *Fairchild* (n 8) at [36]

<sup>617</sup> McIvor, ‘Debunking..’ (n 62) 556

traditional 'but for' test of causation in the toxic tort era wisely have been resisted by courts:" the risk rule, he cautions, "is a risky concept."<sup>618</sup> However, this author contends such changes are in any case already starting to occur in any case, and altered tests for factual causation are already being expanded further and further to many different diseases (especially in very recent years in UK courts). This thesis simply makes a plea for these changes to occur on a more principled basis. The boundaries of the material contribution tests appear, unfortunately, to be becoming even more uncertain and hazy, rather than less so, as time goes on. The *Bonnington* principle was formulated 60 years ago, and it should be a matter of concern that that this state of affairs still prevails. A more radical overhaul of approaches in this area is, it is contended, the need of the hour.

To the extent that the judicial advocacy of 'common-sense' approaches to causation (discussed in Chapter 3, section 1) is a call for pragmatic approaches to causation that are flexible enough to take account of the available evidence, this thesis supports the idea of common-sense approach to causation. The law does not need to be drawn into deep debates about the philosophy of causation in order to evaluate factual causal links in a coherent way. However, to say that questions about causation can be adequately answered by instinct or common-sense *alone* is deeply flawed. 'Common sense', in this judicial definition, becomes so subjective that liability would almost become a matter of the judge's personal instinct.

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<sup>618</sup> Sanders (n 106) 39

There may be some merit in the legal idea that ‘the causal requirements of one rule may be different from those of another’, as Lord Hoffmann concludes in his analysis of the causation caselaw in *McGhee*,<sup>619</sup> *Bonnington*,<sup>620</sup> *Fairchild*<sup>621</sup> and *Barker*.<sup>622</sup> This is also, as Chapter 3 (section 2) earlier illustrated, how scientists are increasingly coming to understand causation: that causation is not a monolithic concept, and that ‘what causes are...can vary from one kind of system of causal relationships to another’.<sup>623</sup> It appears, then, that then law and science may both independently be converging towards this idea. The argument contained in this thesis for a different approach to causation in disease litigation context is thus in line with Lord Hoffmann’s analysis.

However, it is a fundamental principle of justice that the law must be transparent, and legal terms and principles must possess at least a reasonable degree of certainty. As Stapleton notes, navigation of causation would be made more transparent by the clarification of legal terminology:<sup>624</sup> To advocate a balanced, common-sense approach to the assessment of probabilistic evidence about causation is not to assert that examination of the causation question can be answered by subjective preferences and judges’ personal ‘commonsense’ preferences alone. If the law uses a different approach to causation in complex D claims, it is important to justify why this is so, and to have a transparent approach. The assessment of causation indeed requires common sense, but also

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<sup>619</sup> *McGhee* (n 60)

<sup>620</sup> *Bonnington* (n 7)

<sup>621</sup> *Fairchild* (n 8)

<sup>622</sup> *Barker* (n 66)

<sup>623</sup> N Cartwright, *Hunting Causes and Using them: Approaches in Philosophy and Economics* (Cambridge University Press 2007) cited from SS Coughlin, *Causal Inference and Scientific Paradigms in Epidemiology* (Bentham 2010), 15-16- see below n\_

<sup>624</sup> J Stapleton, ‘Cause in Fact and Scope of Liability for Consequences’ (2003) 119 *Law Quarterly Review* 388, 388



requires more than that: it requires an objective scrutiny of the available evidence about factual connections between tort and harm

A more realistic test for disease causation of general applicability would be more principled not only because it would accord with the principles of justice (because it would not draw arbitrarily discriminate between different diseases), but also because it would have as its justificatory basis the best available empirical evidence. The 'risk rule' (i.e. the *Fairchild* rule) can be further refined to have a little more clarity for better congruence with biomedical evidence and terminology: congruence that is vital in disease litigation. Section 3 below will suggest a new framework for such an alternative approach.

### ***SECTION 3: A suggested alternative: assessment of likely contribution to disease***

In view of the inadequacies of the but-for test, academics have proposed a number alternative tests for general application to the factual causation analysis (that is, not specific to disease litigation scenarios). One test that has been the focus of much debate (particularly in the US) is the NESS test.<sup>625</sup> The NESS test, formulated by Wright, defines a cause in fact as 'a necessary element in a sufficient set.'<sup>626</sup> This test is generally viewed as being better able to handle some situations indeterminate causation, although there are a number of conceptual problems with the NESS test. There have been, for example,

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<sup>625</sup>R Wright, 'Causation, Responsibility, Risk, Probability, Naked Statistics and Proof: Pruning the Bramble Bush by Clarifying the Concepts' (1988) 73 Iowa Law Review 1001

<sup>626</sup>R Wright, 'Causation in Tort Law' (1985) 73 California Law Review 1735, 1788-1803

philosophical questions about the concept of sufficiency.<sup>627</sup> However, the most crucial problem for the use of the NESS test in disease litigation is that it is also based in a deterministic view of causation, and cannot deal with probabilistic causation. It does not effectively address the practical problems that arise in the assessment of disease causation: the impossibility of knowing whether a factor was necessary in the onset of a disease. As Berge notes, we do not yet fully comprehend the biological mechanisms that produce birth defects and illnesses such as cancers and auto-immune diseases for which plaintiffs seek compensation, and exposure to the defendant's product is not usually a necessary cause of a particular disease.<sup>628</sup> The legal quest for a but-for cause, which 'is more causal than other causes'<sup>629</sup> in the assessment of disease causation is therefore a doomed quest for the foreseeable future, for all but a small minority of diseases.

This area of the law, this thesis has argued throughout, requires a test that has sufficient flexibility to take account of probabilistic causation, rather than one that views causation in deterministic ways. However, as Fumerton and Kress note about the NESS test: 'Given the way in which we define lawful sufficiency, nothing is ever lawfully sufficient for anything in a radically indeterministic universe. For our purposes, we shall say that a universe is deterministic when each state or "time slice" of the universe is lawfully sufficient for the next and all subsequent states of the universe; an indeterministic universe is one that is not

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<sup>627</sup> R Fumerton and K Kress, 'Causation and the Law': Pre-emption, Lawful Sufficiency, and Causal Sufficiency' (2001) 64(4) *Law and Contemporary Problems* 83, 96-97

<sup>628</sup> MA Berger, 'Eliminating General Causation: Notes Towards a New Theory of Justice and Toxic Torts' (1997) 97 *Columbia Law Review* 2117, 2121

<sup>629</sup> WL Prosser, 'Proximate Cause in California' (1950) 38 *California Law Review* 369, 381

deterministic. A radically indeterministic universe is one in which no state is ever lawfully sufficient for any subsequent state. The NESS test presupposes determinism...Yet it is far from clear that our concept of causal connection precludes the possibility of causes in an indeterministic world.’<sup>630</sup>

The possibility, as Fumerton and Kress note, that there can be causation in an indeterministic universe suggests that we should hesitate before we regard X being a NESS of Y as an analytically necessary condition for X being a causally relevant factor in the occurrence of Y. Further, they point out, neither is X being a NESS of Y an analytically sufficient condition for X being a causally relevant factor in the occurrence of Y. Thus, they conclude, if the law is waiting for philosophers to offer something better than a pre-philosophical grasp of what is involved in one thing causing another, ‘the law had better be very patient indeed.’<sup>631</sup>

This thesis suggests that a better approach to causation in toxic tort litigation is to determine whether the best available evidence indicates that the tortious cause made, on the balance of probabilities, a **significant contribution to the disease**. However, because this approach is probabilistic, liability must only be in proportion to the likely contribution to the disease. Such an approach would be realistic and flexible enough to satisfy the demand for a ‘common-sense’ approach, as well as be underpinned by objective evidence.

### ***3.1 The probabilistic test for disease causation:***

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<sup>630</sup> Fumerton and Kress (n 130) 97

<sup>631</sup> *ibid* (n 130) 102-105

This thesis proposes that, in congruence with modern biomedical approaches to causation, factual causation in respect of disease claims should be assessed on the basis of whether the allegedly tortious factor *significantly contributed to the onset or progression of the claimant's disease*. However, as this approach recognises that individual causal factors often make only partial contributions to disease (and further, because we will rarely know if the outcome would have been different if a particular factor had not been present i.e. whether the factor was *necessary* for the outcome), it also proposes that this test can only work in conjunction with proportionate liability.

The fact that this test applies equally regardless of whether the factor made a contribution to the *onset* of the disease, or only to its further *progression*, is also a further advantage. Contribution in either scenario, this thesis contends, is still a contribution to the injury. At the moment, the material contribution tests only view 'contribution to injury' as referring to a contribution to the progression of the disease, which is what causes legal distinctions such as divisible and indivisible diseases to take on undue importance. Thus, the *Fairchild* approach was only needed in the first place because *Bonnington* was only seen to apply if the progression of the disease was dose-related. Under the single-fibre theory of mesothelioma prevalent at the time, it was believed that mesothelioma, once triggered, could not be worsened by further exposure. Thus the *Fairchild* scenario needed a different test. But what if the initial onset, or triggering of a disease, requires a certain threshold amount of exposure? Surely each contributory factor contributing to the onset of a disease should count as having

contributed to the injury, but such a situation is not covered under the material contribution to injury principle, and has to satisfy the much harder *Fairchild* criteria. This is difficult to justify (and, in any case, the single-fibre theory of mesothelioma is now in doubt, as discussed earlier in Chapter 3). Regardless, under the new test, the same test would apply equally to both the *Bonnington* and *Fairchild* situations. This test thus abolishes the unfair distinctions between diseases in terms of legal outcomes, and avoids courts having to deal with all the controversies and confusion that result from these.

This reframing of the test for factual causation in disease litigation does not need to lead to widening of the scope of liability: it must be remembered that this is a test for factual causation only. And, as this thesis has argued earlier, factual causation is not, and should not be, the be-all and end-all of liability. To say that something has been a contributory cause of an injury is not necessarily to say that there should be legal liability and responsibility attached to it. Courts only need to make causal decisions in regard of *faulty* behaviors, and there are several other strands of the negligence enquiry that must also be assessed before liability is imposed in negligence, such as duty of care, failure to take reasonable care, remoteness and the presence of intervening events. As Professor Honoré emphasises, that the law does not hold individuals legally liable for all actions for which they are in some sense responsible. Liability only attaches where the actor is on notice that the conduct is wrongful, the sanction is proportionate to the gravity of the misconduct, and causation is proved by sufficiently credible

evidence to justify the sanction.<sup>632</sup>

Further, because this test imposes liability for probable contribution, this thesis acknowledges that it is important to build in further safeguards against claims for minor, incidental, or non-specific factors that could arguably have played some small contributory role. There are, for instance, a number of non-specific factors (such as stress, relationship difficulties, previous infections etc) that are hypothesised to slightly increase general predisposition to disease and illness. The test proposed in this thesis should however only be applied in regard of factors that are empirically established to play a *specific*, and *significant*, contributory role in the development of the specific disease that is being claimed for. The definition of significant here requires something more than the current legal definition of significant as anything more than *de minimis*. Further, this test must only be applied on a proportionate basis.

### ***3.2 Proportionate liability:***

As explained above in section 3.1, apportionment of liability is a crucial element of liability under this proposed new approach, in order to avoid gross unfairness to defendants. This test is fairer to claimants because it acknowledges that factors can sometimes work in conjunction with others in order to either trigger or worsen a disease, and thus does not ask for proof of necessity. However, in this acknowledgment, it also recognises making a defendant liable for the whole

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<sup>632</sup> Anthony Honoré, 'Principles and Values Underlying the Concept of Causation in Law' in D Mendelson and I Freckleton (eds.), *Causation in Law and Medicine* (Ashgate 2002) 3-4

harm, when all evidence suggests that they were only partially responsible, would be unfair to defendants, and would also open the doors for excessive liability. It is crucial to avoid such an outcome that would just substitute one patent unfairness for another. The Court of Appeal in *Fairchild*, for example, pointed out that it considered the claimants' argument to be not only illogical but also:

*...capable of unjust results. It may impose liability for the whole of an insidious disease on an employer with whom the claimant was employed for quite a short time in a long working life, when the claimant is wholly unable to prove on the balance of probabilities that that period of employment had any causative relationship with the inception of the disease...If we were to accede to the claimants' arguments, we would be distorting the law to accommodate the exigencies of a very hard case... In a quite different context Lord Steyn has recently said... that our tort system sometimes results in imperfect justice, but that is the best the common law can do.*<sup>633</sup>

Proportionate liability is already applied in regard of diseases deemed 'divisible' in UK law, as discussed briefly in section 2.1 above. This principle was applied in cases such as *Thompson v Smiths Ship Repairers (North Shields) Ltd*<sup>634</sup> (a claim concerning exposure to successive noise) and *Holtby v Brigham & Cowan (Hull) Ltd*.<sup>635</sup> Mr Holtby was exposed to asbestos dust while working for a number of employers, half of which time was spent with the defendants. He developed asbestosis, and sued the employers who were found to have been negligent. The judge found them liable but made a reduction in the total award of damages to reflect the fact that they had only been the employer for part of the relevant time.

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<sup>633</sup> *Fairchild* [2002] 1 WLR 1052 at [103] (Court of Appeal)

<sup>634</sup> *Thompson* (n 30)

<sup>635</sup> *Holtby* (n 25)

The court reduced the damages by 25% rather than the exact proportion of the period of employment (which would have been 50% as he had spent half his time with the defendant).

Further, courts have also attempted to apply proportionate recovery to indivisible diseases. Section 2.2.2 above discussed The House of Lords' application of this principle in the mesothelioma claim in *Barker*<sup>636</sup> (until Parliament intervened to bar the application of proportionate liability in mesothelioma claims). However, the Court of Appeal in *Heneghan*<sup>637</sup> recently applied the proportionate recovery principle to a claim for lung cancer (an 'indivisible' disease), with Justice Jay holding that in non-mesothelioma claims, *Barker* remained good law.

Given that complete certainty is rare, and most disputed situations involve varying degrees of uncertainty about what actually happened, it is crucial for courts to have a clearer understanding about the value and limitations of evidence. Stein, in his treatise,<sup>638</sup> suggests that the key function of evidence law is not so much to facilitate the discovery of truth, as to apportion the risk of error under conditions of uncertainty. He suggests that principles such as cost-efficiency, equal-best and equality are possible drivers of the apportionment of risk in legal fact-finding. Stein's principles may be a useful tool with to approach situations where there is no other objective data available to guide the apportionment process in legal contexts. However, this thesis suggests that the

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<sup>636</sup> *Barker* (n 66)

<sup>637</sup> *Heneghan* (n 81) (Court of Appeal)

<sup>638</sup> For further explorations of these, see A Stein, *Foundations of Evidence Law* (Oxford University Press 2005)



primary purpose of evidence is to minimise injustice by applying the scientific ‘inference to the best evidence’ approach that has been advocated throughout the thesis to questions of apportionment. Thus, liability in the proposed new approach to disease claims should be proportionate to the likely contribution it made to either the onset or progression of the disease, *as indicated by medical, epidemiological or any other available evidence*. This, it must be noted, does not always equate to the amount of time a person has been exposed to a substance, but on many other factors such as its causal potency in the context of that disease. This is why scientific evidence is crucial in this area of the law.

This point is also frequently raised in debates about disease litigation in the US. The American authors Lasagna and Shulman note that: ‘it may be that the all-or-nothing result of the balance of probabilities standard is inappropriate for cases dependent on scientific evidence. This has led many US authors to argue for a more discrete evidentiary standard, under which courts assign liability in proportion to the probability of causation of the substance in question (whether above or below 50 percent).<sup>639</sup>

Following the House of Lords’ judgment in *Barker* on proportionate recovery, Parliament rapidly intervened to reverse that part of the judgment and imposed joint and several liability. Allan contends that it is difficult to see what basis there is for distinguishing between the mesothelioma victim and the lung cancer victim. The lung cancer victim already faces the hurdle of proving on a

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<sup>639</sup> L Lasagna and SR Shulman, ‘Bendectin and the Language of Causation in KR Foster, DE Bernstein and PW Huber (eds), *Phantom Risk: Scientific Inference and the Law* (MIT Press 1993), 116

conventional "but for" basis that asbestos or some other occupational exposure was the cause. The case for an extension of s.3 of the Compensation Act to lung cancer victims appears compelling.<sup>640</sup>

This author agrees that there is little basis for discriminating between diseases, as this would make liability a matter of luck. However, the solution suggested by Allan it would not only open the doors for excessive liability, but run counter to the evidence, because given the interactive and only partially understood process by which multiple factors usually bring about an illness, it is relatively rare for a single factor to entirely bring about a complex disease. Liability must thus be proportionate to the likely causal contribution of each factor, as estimated by epidemiological and any other scientific evidence. This author thus argues that the solution to the problem raised by Allan is that proportionate liability should be applied to all diseases, and section 3 of the Compensation Act 2006 should be overturned.

The evidentiary model of epidemiology is a correct fit for this suggested approach, because it is fundamentally probabilistic. Epidemiology holds that a cause is simply one that increases the probability that a disease or harm will occur: a conceptualisation that does not require engagement with futile and 'unanswerable' debates about whether a causal factor was necessary, nor sufficient, for a disease to occur.<sup>641</sup> The Court of Appeal in *Heneghan* also acknowledged<sup>642</sup> the submission of the defendant's expert, Dr Rudd, who

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<sup>640</sup> Allan (n 82) 65

<sup>641</sup> SS Coughlin, *Causal Inference and Scientific Paradigms in Epidemiology* (Bentham 2010) 9

<sup>642</sup> *Heneghan* (Court of Appeal) (n 81) at [31]

emphasised that the "current understanding of biological mechanisms does not form a basis for the practical attribution and apportionment of causation of particular cancers. This still rests upon epidemiological evidence and theories about biological mechanisms should not be used to undermine conclusions based upon epidemiological evidence". Notes Karhausen<sup>643</sup> about the epidemiological approach: 'we merely observe tendencies toward sufficiency or tendencies toward necessity: cohort studies evaluate the first tendencies, and case-control studies the latter.' In applied sciences, such as medicine and epidemiology, causes are intrinsically connected with goals and effective strategies- causes do not explain event E but event E rather than event F.<sup>644</sup> This is congruent with the argument advanced throughout the thesis, that the role of the court is only to decide whether it finds the claimant's or the defendant's evidence more persuasive, not to find out what actually happened.

Thus, this thesis advocates for a better legal test for disease causation that can take account of the complexities of disease, and can accommodate probabilistic evidence. Further, it strongly argues for apportionment of liability, and suggests that epidemiological causal models have a valuable contribution to offer, both to establishing factual causation and then to calculating the likely contribution.

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<sup>643</sup> LR Karhausen, 'Causation: The Elusive Grail of Epidemiology' (2000) 3 *Medicine, Health Care and Philosophy* 59, 59

<sup>644</sup> *ibid*

## CHAPTER 5

### BETTER USE OF SCIENCE: THE VALUE OF EPIDEMIOLOGY IN ASSESSING SPECIFIC CAUSATION

As a relatively young science, epidemiology can sometimes be perceived as inferior to some of the more established sciences such as clinical medicine, or to hard sciences such as physics, which seem to offer theorems and laws with mathematical proof. Unfortunately for legal expectations (as this thesis has attempted to illustrate through the preceding discussions) such proof is simply impossible for disease causation, at least in our current state of human knowledge. Further, the notion that any science can offer complete conclusive proof regarding a question is fallacious: something that is now becoming increasingly evident even in the world of physics, the discipline which was once held as the benchmark for the ideal of science as conclusive and deterministic (as discussed earlier in Chapter 2). This chapter will now attempt to re-assess the legal and judicial misconceptions about epidemiology in light of the above realities, and will conclude that the devaluation of epidemiological inferences of causation is both unfair and impractical. It will suggest a way forward for the better legal use of scientific evidence generally, and epidemiological evidence in particular. It is important to note at the outset that this thesis does not hold that all problems in the law-science relationship are due to inadequacies on part of the law. Unrealistic legal expectations from science are at least partially a consequence of the fact that some scientific expert witnesses give courts a false impression of how 'scientific' their assertions are, and often do not provide

courts with an accurate account of the extent of subjectivity, or potential for error, in their testimony (an issue that will be further explored in section 4 of this chapter).

This author does not dispute the judicial observation that epidemiology cannot offer *conclusive* proof of specific causation. Epidemiological inferences about causality are indeed subject to limitations and caveats. However, such evidence is based in sound empirical foundations and is, in many situations, the best *available* evidence about disease causation that science may be able to offer, at least at present. Thus, it is submitted, courts have a responsibility, in the interests of justice, to give serious consideration to this form of evidence where it exists, *particularly* where there is no other evidence to help assess specific causation (as will very often be the case given the very complex and fundamentally unobservable mechanisms of disease causation). However, as this evidence does have potential for error, it must be evaluated in a nuanced and judicious way. Such data should guide, rather than determine, the legal outcome.

Over the centuries, notes Klinkner,<sup>645</sup> philosophers of science have struggled to answer questions about what makes scientific findings truthful and to formulate standards that would help identify good scientific explanations. They have considered whether scientific pronouncements ought to be causal, unified, nomological, statistical, deductive, inductive or any combination of these. Given that philosophers of science wrestle with such questions, how are judges, lacking

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<sup>645</sup> M Klinkner, 'Forensic Science Expertise for International Criminal Proceedings: an Old Problem, a New Context and a Pragmatic Resolution (2009) 13 International Journal of Evidence and Proof 102, 102

in scientific expertise and with little time for deliberation on metaphysical and methodological issues, to determine what makes scientific results valid and reliable, truthful and credible?<sup>646</sup>

The case this thesis has made for judges to display better understanding about science is not a demand that they should transform into ‘super-scientists with access to a level of knowledge superior to those who have given the evidence’<sup>647</sup> as Mackay J put it, when expressing frustration with the ‘unyielding’ scientific debates about causation. This thesis is a plea only for the law to more correctly identify the key features that differentiate ‘good’ science from poor science (rather than valuing misleading criteria such as necessity and certainty); and also for courts to be clear that there is *no* perfect science. To people who lack basic understanding of science, ‘science is a monolith, a mystery, and an authority, rather than a method’<sup>648</sup> states Dr. Ben Goldacre, in his bestselling book, *Bad Science*. The judicial frustration frequently seen in disease litigation, this thesis has argued, is to a large extent a consequence of legal conceptual models that misunderstand science, and align poorly with available scientific evidence about causation (particularly disease causation). Over-inflated perceptions of the probative value of some forms of scientific evidence (such as some kinds of forensic testimony)<sup>649</sup> is as pernicious a threat to the cause of justice as is the undervaluation and step-sisterly treatment accorded to other scientific disciplines such as epidemiology. Broadbent observes that Lord Nimmo-Smith’s undermining of the epidemiological evidence in *McTear v*

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<sup>646</sup> *ibid*

<sup>647</sup> *XYZ v Schering* [2002] EWHC 1420 (QB) at [33]-[34] (Mackay J)

<sup>648</sup> B Goldacre, *Bad Science* (Harper Perennial 2009) 1

<sup>649</sup> See Chapter 2 section 2

*Imperial Tobacco*<sup>650</sup> on the grounds that no amount of such testimony could convince him of the truth of the assertion that smoking caused Mr. McTear's lung cancer is deeply fallacious, because it seeks truth as the justificatory basis for a conclusion. However, argues Broadbent, the notion of 'truth' is not sufficient to evaluate the validity of a scientific claim, because it assumes that there are, somewhere, sciences, that can provide absolute confirmation of the truth.<sup>651</sup>

Chapter 2 of this thesis earlier attempted to dispel this myth. 'Perhaps the most dramatic counterexample to the idea that a scientific theory must be true in order to be reasonably believed or relied upon is Newtonian physics' notes Broadbent.<sup>652</sup> 'Nobody could reasonably suggest that Newton was wrong to hold that space and time are absolute, even though modern physical theory suggests that he was *in fact* wrong: what makes Newton's theory reasonable is that he advanced compelling arguments for his view and made use of available evidence in a rational way.'<sup>653</sup> This thesis contends that the yardsticks UK tort law uses to evaluate the validity of scientific evidence, and to differentiate between science and pseudo-science, must be based on more sophisticated and informed criteria than those it currently uses, such as truth and certainty.

All legal decisions involving scientific evidence require management of the costs of error. Many legal commentators have expressed that judges need at least a basic understanding of the methods and statistics used to generate the error

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<sup>650</sup> [2005] CSOH 69

<sup>651</sup> A Broadbent, *Philosophy of Epidemiology* (Palgrave-Macmillan 2013) 62

<sup>652</sup> *ibid*

<sup>653</sup> *ibid*

rates.<sup>654</sup> This requires some basic degree of familiarity with research methods, and when and why different methods are employed. It also requires courts to be able to take a more critical approach to data, such as knowing the need to examine the size of the study sample, the strategies employed to reduce bias and confounding, etc. The Criminal Practice Directions<sup>655</sup> exhort judges to "be astute to identify potential flaws" in scientific evidence. Thus judges are expected to do more than passively wait for the opponent of the evidence to identify any flaws, which can only happen if they possess at least a preliminary foundational understanding of scientific evidence and how to utilise it appropriately.<sup>656</sup>

Chapter 1 cited the epistemic concerns courts express about epidemiology that hinder its effective application in UK law. Having now clarified the underlying legal misconceptions regarding science (Chapter 2), disease causation (Chapter 3) and the reasons for the futility of deterministic 'but for' questions in disease disputes (Chapter 4), this chapter will reevaluate the misconceptions about epidemiology that we outlined in Chapter 1. A closer analysis reveals that none of these constitute a fair justification for treating epidemiological evidence as invalid in this legal context. This thesis concludes that the judicial rejection of epidemiological evidence on grounds of its inconclusive nature is misconceived and deeply impractical.

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<sup>654</sup> A Wilson, 'Away From the Numbers: Opinion in the Court of Appeal' (2011) 75 *Journal of Criminal Law* 503, 504-505

<sup>655</sup> Criminal Practice Directions 2015, para.19A.6

<sup>656</sup> T Ward, 'DNA Evidence Alone as a Case to Answer' (2016) 80 *Journal of Criminal Law* 7, 9



Sections 1 and 2 highlight that the UK judicial view of epidemiology as little more than ‘statistics’ is misconceived, and does the discipline a disservice. Section 1 will examine how epidemiologists make inferences about causation, and the decision-making strategies that guide this. Section 2 explores in detail the specific judicial misconceptions about epidemiology. Section 3 discusses a better approach to the use of scientific evidence, generally, in the law. Section 3.1 examines admissibility issues, and assesses whether UK courts need better guidelines in this regard. Section 3.2 examines the legal tendency to fall victim to the ‘tyranny of numbers’: that is, the tendency to treat numerical or statistical data as an absolute that must be determinative of the outcome if considered. The resulting occasionally over-zealous interpretation of numbers leads to inevitable inaccuracy and legal disillusionment, causing courts to then react by swinging the other way and barring scientists from expressing probabilities in numbers altogether,<sup>657</sup> (believing that the problem is with numbers rather than the rigidity in their interpretation). This is an unfortunate over-reaction, and will result in the loss of a valuable epistemic and scientific communication tool. The better solution would be help decision-makers to recognise the need for judiciousness in the use of numerical and statistical evidence. Section 3.3 discusses the need to use expert testimony more effectively, evaluate its evidential validity, and to distinguish between fact and opinion in courtrooms. Section 3.4 discusses the need for more transparency and caution on part of expert witnesses. Section 4 suggests ways forward for a better use of epidemiological evidence in legal decision-making. Section 4.1 discusses the

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<sup>657</sup> As occurred in *R v T* [2010] EWCA Crim 2439

importance of judiciousness when weighing up the probative value of such evidence. Section 4.2 discusses the use of epidemiological evidence in evidence-based medicine (EBM), where population evidence is routinely used to make decisions about specific cases. This section suggests that the law could usefully borrow from such an approach.

Undoubtedly, such inferences suffer from methodological limitations and are not conclusive. Undoubtedly, too, courts must not base liability decisions on epidemiological evidence alone, but on the totality of all available evidence (as well as consideration of all other factors apart from factual causation that are relevant to liability in negligence). The methodological concerns raised in UK law are entirely appropriate if they are intended as a caution against blindly accepting all epidemiological inferences as necessarily always reliable. However, these objections do not constitute valid grounds for dismissing epidemiological evidence from consideration in legal disputes.

Disease causation is not just important to lawyers, it is also important for medicine, as causation plays a vital role in both treatment and prevention of disease. Lawyers seem to operate on the fundamental assumption that what they term 'population' evidence is entirely irrelevant and useless for individual decision-making; and this is a significant factor that leads to judicial reluctance about epidemiology in causal disputes. However, to many other professionals, that assumption would sound absurd. In medicine, for example, individual clinical diagnosis and treatment not only *is* routinely informed by general evidence, but *should be* informed by this, under the principles of evidence-based

medicine (EBM) that all practitioners are required to follow. The law should borrow from the methods that evidence-based medicine (EBM) uses when applying probabilistic, population-level evidence to individual cases.

Epidemiology, with its probabilistic causal models that do not assess potential causal factors in terms of necessity/sufficiency, but instead evaluate these factors in terms of the likelihood of their potential to *contribute* to disease, has much to offer under a probabilistic legal understanding of disease causation.

### ***SECTION 1: How epidemiology makes causal inferences***

*“Who knows, asked Robert Browning, but the world may end tonight? True, but on available evidence most of us make ready to commute on the 8.30 next day.”*<sup>658</sup>

-Sir Austin Bradford Hill (Professor Emeritus of Medical Statistics, University of London), 1965

The approach of epidemiology to causation is pragmatic and task-focussed, but it does not aim to provide a complete or conclusive account of causal mechanisms. As Coggon puts it, epidemiology: “has much to offer, but we cannot expect it to provide a complete understanding of why some people get a disease and others do not.”<sup>659</sup> In practice, as Broadbent<sup>660</sup> points out, epidemiological hypotheses are explicitly exception-ridden. They are framed not as universal generalisations, but as ‘measures of the *influence* of one factor on outcome, or on measures of the *strength* of an association, or of the *proportion* of an effect that is due to a

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<sup>658</sup> AB Hill, ‘The Environment and Disease: Association or Causation’ (1965) 58 Proceedings of the Royal Society of Medicine 295, 300

<sup>659</sup> D Coggon, ‘Commentary: Complex Disease- Responding to the Challenge’ (2006) 35 International Journal of Epidemiology 581, 582

<sup>660</sup> A Broadbent, ‘Inferring Causation in Epidemiology’ in P Illari, F Russo, J Williamson (eds.) *Causality in the Sciences* (Oxford University Press 2011) 47

particular factor or group of factors.’<sup>661</sup> Often, for many diseases, where events may have many causes, the aim is to measure the contribution of a specific factor on the disease: thus, probability theory and statistical inference are central to epidemiologic research.<sup>662</sup> Sir Austin Bradford Hill clarifies the probabilistic and pragmatic philosophy underlying epidemiology: ‘the ‘cause’ of a disease may be immediate and direct; it may be remote and indirect underlying the observed association. But...the decisive question is whether the frequency of the...event B will be influenced by a change in the environmental factor A. How such a change exerts that influence may call for a great deal of research. However, before deducing ‘causation’ ...we do not have to sit around awaiting the results of that research. The whole chain may have to be unraveled or a few links may suffice. It will depend on the circumstances.’<sup>663</sup>

Contrary to the judicial belief that epidemiological inferences of causation rest on simplistic numerical values or observed associations, the process of inferring causation in epidemiology is complex, and subject to much scrutiny.

Epidemiologists are alive to the risk of ‘false positives’, and thus robust, good-quality studies use a variety of techniques (both statistical and non-statistical) to reduce this risk. Coughlin<sup>664</sup> explains that epidemiologic researchers formulate hypotheses based upon their own insights and the insights of others. In order to test hypotheses, empirical data is collected and analysed according to a research protocol, and observations are assessed to see if a causal hypothesis should be accepted or rejected. Epidemiological results are examined across studies (for example, to examine the consistency of findings and to determine whether they

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<sup>661</sup> *ibid* 47

<sup>662</sup> SS Coughlin, *Causal Inference and Scientific Paradigms in Epidemiology* (Bentham 2010), 11

<sup>663</sup> Hill (n 14) 295

<sup>664</sup> Coughlin (n 18) 8-9

can be replicated) to draw causal inferences.<sup>665</sup> This is similar to Kuhn's description of how theories in most empirical sciences evolve, where existing scientific theories may be modified and new hypotheses generated for further testing.<sup>666</sup> The adequacy of a theory may be evaluated based upon its accuracy, consistency, simplicity, fruitfulness, and scope or reach.<sup>667</sup>

Epidemiological methods of data analysis rest on using techniques to put together, or synthesise, the evidence that has been discovered.<sup>668</sup> The aim is to use strategies that give other researchers the knowledge to separate causal from non-causal associations. Thus epidemiology emphasises the use of inductive reasoning of sufficient strength and rigour. One of the ways in which statistical information from multiple similar studies is combined is a technique known as meta-analysis, which provides statistical tests for the overall results, which can also sometimes be incorporated into a systematic review of the literature, and using experts to examine the evidence.<sup>669</sup> Epidemiological causal claims are substantiated by the use of both quantitative (e.g. counterfactual and structural models) and qualitative approaches (such as background knowledge and subject matter expertise about chronic disease epidemiology), as well as by using the Bradford Hill guidelines (outlined earlier in Chapter 1 section 1) as a heuristic aid for assessing whether observed associations are causal.<sup>670</sup>

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<sup>665</sup> *ibid*

<sup>666</sup> TS Kuhn, 'Objectivity, Value Judgment, and Theory Choice' in TS Kuhn (ed.) *The Essential Tension* (University of Chicago Press 1977) 320-343

<sup>667</sup> *ibid*

<sup>668</sup> GR Law and SW Pascoe, *Statistical Epidemiology* (CABI 2013) 44

<sup>669</sup> *ibid*

<sup>670</sup> Coughlin (n 18) 11

The eminent epidemiologist Sir Austin Bradford Hill was emphatic, however, that the Bradford-Hill factors were only meant as ‘viewpoints to examine associations’,<sup>671</sup> and that there can be no hard-and-fast rules of evidence that *must* be obeyed to determine when epidemiological evidence indicates causation: none of these criteria could bring indisputable evidence for or against cause-and-effect hypotheses, and none should be required as *sine qua non*.<sup>672</sup> Unfortunately, notes Haack, Hills’ ideas have often been misinterpreted and misapplied in legal contexts: the ‘legal penchant for convenient checklists’ has led many to construe his list as required criteria for the reliability of causation testimony.<sup>673</sup> Courts dealing with epidemiological evidence should be alive to such professional mispractice.

Probabilistic relationships are seen as surface phenomena of underlying causal mechanisms and relationships. Probabilistic theories only require that the cause should raise the possibility of the effect, and are applied more commonly to the assessment of general causal claims than of specific claims.<sup>674</sup> But: and this is a crucial aspect of probabilistic epidemiological evidence that bears repeating: there are no hard-and-fast rules about when a relationship becomes causal.<sup>675</sup>

The inference of causation is a complex one that rests on a number of considerations.

## **SECTION 2: Re-examining the judicial concerns about the validity of epidemiology**

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<sup>671</sup> Hill (n 14) 299

<sup>672</sup> *ibid* 299

<sup>673</sup> S Haack, ‘Correlation and Causation: The “Bradford-Hill Criteria” in Epidemiological, Legal, and Epistemological Perspective’ in M Martin-Casals, DM Papayannis (eds.) *Uncertain Causation in Tort Law* (Cambridge University Press 2016) 180

<sup>674</sup> Coughlin (n 18) 9

<sup>675</sup> *ibid* 11

As discussed earlier in Chapter 1, courts in the UK have expressed varying degrees of scepticism about such epidemiology in courtrooms; ranging from outright hostility (as seen in *McTear v Imperial Tobacco*<sup>676</sup>) to the more ambivalent but generally dismissive attitudes seen in *Sienkiewicz v Greif*<sup>677</sup> where several members of the Supreme Court cited various reasons for their discomfort with giving too much weight to epidemiological evidence. Lord Phillips' speech in *Sienkiewicz* offers a poor portrayal of epidemiology. Epidemiological evidence, His Lordship appeared to indicate, is not a satisfactory basis upon which to assess factual causation. McIvor points out that Lord Phillips' leading speech in *Sienkiewicz*, where he expresses serious doubts about the adequacy and reliability of epidemiological evidence, "seriously misconceives what epidemiologists actually do and the type of evidence that they bring to the legal table... (It) reflect(s) a mistaken belief that epidemiologists are mere statisticians, concerned solely with the calculation of incidence rates and capable only of producing 'naked statistics'."<sup>678</sup> This section will more closely examine the three distinct but related judicial beliefs about epidemiology that that seem to most hinder the effective application of epidemiology, as outlined in Chapter 1 (section 3).

## **2.1: Epidemiology and observational methods: exploring the "fetishisation of evidentiary hierarchies"<sup>679</sup>**

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<sup>676</sup> *McTear* [2005] CSOH 69

<sup>677</sup> *Sienkiewicz* [2011] UKSC 10

<sup>678</sup> C McIvor, 'Debunking Some Judicial Myths About Epidemiology and its Relevance to UK Tort Law' (2013) 21 *Medical Law Review* 553, 553-554 (Hereafter, McIvor, 'Debunking...')

<sup>679</sup> Sir Robert Jay, 'The Richard Davies QC Memorial Lecture 2015: "Standards of Proof in Law and

“Epidemiology” emphasised Professor Bradford Hill, “deals with the characteristics of human populations and is therefore more an observational than an experimental discipline.”<sup>680</sup> Thus epidemiological causal inferences rely heavily on inductive reasoning and observational study designs, rather than experimental methods. To many members of the UK judiciary, this appears to indicate that epidemiology cannot offer any useful evidence for causation. The Supreme Court in *Sienkiewicz v Greif* went to great lengths to discuss the methodological inadequacies of epidemiology, as cited earlier (Chapter 1, section 2.1.2). Most judges, Justice Jay points out in an extrajudicial article, subscribe to the fallacious notion that ‘gold plated experimental evidence is always superior to data derived from observational studies.’<sup>681</sup> There is a blinkered belief, both within the legal profession and outside it, that ‘real’ sciences must derive their data from experimental methods, rather than through observation (with the RCT usually placed at the pinnacle of this hierarchy, as the ‘gold’ standard).

The belief that experimental evidence is the only respectable form of scientific evidence is not only naïve (as the rest of this subsection will discuss), but it ignores the most fundamental problem about disease causation and toxic tort research: that it is usually impossible to carry out experiments to test out the cause of a disease without causing serious harm to participants, or intentionally risking their wellbeing. Further, Lord Dyson’s criticism in *Sienkiewicz* (cited earlier in chapter 1 section 3.1) that epidemiological methods can only establish associations between alleged causes and effects that are insufficient to

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Science: Distinctions Without a Difference?” (2016) *Journal of Personal Injury Law* 1, 4  
<sup>680</sup> AB Hill, ‘Observation and Experiment’ (1953) 248 *New England Journal of Medicine* 995-1001  
<sup>681</sup> Jay (n 35) 4



*conclusively* prove causation,<sup>682</sup> indicates a failure to recognise that **no** scientific method can conclusively prove causation. The blanket dismissal of evidence from observational sciences is mistaken on at least 2 counts, contend Rothman et al:<sup>683</sup> first, the non-experimental nature of a science does not preclude impressive scientific discoveries. They provide the example of plate tectonics, the evolution of species, planets orbiting other stars, and the effects of cigarette smoking on human health. Secondly, note Rothman et al, even causal theories that are based on evidence drawn from the most stringent experimentation are also only tentative: even the most careful and mechanistic dissection of individual events cannot provide more than associations, albeit at a finer level. ‘Experiments (including randomised controlled trials) do not provide anything approaching proof and may be controversial, contradictory or non-reproducible’ as the authors point out.<sup>684</sup>

Observational studies are usually employed to investigate whether certain exposures (or risk factors) are associated with the occurrence or progression of disease (that is, causal links).<sup>685</sup> Investigators might compare, for instance, outcomes of people who or suffer an exposure (or receive a treatment), with those who do not. However, they do not allocate patients to receive the exposure or intervention, and do not administer it. The most common observational studies are case studies, case series, case-control studies, cohort studies, and

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<sup>682</sup> *Sienkiewicz* (n 33) at [302]

<sup>683</sup> KJ Rothman, S Greenland, C Poole and TL Lash, ‘Causation and Causal Inference’ in KJ Rothman, S Greenland and TL Lash (ed), *Modern Epidemiology* (Lippincott, Williams and Wilkins 2008) 24

<sup>684</sup> *ibid*

<sup>685</sup> M Jeffreys and Y Ben-Shlomo, ‘Observational Studies’ in Y Ben-Shlomo, S Brookes, and M Hickman (eds), *Lecture Notes: Epidemiology, Evidence-Based Medicine and Public Health* 6<sup>th</sup> edn (Wiley-Blackwell, 2013) 36

historically controlled studies.<sup>686</sup> Although a detailed description of these is outside the scope of this thesis, it is important to note that, essentially, they are not experimental, controlled or randomised studies.

It is crucial for the legal profession to understand that for many research questions related to potential toxicities and disease causation, observational study designs may often be the methodology of choice. For such questions, there is often no experimental data to be found anywhere: not because scientists lack the required will or resource, but because the experiments usually cannot be conducted without harming participants in the study, or intentionally placing them at risk of harm. Carrying out 'gold standard' RCTs in order to experimentally test the potentially harmful effect of a substance on humans might require a scientist to administer a potentially deadly substance to a (willing or unwilling) human participant, in order to examine whether the consequences are, indeed, deadly. For obvious reasons, such research would be morally and ethically impossible. Observational research methods can provide invaluable- if probabilistic- empirical evidence in such situations; and often constitute the best available evidence about causal links, in the absence of which there would no data at all to guide decisions about prevention or treatment of harms resulting from toxic exposure.

Smith and Pell suggest, in a tongue-in-cheek article in the *British Medical Journal*, that those who look on observational data as an epistemically invalid and inferior way to acquire knowledge might wish to volunteer to verify whether

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<sup>686</sup> JH Howick, *The Philosophy of Evidence-Based Medicine* (Wiley-Blackwell 2011) 40

parachutes prevent death or major trauma in a randomised trial.<sup>687</sup> The tendency to idealise experimentation and devalue observational data is not unique to the legal world. It also has a long- and unfortunate- history in the world of science.<sup>688</sup> Early philosophers in the 17<sup>th</sup> century believed science could only be understood through experimentation, and the method of “deductive” inference: a trend that was further strengthened by the influential writings of Karl Popper, who held that science always required experiments that were designed and conducted to prove a hypothesis false.<sup>689</sup> Popper’s approach of “deductive falsification”<sup>690</sup> has attracted a huge and lasting following. Professor Sir Michael Rawlins, who was for several years Chair of the National Institute for Health and Care Excellence (NICE) (since its inception in 1999) notes in his Harvien Oration lecture that this tendency to formulate “hierarchies of evidence” (usually with RCTs placed at the top, and observational studies at the foothills) currently ‘bedevils’ the clinical world and medical policy-making.<sup>691</sup> This, Professor Rawlins notes elsewhere, is a deeply unfortunate development: “The notion that evidence can be reliably placed in hierarchies is illusory. Hierarchies place RCTs on an undeserved pedestal... although the technique has advantages it also has significant disadvantages. Observational studies too have defects but they also have merit. Decision makers need to assess and appraise all the available evidence irrespective as to whether it has been derived from RCT’s or

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<sup>687</sup> GCS Smith and JP Pell, ‘Parachute Use to Prevent Death and Major Trauma Related to Gravitational Challenge: Systematic Review of Randomised Controlled Trials’ (2003) 327 *British Medical Journal* 1459, 1459

<sup>688</sup> As explained by Professor Sir Michael Rawlins, ‘What Constitutes Credible Evidence of Effectiveness?’ (*Office of Health Economics*, June 2012) 1 <<https://www.ohe.org/publications/what-constitutes-credible-evidence-effectiveness>> accessed 04 April 2016

<sup>689</sup> KR Popper, *The Logic of Scientific Discovery* (first published 1959) (Routledge 2002) 57-72

<sup>690</sup> *ibid*

<sup>691</sup> Rawlins (n 44) 1-5

observational studies, and the strengths and weaknesses of each need to be understood if reasonable and reliable conclusions are to be drawn."<sup>692</sup>

Although most clinical “hierarchies of evidence” consider RCTs to offer stronger evidence than observational studies, *there are actually many situations where* observational studies would be preferred over experimental studies: e.g. where experimentation may be unethical, difficult to implement, inappropriate, or may lack generalizability.<sup>693</sup> Further, they often cannot be generalized beyond the population that has been studied, and can be ‘outrageously expensive.’<sup>694</sup>

Observational studies may be more tentative, but have three potential strengths, emphasises Professor Rawlins: they provide an alternative to RCTs in assessing benefit, play a critical role in the assessment of harms, and provide valuable data about generalisability.<sup>695</sup>

Howick points out that it is an odd but paradoxical truth of medicine that many therapies ‘in whose effectiveness doctors have the greatest confidence’ (such as tracheostomy to open a blocked airway passage, or the Heimlich manoeuvre to dislodge airway obstructions) evolved through observation, and have never been supported by randomised trials of any description.<sup>696</sup> Rothman et al<sup>697</sup> note that Edward Jenner developed the smallpox vaccine after observing the low incidence of smallpox amongst dairymaids, which led to an initial inductive hypothesis that

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<sup>692</sup> Professor Sir Michael Rawlins, ‘On the Evidence for Decisions about the Use of Therapeutic Interventions’ *The Harviean Oration* (Royal College of Physicians 2008) 2

<sup>693</sup> M Jeffreys and Y Ben-Shlomo (n 41) 37

<sup>694</sup> Rawlins, ‘What Constitutes Credible Evidence’ (n 44) 3

<sup>695</sup> *ibid* (n 44) 8

<sup>696</sup> Howick (n 42) 39

<sup>697</sup> Rothman et al (n 39) 18-19

having cowpox somehow conferred immunity to smallpox. (Under strict, anti-inductivist logical analysis, such a causal hypothesis based on an observed association would constitute an unjustified assumption about causation). Jenner, however, discovered after further investigation of his observational findings that the observed association in this case **was** evidence of a causal connection: the cowpox virus did confer immunity to smallpox. Jenner used this to develop a vaccine for smallpox that led to a revolution in healthcare for humankind. The widespread global public health vaccination campaigns that occurred in the 19<sup>th</sup> and 20<sup>th</sup> centuries based on Jenner's observational studies have led to the complete eradication of smallpox, a devastating disease that is estimated to have killed 300-500 million people in the 20<sup>th</sup> century alone. *Similarly*, the physician John Snow's investigation of the cholera epidemics in nineteenth century London used observational techniques to discover that cholera was spread by contaminated water (a discovery that was later confirmed through classic epidemiologic methods such as case control and retrospective cohort studies).<sup>698</sup> The discovery that scurvy is caused by Vitamin C deficiency, the discovery of the causes of malaria, and more recently the identification of HIV as the cause of AIDS, are all classic examples of inferring causality using traditional epidemiologic observational methods.<sup>699</sup>

Much of the scepticism about observational research arises from the fact that the conclusions of such research rely heavily on inductive reasoning. Chapter 2 (section 3.2.1) discussed some philosophical problems that beset inductive

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<sup>698</sup> AS Evans, *Causation and Disease: A Chronological Journey* (Plenum Press, 1993) 4-6

<sup>699</sup> For more detailed discussion of the significant contributions epidemiology has made to discovery of the causes of disease, see M B Bracken, *Risk, Chance, and Causation* (Yale University Press 2013)

reasoning. The logical conundrum inherent in inductive reasoning is referred to in philosophical and epistemological literature as the 'problem of induction'. The problem arises because 'no matter how many times a proposition is tested and shown to be true, it is logically impossible to be absolutely certain we will have the same result next time it is tested.'<sup>700</sup> Thus, no matter how many times my flicking of a switch results in the light turning on, it is impossible for me to prove with certainty through observation alone that the flicking of the switch causes the light to turn on. There is no point at which I could guarantee that that the same thing will happen the next time I flick the switch. The problem arises because observers cannot perceive causal connections, but only a series of events. Similarly, the failure of a case cohort study to find statistically significant results is not always evidence of a lack of causation.<sup>701</sup>

Professor Rawlins points out that disputes about whether deduction is superior to induction are "absurd": whole swathes of science such as astronomy, geology, evolutionary biology and genetics depend largely or exclusively on inductive inference.<sup>702</sup> Notes Miller: "The problem of induction cannot be escaped. But only philosophers are troubled by their failure to find a synthetic and a priori concept of cause. For most people, pragmatism is all that is needed to make sense of the causes of the great majority of the phenomena they encounter in everyday life."<sup>703</sup>

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<sup>700</sup> As also acknowledged by Popper and others. See further A Rosenberg, *Philosophy of Science: A Contemporary Introduction* (Routledge 2005) 121

<sup>701</sup> A Broadbent, 'Epidemiological Evidence in Proof of Specific Causation' (2011) 17 (4) *Legal Theory* 237, 239

<sup>702</sup> Professor Sir Michael Rawlins (n 44) 1

<sup>703</sup> C Miller, 'Causation in Personal Injury: Legal or Epidemiological Common Sense?' (2006) 26 *Legal Studies* 544, 547

Sir Austin Bradford-Hill (1897-1991), amongst the most eminent British medical statisticians, who was amongst the leading proponents of the much-valued RCTs as well as author of the Bradford Hill factors we have discussed at several points through this thesis, had this to say about evidentiary hierarchies: “Any belief that the controlled trial is the only way would mean not only that the pendulum had swung too far, but that it had come right off the hook.”<sup>704</sup> Cranor points out the utility of case studies in examining causal links from toxic exposures, and notes that they deserve much more respect in this area of the law than they receive from courts.<sup>705</sup> Recent critiques of evidence-based medicine (EBM) suggest that rigid hierarchies of evidence should be replaced by the requirement that all evidence of a sufficiently high quality (including observational studies) should be considered in trying to come to a decision.<sup>706</sup>

Case control studies do not just use observations to make causal inferences from simple associations, but subject observational data to a great deal of further scrutiny to decide if the associations do, indeed, suggest causation. There are, undoubtedly, some methodological problems with observational studies. For example, they are prone to a variety of biases, such as self-selection bias, allocation bias and performance bias.<sup>707</sup> They suffer from confounding, unlike well-conducted RCTs. However, it is equally important to note that there are special observational designs that have been designed to help reduce these

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<sup>704</sup> A Bradford Hill, ‘Reflections on the Controlled Trial’ (1966) 25 *Annals of the Rheumatic Diseases* 107

<sup>705</sup> C Cranor, *Toxic Torts: Science, Law and the Possibility of Justice* (Cambridge University Press 2007) 115

<sup>706</sup> Howick (n 42) 39-45

<sup>707</sup> Howick (n 42) 40

problems. Cohort studies often provide the best evidence that the exposure-outcome association is causal. Case-control studies also help to reduce other sources of bias, and are superior to cross-sectional studies, because epidemiologists attempt to ascertain subjects' exposure status before the onset of disease.<sup>708</sup> Case studies can also be used, in fact, to *refute* false causal hypotheses drawn from associations between an event and its hypothesized effect. An important example is the now-discredited theory of the link between the MMR vaccine and autism (a theory put forward by Dr. Andrew Wakefield and others in *The Lancet* in 1998).<sup>709</sup> This study was later retracted due to evidence of deliberate falsification of data: a fact that *observational* studies helped bring to notice.

Thus, it is true, as Lord Dyson notes, that associations do not necessarily indicate causal connections. However, associations do indicate the possibility of a connection that can be tested. Thus, inferring that the rooster's crowing causes the sun to rise simply because you have observed an association between the two events would indeed be fallacious, but this observation does supply a hypothesis that can be tested, as Rothman et al. point out: if you wring the rooster's neck before the sun rises and the sun still rises, you have disproved that the rooster's crowing is a necessary cause of sunrise.<sup>710</sup> Good case studies rest on a principle of diagnostic reasoning that is essential to all causal

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<sup>708</sup> Jeffreys and Ben-Shlomo (n 41) 41-43

<sup>709</sup> F Godlee, 'Wakefield's Article Linking MMR Vaccine and Autism Was Fraudulent' (2011) 342 *British Medical Journal* 22

<sup>710</sup> Rothman et al (n 39) 20



judgments: 'What makes case studies good evidence about causation is the *analysis* to which they are subjected and *how scientists reason about them*.'<sup>711</sup>

Evidence, as Professor Sir Michael Rawlins put it, has but one purpose: to inform decision makers. Both experiments and observations have a crucial evidentiary role to play in this. It is now imperative for decision-makers to 'avoid adopting entrenched positions about the nature of evidence, and for both to accept that the interpretation of evidence requires judgment.'<sup>712</sup> The use of hierarchies as a replacement for judgment is dangerously wrong, as Rawlins emphasises: it is judgment, conditioned by the totality of the evidence base, that lies at the heart of decision-making.<sup>713</sup> Scientists are at long last starting to acknowledge that observational methods may have long been undervalued and overlooked in the tendency to 'fetishise evidentiary hierarchies'. More prescient philosophers of science (including those of such eminence as Francis Bacon, Rene Descartes and Thomas Hobbes) had many centuries ago pointed out that on philosophical grounds the observational approach was to be preferred to the experimental.<sup>714</sup> However, while scientists are beginning to properly recognise the value of observational methodologies, the law (in the UK, at least) appears to have remained entrenched in rigid and outdated beliefs.

## ***2.2: General versus specific causation:***

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<sup>711</sup> Cranor (n 61) 115

<sup>712</sup> Rawlins (n 44) 13

<sup>713</sup> *ibid* 13

<sup>714</sup> *ibid* 1

The second major legal objection to epidemiological evidence is that epidemiologists derive their knowledge primarily from the study of populations.<sup>715</sup> The judicial perception that it is impossible to apply epidemiological studies to determine causation in individual cases is amongst the main reasons that the claim in *McTear*<sup>716</sup> did not succeed. Lord Nimmo-Smith stated, in unqualified terms, that there is a "fallacy of applying statistical probability to individual causation."<sup>717</sup> It will be recalled from Chapter 1 (section 2.1.1) that the court in *McTear* entirely dismissed epidemiological evidence in a claim for lung cancer allegedly caused by smoking, asserting rather oddly that not only could epidemiology not prove causation, but that it could not even provide information on the *likelihood* that there was a causal connection. The population attributable risk, His Lordship went on to assert as we saw earlier, was a measure for populations only, and did not imply a likelihood of disease occurrence within an individual.

The distinction between general and specific causation has been debated for many decades, and such anxieties are not exclusive to the law. From the time of John Stuart Mill, notes Coughlin, philosophers have distinguished between 'singular' causal claims (e.g. person A's lung cancer was caused by smoking) versus general causal claims (cigarette smoking causes lung cancer).<sup>718</sup> However, such philosophically engaging debates have not obstructed us from utilising the value that 'general' evidence can add to specific decision-making in professions apart from the law. Medicine is one obvious example. Routine clinical practice

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<sup>715</sup> See Chapter 1 section 3.2

<sup>716</sup> *McTear* (n 32)

<sup>717</sup> *McTear* (n 32) at [6.184]

<sup>718</sup> Coughlin (n 18) 9

and evidence-based medicine (EBM) requires practitioners to constantly apply data obtained from population studies in order to make (often life-and-death) diagnostic and treatment decisions about the individual before them. Judges have missed the point that most empirical research is done at the population level, yet it is often used to make statements about particular cases.<sup>719</sup> UK law has very high regard from experimental or RCTs (see above, section 2.1), but even RCTs are a population-based empirical method. For example, an RCT to test out the efficacy of a new treatment, whether for cancer or schizophrenia, is conducted with large random samples of individuals drawn from within a population. The results are then extrapolated to individual cases in order to assess the chances of success and to make specific clinical decisions. There is no guarantee that any treatment that works for a large number of people in the study population will work for every individual it is administered to. Nonetheless, population data informs us about the likely outcomes for individuals, and that is the best that science can do.

Wright<sup>720</sup> refines the argument against the legal use of epidemiology further, by drawing a distinction between *ex ante* (before the event has happened) and *post ante* (after the event has happened) probabilities. Wright contends that it is epistemically valid to use 'general' or population evidence in individual decision-making *ex ante*, but not *ex post*. Wright's reasoning, based mostly in 'speculative metaphysics',<sup>721</sup> is, for uncertain reasons, highly convincing to lawyers. This has caused many practical problems in the proper legal application of

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<sup>719</sup> D Faigman, 'Judges as "Amateur Scientists"' (2006) 86 Boston University Law Review 1207, 1220

<sup>720</sup> Richard Wright, "Liability for Possible Wrongs: Causation, Statistical Probability, and the Burden of Proof" (2008) 41 *Loy. LAL Rev.* 1295, 1320-1322

<sup>721</sup> A Broadbent, 'Epidemiological Evidence in Proof of Specific Causation' (n 60) 263

epidemiological evidence. As Miller notes: 'In particular, it is the common law's reluctance to accept probabilistic estimates of the uncertainty of past events (in contrast to its ready acceptance of the counterfactual chance of 'what might have been' and of the future chance of 'what might yet occur') which accounts for a large part of the incoherence.'<sup>722</sup>

With respect, this thesis contends that Wright's distinction (between the validity of this evidence for estimating *ex ante* versus *post ante* probabilities) is, to borrow a phrase from Prosser, "moonshine and vapour",<sup>723</sup> at least in terms of its practicality. It is difficult to find logical force in the argument that data that is epistemically valid for making probabilistic predictions about future events is epistemically invalid for formulating probabilistic explanations of past events (particularly when we have no other evidence to ascertain the past event, as is usually the case in disease claims; and providing we assume the absence of extrasensory abilities, we can usually safely do). The tasks of predicting the future or explaining the past are similar: both need to be able to accommodate probabilistic reasoning. Thompson notes that: 'what is true of prediction is equally true of explanation. Although there is no tight symmetry between explanation and prediction, they are two sides of the same coin.'<sup>724</sup> Future predictions are, in any case, often based on past information and experience. Making causal inferences requires a synthesis of both sources of data. As Prosser points out, proof of the relation of cause and effect can never be more than "the

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<sup>722</sup> C Miller, 'Causation in Personal Injury' (n 59) 545

<sup>723</sup> WL Prosser, 'Proximate Cause in California' (1950) 38 California Law Review 369, 382 (Professor Prosser, it must be clarified, used this phrase in a different context)

<sup>724</sup> RP Thompson, 'Causality, Theories and Medicine' in P Illari, F Russo, J Williamson (eds.) *Causality in the Sciences* (Oxford University Press 2011) 34

projection of our habit of expecting certain consequents to follow certain antecedents merely because we have observed those sequences on previous occasions.”<sup>725</sup>

Further, Wright objects to ‘naked statistics’ on the grounds that it is not ‘probative’: that is, that statistics only comprise general statements that do not remove the uncertainty surrounding the causal connection between tortfeasor’s action and the claimant’s injury.<sup>726</sup> But what Wright fails to note is that particularistic evidence, too, has no claim to certainty. Why, then, does particularistic evidence necessarily have greater epistemic value than ‘general’ evidence? Professor David Kaye points out the naiveté of the legal scepticism about purely statistical data. All evidence, he points out, whether it is particularistic empirical data or pure statistics, is ultimately probabilistic, so that the legal attitude that particularistic data is somehow ‘qualitatively different, more reliable, or more trustworthy than statistical data is unsupportable.’<sup>727</sup> An example of this is eyewitness testimony (that is, incidentally, so frequently used in criminal litigation): this evidence is ‘particularistic’, but as Justice Jay notes: ‘...is almost always flawed because human memory is so unreliable. No one would claim this resolution to be scientific.’<sup>728</sup> It is difficult to imagine what evidence would satisfy the quest for particularistic evidence in disease litigation, given that no one can see or record the process of a toxin lodging in, for example, a person’s lungs and altering cellular and chemical structures. Wright offers no

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<sup>725</sup> Prosser (n 79) 382

<sup>726</sup> R Wright ‘Causation, Responsibility, Risk, Probability, Naked Statistics and Proof: Pruning the Bramble Bush by Clarifying the Concepts’ (1988) 73 Iowa L Rev 1001

<sup>727</sup> D Kaye, ‘The Limits of the Preponderance of the Evidence Standard: Justifiably Naked Statistical Evidence and Multiple Causation’ (1982) American Bar Foundation Research Journal 487, 488-492

<sup>728</sup> Jay (n 35) 3

solution to this problem, but continues to advocate a rejection of generalised statistical evidence. The sooner the law gives up the fantasy that we will very shortly find conclusive and particularistic evidence of disease causation (at least, in the present stage of human evolution), the clearer it will become that it is time to give due value to pragmatic, best evidence about the causal question. Any evidence an individual has that Paracetamol will help their headache, or chemotherapy their cancer, is ultimately probabilistic and uncertain. When a person makes a decision to accept a medically recommended treatment, the personal decision (as well as the medical practitioner's recommendation) is largely informed by general evidence of efficacy and risk. Uncertainties about whether that treatment will work for us, as individuals, does not lead us to turn away from these treatments and choose prayer as our only recourse (at least, not most of us). Using population evidence to guide our treatment decisions when there is no specific evidence is a rational, scientific decision: we have used the best evidence that is available, and made an inference to the best explanation. Wright, it must be noted, advocates the very opposite of the scientific approach when he proposes that the available evidence must be rejected as it is imperfect, without providing any better alternative solution. To believe that personal beliefs and hunches are about as useful a guide to making factual decisions as statistical data would be akin to deciding that prayer or homeopathy is about as well-evidenced a treatment for cancer as chemotherapy, because chemotherapy is not always effective.

***2.3: Is a Relative Risk ratio (RR) of >2 necessary, or sufficient, to establish causation?***

The RR (relative risk) is a measure that epidemiologists use to compare the incidence of a condition (or disease) in a group that has been exposed to a factor, with a group that has not been exposed to that factor. The relative risk, in other words, tells us how much larger the exposed risk is than the unexposed.<sup>729</sup> The balance of probabilities or ‘more likely than not’ test has sometimes been misinterpreted by courts in statistical terms as the requirement that the statistics should show that disease incidence in individuals exposed to the agent should be at least twice the incidence of disease in those not so exposed:<sup>730</sup> that is, courts tend to view an  $RR > 2$  as equivalent to causation on the balance of probabilities. Courts call this the ‘doubles the risk’ test (described earlier in Chapter 4 section 2.3).

This is a crude conceptualisation of how epidemiologists make causal inferences, and does epidemiology a disservice as it leads to the legal undermining of this discipline. Further, this belief also leads to a variety of legal errors in disease litigation, through mechanistic applications of rigid RR criteria in the form of the ‘doubles the risk’ test. Epidemiologists do not view ‘doubling of the risk’ alone as proof of specific causation. The epidemiological inference of causation, as we have discussed above (see section 1) and extensively throughout this thesis, is a complex one that rests on a number of considerations. Epidemiologists constantly acknowledge that inferences about both general and individual causation cannot be proved by statistical probability alone.

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<sup>729</sup> A Broadbent, ‘Causation in Epidemiology and Law’ in M Freeman and M Zeegers (eds.) *Forensic Epidemiology: Principles and Practice* (Elsevier 2016) 118

<sup>730</sup> P Feldschreiber, L Mulcahy and S Day, ‘Biostatistics and Causation in Medicinal Product Liability Suits (2011) in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 188-189

An RR of 2 or more does not necessarily discharge the burden of proof on the balance of probabilities, and courts may need to take account of additional evidence: every association does not always turn out to be causative.<sup>731</sup> Further, epidemiologists take pains to stress, RR is inadequate in many ways as a sole yardstick: it needs to be interpreted in the light of absolute risk or background risk.<sup>732</sup> The background incidence rate of a disease is an important contextual factor to consider in interpreting RR values. For example, let us assume an incidence of a disease in an unexposed population is 1 in 1000 people, and the incidence in an exposed population is 2 in 1000 cases. The RR here in this simple hypothetical instance would be 2, but the absolute risk in such a situation is so low, that it would be difficult to make any reliable inference from it: in other words, it is very difficult to rule out such a result occurring by chance. Basing a causal inference on an  $RR > 2$  in such a scenario could lead to false positive conclusions about causation, and more evidence would certainly be needed.

On the other hand, however, if the absolute risk of a disease is very high, then even an RR of something *less* than 2 might be significant. Let us alter our hypothetical scenario slightly, so that let us imagine the incidence of a different disease in an unexposed population is 200 in 1000 people; and the incidence of that same disease in an exposed population rises to 350 in 1000 cases. The RR here is slightly *less* than 2, but in absolute terms, this indicates a far more significant increase in incidence of the disease following exposure than in our

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<sup>731</sup> *ibid* 188-189

<sup>732</sup> S Greenland and JM Robins 'Epidemiology, Justice, and the Probability of Causation' (2000) 40 *Jurimetrics* 321 at 322-323



previous scenario. 150 *more* people out of every 1000 people in exposed population contracted the disease. This has a far lower chance of having occurred by chance than our previous scenario, and depending on other evidence, may indicate causation. Epidemiologists would however consider several other possibilities and try to rule out sources of error before making any causal hypotheses. They might scrutinise factors such as the study design and sample size; the possibility of confounding factors and whether these were controlled for (for example, was the exposed population coincidentally much more elderly and thus more prone to infection than the unexposed population?) and so on. If the two groups were relatively well matched in terms of demographic characteristics, the conclusion is likely to be much more robust). Thus the RR values viewed in isolation are an inadequate basis on which to base inferences of causation. The significance of the RR value would depend on absolute risk, study design and many other contextual factors. This illustrates the importance of considering the total evidence and contextual factors when examining scientific data. Mechanistic applications of numbers or statistics, without consideration of the context, will lead to false and illogical conclusions.

There are several reasons why an RR value of less than 2 does not necessarily negate causation. It is often unusual for a new risk factor affecting large numbers of people to be confirmed as carrying a relative risk  $>2$  for a major disease, notes Coggon,<sup>733</sup> and epidemiologists have responded to the challenges by developing much more sophisticated statistical techniques to reduce the possibility of erroneous causal inferences. One approach, he explains, has been to conduct

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<sup>733</sup> D Coggon, 'Commentary: Complex Disease' (n 15) 581

very large studies with detailed assessment of exposure to a wide range of risk factors, carefully control for confounding, and then to apply advanced statistical techniques to look at the effects. With this method even an RR of 1.5 can be statistically significant, and the findings from such methodologies have achieved wide enough acceptance to influence public health policy, as Coggon points out.<sup>734</sup>

Goldberg<sup>735</sup> notes that US courts also disagree as to the proper role of the doubling of risk theory in deciding questions of both sufficiency and admissibility of scientific evidence of causation in toxic tort cases. They do not agree on whether to adopt the doubling of risk as a threshold, nor do they agree on the meaning of such a threshold: 'Many courts accept the doubling of the incidence of disease in group studies; some courts insist on doubling of risk as a minimum threshold for establishing specific causation. Others have recognised that if other known causes can be identified and eliminated, something less than a doubling would still be sufficient to find specific causation.'<sup>736</sup> Thus, notes Goldberg, the requirement of an RR>2 for the admissibility or sufficiency of epidemiological evidence is subject to much scepticism.<sup>737</sup>

The legal insistence on an RR>2 displays the rather superficial and incomplete legal understanding about epidemiological measures, and ignorance about the fact that these must be seen and interpreted in context. The tendency to base

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<sup>734</sup> *ibid*

<sup>735</sup> R Goldberg, 'Epidemiological Uncertainty, Causation, and Drug Product Liability' (2014) 59 *McGill Law Journal* 777, 787

<sup>736</sup> RS Carruth & BD Goldstein, 'Relative Risk Greater Than Two in Proof of Causation in Toxic Tort Litigation' (2001) 41 *Jurimetrics Journal* 195, 202–03.

<sup>737</sup> CF Cranor, 'The Challenge of Developing Science for the Law of Torts' in Richard Goldberg (ed), *Perspectives on Causation* (Hart 2011) 234–238; 281

legal conclusions on rigid statistical data such as an RR value of >2 is proof also reflects a wider fallacy that afflicts the legal use of scientific evidence generally: the ‘tyranny of numbers’. Due to this fallacy, numbers, which for scientists are a valuable assessment and communication tool to clarify expressions of probability, are interpreted as meaning much more than they do. This fallacy also played a significant role in the wrong decision in the case of *R v Sally Clark*,<sup>738</sup> discussed earlier in Chapter 2 (section 2.2).

### **SECTION 3: More effective use of general scientific testimony**

The preceding discussions in this thesis have attempted to illustrate that, as Rothman et al. put it,<sup>739</sup> all fruits of scientific work, whether in epidemiology or in other scientific disciplines, are at best only tentative formulations of a description of nature, even when the work itself is carried out with no mistakes. Even causal hypotheses that have an extremely high degree of certainty (for example, the link between smoking and lung-cancer is almost universally accepted) are not definitely proved with absolute certainty as might accompany, say, a mathematical theorem.<sup>740</sup> This is, simply, the nature of empirical inferences. The subsections below outline some specific suggestions that will aid better use of scientific testimony generally in legal decision-making.

#### **3.1: Admissibility issues: *do we need Daubert-type guidelines in UK courts?***

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<sup>738</sup> (No 2) [2003] EWCA Crim 1020

<sup>739</sup> Rothman et al (n 39) 24

<sup>740</sup> *ibid*

Much of the debate around the scientific evidence in US law has focussed itself around issues of admissibility. *The Daubert*,<sup>741</sup> *Joiner*<sup>742</sup> and *Kumho*<sup>743</sup> decisions of the US Supreme Court clarify a more active role “gatekeeping” for judges in evaluating scientific testimony, both for relevance and reliability. Scientific evidence presented before courts must meet specified criteria and demonstrate that it is the product of sound scientific methodology and reasoning.<sup>744</sup> By contrast, as Edmond<sup>745</sup> notes, the ‘laissez-faire’ attitude of UK courts regarding admissibility shows that the UK is lagging far behind many similar jurisdictions in this area. When it comes to determining the admissibility (and probative value) of expert evidence in criminal proceedings, English courts have placed great store in trial safeguards and relied heavily on a range of relatively simple heuristics such as: whether the forensic analyst has training, study or experience in a legally-recognised “field” (a peculiarly legal incarnation of “reliability”, observes Edmond) and perceived utility or necessity.<sup>746</sup> The Law Commission of England and Wales has recently added its voice to the debate with the publication of a Consultation Paper on the admissibility of expert evidence, which recommends the creation of a new statutory rule that would require the trial judge to assess evidentiary reliability as a matter of admissibility.<sup>747</sup>

However, this is not, of itself, sufficient. Formulating more stringent admissibility guidelines that end up being applied mechanistically, without improving legal

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<sup>741</sup> *Daubert* 590 US 579 (1993)

<sup>742</sup> *General Electric Co. v Joiner* 522 US 136 (1997)

<sup>743</sup> *Kumho Tire Co. Ltd. v Carmichael* 526 US 137 (1999)

<sup>744</sup> *Convergence of Science and the Law: A Summary Report of the First Meeting of the Science, Technology and Law Panel* (National Academies Press 2001), 2, 6.

<sup>745</sup> G Edmond, ‘Legal versus Non-legal Approaches to Forensic Science Evidence’ (2016) 20 *International Journal of Evidence and Proof* 3, 3-5

<sup>746</sup> *ibid* 5

<sup>747</sup> Law Commission, *Expert Evidence in Criminal Proceedings in England and Wales* (Law Com No 325, 2011) 1.8-1.9

understanding of how the scientific method works will not resolve all problems that arise in the legal use of science. Cranor observes that American courts, where federal judges have been given greater responsibility to screen expert testimony, still do not always understand the nature of scientific inferences and the legal relevance of different kinds of scientific studies, which has prevented scientists in American courts from utilising the full body of data they would normally consider in coming to conclusions about causation.<sup>748</sup> Unless judges are more informed about at least the basic principles that determine the reliability and validity of scientific testimony, such criteria only become mechanistic exercises that do little to help courts identify the right expert, ask them the right question, or to judge whether they have the expertise to answer it. Nonetheless, it is undeniable that the responsibilities imposed on judges in the US in regarding of scrutinising evidence and the guidelines provided to them to do so is a great improvement on the situation in UK law. With clearer guidelines for courts and the availability of training for judges, the responsibility to assess reliability should not be too onerous for courts. As Ward<sup>749</sup> notes, in assessing reliability for the purpose of determining admissibility, the judge's task is not to determine whether the evidence is, in fact, accurate: it is only to determine whether there are sufficient indicia of reliability that a jury could rationally rely on the evidence in a way that could make a difference to the verdict. How strong the indicia of reliability have to be depends on many factors, including on the other evidence in the case.<sup>750</sup>

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<sup>748</sup> Cranor, 'The Challenge of Developing Science' (n 93) 261

<sup>749</sup> T Ward, 'A New and More Vigorous Approach' to Expert Evidence in England and Wales' (2015) 19 *International Journal of Evidence and Proof* 228, 232

<sup>750</sup> *ibid*

### ***3.2: Escaping the ‘tyranny of numbers’: “People are not numbers”, but numbers can help scientific communication***

Courts tend to find the idea of using numerical or statistical data in legal decision making a heavy burden. Section 2 (above) discussed the discomfort courts feel about applying statistics to decision-making to individual cases. Pundik<sup>751</sup> notes the wide contradictions that exist in the use of statistical data in the law. Despite the fact that such evidence is regularly adduced in litigation (e.g. in DNA matching, medical negligence cases to prove loss of chance, human rights law etc.), its use is accompanied by a ‘vaguely articulated, almost intuitive’ judicial anxiety that statistical data is somehow epistemically inadequate for legal decision-making’.<sup>752</sup> This judicial view is expressed every now and again across different legal settings,<sup>753</sup> but has received little rational scrutiny or justification. Even people who object intuitively to the use of statistical evidence in legal decision-making, points out Pundik,<sup>754</sup> acknowledge that such evidence actually improves the accuracy of decisions. Meadow and Sunstein<sup>755</sup> argue strongly that reliance on statistical data would dramatically increase the accuracy and rationality of tort law in many settings (including but not limited to medical law). The fallible opinions of isolated experts, they argue, should be supplemented or replaced by statistical data, as opinions are crude, second- best alternatives, and are inferior to the data that they approximate: “In any case in which a disputed question calls for expert testimony about ordinary practice, it is hazardous to

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<sup>751</sup> A Pundik, ‘The Epistemology of Statistical Evidence’ (2011) 15 *International Journal of Evidence and Proof* 117, 117

<sup>752</sup> *ibid* 117-118

<sup>753</sup> It also occasionally surfaces, for example, in UK criminal law contexts, as seen in *R v T* (n 13)

<sup>754</sup> Pundik (n 107) 132-136

<sup>755</sup> W Meadow and CR Sunstein, ‘Statistics, Not Experts’ (2001) 51 *Duke Law Journal* 629, 631

rely on what particular experts recall. If the goal is accuracy in adjudication or regulation, it is far more sensible to make the outcome turn on statistical evidence.”<sup>756</sup>

The objections about statistical evidence, then, may largely be due to the anxiety that such evidence will be given too much weight by juries and judges when adduced: in other words, the objections may not be so much about the statistics, but about the worry that they may be interpreted erroneously. This has sometimes led to barring statistical or numerical testimony altogether in some contexts. The Court of Appeal in *R v T*<sup>757</sup> held, unequivocally, that evidence regarding footwear prints should **not** be expressed in numerical terms, such as likelihood ratios. Probabilistic calculations in regard to footwear mark comparisons was, in the view of the court, “inherently unreliable and gives rise to a verisimilitude of mathematical probability. It cannot be right to seek to achieve objectivity by reliance on data which does not allow this to be done.”<sup>758</sup> Cases such as *R v Sally Clark*,<sup>759</sup> discussed earlier, may have served to intensify such anxieties about statistics. The Court of Appeal in that case, it might be recalled from Chapter 2 (section 2.2), attributed the earlier erroneous guilty verdict in the trial court to the overly persuasive nature of statistics, stating with disapproval that Professor Meadows’ ‘graphic reference’ to long odds winners at the Grand National was likely to have had a major impact on the minds of the jury.<sup>760</sup> However, the Court of Appeal may have missed the point that the ‘guilty’

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<sup>756</sup> *ibid* 645-646

<sup>757</sup> *R v T* (n 13)

<sup>758</sup> *R v T* (n 13) at [87]

<sup>759</sup> *Sally Clark* (n 94)

<sup>760</sup> *ibid* (n 94) at [178] (per Lord Justice Kay)

verdict in *Clark* was actually due a combination of many unfortunate factors. The statistical errors were also partly due to fact that the court relied on statistical testimony from an expert who was not a statistician but a doctor, and thus the name and reputation of the expert became decisive, rather than their science or specialism: a common legal problem that we have already pointed out. Further, another expert had deliberately withheld evidence in that case, and that had also played a significant role in the erroneous decision.

Most importantly, however, the problem of inflated importance being given to the statistics can be easily remedied if courts realise that the numbers generated by statistical computations are simply probabilistic communication tools. In other words, we need to find a way for courts to be *informed* by the numbers, while resisting the *tyranny* of the numbers. If courts reject potentially valuable and illuminative statistical information just because of such anxieties, this would be a knee-jerk reaction that would discard ‘the baby with the bathwater’, so to speak. Redmayne et al,<sup>761</sup> while crediting the court in *R v T* for emphasising the importance of pre-trial hearings and robust case management,<sup>762</sup> are critical of its ‘irrational directive’ that footwear evidence cannot be expressed in numerical terms at all. This directive, they point out, applies solely to footwear analysis, while courts will readily accept likelihood ratios in regard to several other forms of forensic evidence such as DNA matching. An even bigger problem with this approach is that prohibiting experts from presenting data in numerical terms altogether, and insisting they replace numbers with terms such as “moderately likely”,

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<sup>761</sup> M Redmayne, P Roberts, C Aitken, G Jackson, ‘Forensic Science Evidence in Questions’ (2011) 5 Criminal Law Review 347, 347-355

<sup>762</sup> *R v T* (n 13) at [105]



“highly likely” etc. would deprive experts of the standard tools of their science, and lead to absurd inconsistencies and subjectivity. Such a situation, Redmayne et al. caution, is likely to lead to “a forensic free-for-all, with one (cautious) expert's “moderate support” equating to another (cavalier) expert's “very strong support”, predictably leading jurors astray or leaving them entirely baffled.”<sup>763</sup> Further, numerical quantification also has several other advantages, such as its ability to encourage greater transparency and critical scrutiny of evidence, as well as the use of logical thinking and rigour in the production of evidence. All these advantages, Redmayne et al caution, would be lost by such a blanket prohibition.

The worry that numbers will end up dictating the decision can be resolved by better awareness and training for lawyers about what the numbers do- and do not- mean. Numbers generated by statistical tools are simply a standardized way of communicating the strength of probability, not guarantees or absolutes. As Redmayne et al clarify: “Suffice it here to emphasise that numerical quantification does not necessarily imply any claim to objectivity. When a weather forecaster speaks of a 20 per cent chance of snow, it should be clear that, rather than expressing any verifiably objective fact about the empirical world, this is a statement of probability regarding future events informed by appropriate meteorological data and expertise.”<sup>764</sup>

Rather than discard all evidence that is probabilistic or possesses a degree of subjective interpretation, experts should be clearly required to be transparent about the subjectivity in their conclusions. As *R v T* rightly stresses, it is vital for

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<sup>763</sup> Redmayne et al (n 117) 351-353

<sup>764</sup> *ibid* 354

scientists to heed some basic principles in the presentation of their numerical evidence, such as ensuring they do not mislead courts into believing the likelihood ratios are possessed of false precision.<sup>765</sup> Numerical conclusions should be properly supported by adequate data, and juries must not be bamboozled by statistical evidence, but provided that experts do so, it is “(b)etter all round if experts are simply encouraged to give their best evidence as clearly, intelligibly, honestly, and transparently as they are able, and in accordance with the precepts of scientific validity, logic and reason.”<sup>766</sup>

### ***3.3: Expert testimony: evaluating weight of evidence, and distinguishing between fact and opinion***

An associated point, raised earlier in Chapter 2 (section 2.3), is that both lawyers and scientific experts must be mindful of the distinction between facts and opinion. In the context of epidemiology, this is a crucial issue, because it often leads to courts favouring, as a general rule, sometimes questionably scientific ‘specific’ (e.g. medical or clinical) evidence over more scientifically valid ‘general’ evidence. Courts need to be aware that in many situations there simply is no available scientific evidence that maps directly to the legal question (an especially frequent scenario in disease disputes). Where the evidence cannot directly answer what caused this particular claimant’s disease, an expert giving testimony about specific causation will often take available general evidence about factors that contribute to that disease, and then apply this to the specific claimant in the context of his or her exposure history, background risks etc.

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<sup>765</sup> *R v T* (n 13) at [96]-[99]; [104]-[105]

<sup>766</sup> *Redmayne et al* (n 117) 354-355

Where scientific evidence can inform a question, but is not sufficient of itself to answer the specific question, an expert can then provide an opinion about what may or may not have caused the disease. But this is no more than an opinion that is based on some evidence.

Cooke<sup>767</sup> points out, as cited in chapter 2, that used judiciously, expert opinion can be very valuable. Further, because interest, biases and uncertainty play a larger role in science than many philosophers would care to acknowledge, expert *opinion* has become an increasingly visible source of justification in science.<sup>768</sup> ‘There can be no doubt that experts know a great deal about topics on which ordinary people lack information’ note Meadow and Sunstein.<sup>769</sup> But experts, like anyone else, are subject to biases. Their judgments about risk are affected by the same heuristics and biases to which most people are subject, even if (and this is a disputed question) expertise tends to reduce the most serious errors.<sup>770</sup>

Further, courts can be misled by the eminence of an expert into believing that ‘scientific’ evidence all belongs to one related category, and that sound qualifications in one scientific discipline gives an expert the ability to testify questions from other areas of ‘science’, even if they are not from within their area of expertise (Chapter 2). Judgments about the admissibility of the expert’s testimony must not be guided by generic principles based on their stature in

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<sup>767</sup> RM Cooke, *Experts in Uncertainty: Opinion and Subjective Probability in Science* (Oxford University Press, 1991) 3

<sup>768</sup> *ibid* 18

<sup>769</sup> Meadow and Sunstein (n 111) 630

<sup>770</sup> *ibid* (n 111) 631

their own field: it is crucial that each discipline, and each expert, must be considered on their merits on a case-by-case basis.<sup>771</sup>

### **3.4: More transparency and better understanding of legal issues on part of scientific expert witnesses**

Does the fault for the often-controversial encounters between law and science lie exclusively with courts, queries Jasanoff, or do our own expectations concerning science and technology need to be modulated in the light of what we know about adjudication and the nature of scientific inquiry?<sup>772</sup>

Better use of scientific evidence in the law requires change not only on part of lawyers, but also on part of scientific expert witnesses. While lawyers must guard against the temptation to 'push' experts into answering questions that they cannot validly answer, it is also crucial for experts, on their part, to resist the temptation to present their opinions and speculations as scientific evidence. Expert witnesses must be more transparent about the methodological, statistical and epistemic limitations of their testimony than many have so far tended to. Unreliable expert evidence can put justice in jeopardy. A person called upon to give expert testimony must consider whether they are sufficiently schooled, skilled and knowledgeable enough to fulfill that role.<sup>773</sup> Sound expert testimony

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<sup>771</sup> Adam Wilson, 'Expert Opinion Evidence: the Middle Way' (2009) 73(5) *Journal of Criminal Law* 430, 430

<sup>772</sup> S Jasanoff, *Science at the Bar: Law, Science and Technology in America* (Harvard University Press 1997) 1-2

<sup>773</sup> GM Davies and AR Beech, *Forensic Psychology: Crime, Justice, Law, Interventions*, (2<sup>nd</sup> ed British Psychological Society/Wiley 2012) 299

will undoubtedly help achieve fairer results, but it is important for experts to be mindful that such witnessing must be ethical.

Unfortunately, there are many instances where the practice of expert witnesses has fallen short of what could be described as ethical. In a landmark report on the law-science relationship, a committee of the US National Academy of Sciences (NAS) drew attention to questionable practices and the lack of research in many areas of forensic science.<sup>774</sup> The committee was surprised to discover that many forensic science disciplines are typically not supported by scientific research and that analysts are not necessarily bound by experimentally derived standards to ensure the evidence offered in courts is valid and reliable. The report also noted a tendency on part of scientists to appear somewhat “leery” of lawyers and the legal process, while lawyers often expressed feeling frustrated by a scientific community that ‘believes that its methods and procedures are above legal scrutiny and questioning.’<sup>775</sup>

*R v T*, despite going too far by imposing overarching barriers against useful communication tools such as normal scientific terminology, likelihood ratios etc., must also be lauded for recognizing and attempting to rectify some of the problems in the legal use of science. The Court of Appeal insisted that experts were required to note in their report whenever they expressed any views that were subjective and based on their experience.<sup>776</sup> Further, the Court was clear that report writing should be transparent, and pre-trial safeguards should be

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<sup>774</sup> Cited from G Edmond, ‘A Guide to Interpreting Forensic Testimony: Scientific Approaches to Fingerprint Evidence’ (2014) 13, *Law Probability and Risk* 1, 1

<sup>775</sup> *Convergence of Science and the Law* (n 100) 2

<sup>776</sup> *R v T* (n 13) at [96]

used to ensure that only sound and credible data is adduced before juries.<sup>777</sup> All of these safeguards can also further help courts to recognise and reduce expert malpractice e.g. case management can help identify an expert's previous tendency to exceed his expertise,<sup>778</sup> and thus can help to weed out experts whose practice is below the required standard of competence or ethics.

#### **SECTION 4: The way forward: A more constructive role for science and epidemiology in UK law**

William Blackstone<sup>779</sup> exhorted that the law well practiced requires an insight into its fundamental ideals and purposes of justice: a need that becomes more acute especially when the law is brought into new areas, note Spicker et al. As Spicker et al point out: 'law and public policy must deal with notions of causal relations that establish lines of responsibility and accountability. This calls into play examination of philosophical and epistemological issues. Epistemological questions assume practical importance at the interface of law and medicine, where it often appears that law and medicine are making appeals to quite different notions of causality or causation.'<sup>780</sup> This thesis has strongly argued that the legal and medical notions of causation are now so widely divergent that it is impossible to bring any coherence to the fraught area of disease causation within existing models. Coherence in the approach to causation, and to the entire

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<sup>777</sup> *ibid* [97]- [99]

<sup>778</sup> Wilson (n 10) 527

<sup>779</sup> W Blackstone, *On the Study of the Law*, quoted from SF Spicker, JM Healey and HT Engelhardt (eds.) *The Law-Medicine Relation: A Philosophical Exploration* (D Reidel 1981) xiii-xxvii

<sup>780</sup> *ibid* xiii-xxvii

area of disease litigation, urgently requires effort to bring about greater compatibility and integration between legal and medical models of causation.

The above discussions have tried to illustrate that in the drawing of scientific inferences, rigid rules and criteria that govern what scientific evidence is to be accepted and what is to be rejected will inevitably lead to errors if applied without being evaluated in context. Thus, it is as fallacious to outrightly reject epidemiological evidence in specific causation disputes (as UK courts have sometimes done and as Wright urges both them and his native US counterparts to do<sup>781</sup>); as it is to go to the opposite extreme and insist that epidemiological evidence *must* be adduced by the claimant in toxic tort disputes (as some courts in the US have occasionally done).<sup>782</sup> For the same reasons, it is as erroneous to impose a rigid threshold requirement that an  $RR > 2$  is essential to infer causation from epidemiological data (as discussed above section 2.3); as to believe that there are 'hierarchies of evidence' in which experimentation and deductive inferences are *always* superior to observation or inductive inferences (above, section 2.1). All of these notions are erroneous if they are applied as hard-and-fast rules (as the judiciary currently tends to do).

#### ***4.1: Better recognition of the value of epidemiology***

Epidemiology, particularly clinical epidemiology, deals with decision making under uncertainty in a systematic way. The Bayes theorem, for example,

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<sup>781</sup> E.g. Wright, 'Bramble Bush...' (n 82)

<sup>782</sup> L Lasagna and SR Shulman, 'Bendectin and the Language of Causation in KR Foster, DE Bernstein and PW Huber (eds), *Phantom Risk: Scientific Inference and the Law* (Cambridge, Mass, MIT Press, 1993) 110-111

provides a technique provides a method by which prior knowledge of the probabilities (i.e., 'generalised' evidence) can be used, in combination with any additional specific evidence, to arrive at 'particularistic' individual diagnostic and treatment decisions. Many commentators (both lawyers and statisticians) argue that the Bayes theorem has great potential utility for toxic tort claims, because it can help to 'particularise' generalised statistical evidence for causation.<sup>783</sup> The aims and methods of epidemiology must be understood within the context of the general goals of science, as Weed explains: "The aim of science is to explain the world, to gain an understanding of it, to generate a form of knowledge, not certain nor proven, but well-evidenced, carefully considered in an open forum by a rather specialized community of practitioners, and subject to public scrutiny."<sup>784</sup> Morabio<sup>785</sup> emphasises that: "When proof is not available, pragmatic epidemiologists simply acknowledge that there is no alternative to the causal criteria logic that... before inferring causation it is imperative to check for illogicalities and rule out gross contradictions between what has been found and what we think we know."<sup>786</sup>

The cause and effect relationship in epidemiologic studies is determined through induction, inferred for the general population based on findings at the level of the individual, and then predicted back to new individuals through deductions

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<sup>783</sup> See e.g. CGG Aitken and F Taroni, 'Fundamentals of Statistical Evidence: A Primer for Legal Professionals' (2008) 12 *International Journal of Evidence and Proof* 181, 203-204; 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, 2011) 162-163

<sup>784</sup> DL Weed (2006), 'Commentary: Rethinking Epidemiology' 35 *International Journal of Epidemiology* 583, 586

<sup>785</sup> A Morabio, 'On the Origin of Hill's Causal Criteria' (1991) 2 *Epidemiology* 367, 367-369

<sup>786</sup> Cited from Feldschreiber, Mulcahy and Day (n 86) 181-182.



from the population level.<sup>787</sup> It is a method that is neither perfect nor without its limitations, but this pragmatic approach has answered a plethora of questions about the causes of disease, and revolutionised healthcare in recent decades: for individuals, as much as for ‘the public’. If the ultimate aim is to provide a complete causal account of disease and know why one person gets a disease while another does not, then success is unlikely, argue Coggon and Martyn.<sup>788</sup> The stochastic nature of disease, discussed earlier in Chapter 3 (section 2) results in the fact that whether or not someone develops a disease depends on combinations of events at a molecular or cellular level that science cannot yet measure.<sup>789</sup> At best, epidemiologists might hope to obtain clues to causation. Nonetheless, notes Weed about epidemiology: “We are not stuck, it seems to me, behind thick wooden doors in some medieval ivory tower, hunched over bubbling potions in an alchemist’s hideaway, futilely experimenting with one substance after another. We have actually found what we were looking for, preventable causes of diseases, some with strong effects others more modest in their effects. Our critics have done us a disservice by not including a table of accepted risks (some manipulable, some not) for the so-called complex diseases. It would be a very large table indeed. Epidemiology has made (and will continue to make) many discoveries.”<sup>790</sup>

That UK law would choose to dismiss such evidence on the grounds of its inability to give certainty about specific causation (despite the short supply of

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<sup>787</sup> AV Buchanan, KM Weiss and SM Fullerton, ‘Dissecting Complex Disease: The Quest for the Philosopher’s Stone?’ 35 *International Journal of Epidemiology* 562, 567

<sup>788</sup> D Coggon, C Martyn, ‘The Stochastic Nature of Disease Causation.’ (2005) 365 *Lancet* 1434, 1434

<sup>789</sup> Coggon (n 15) 582

<sup>790</sup> Weed (n 140) 583-584

alternative sources of evidence about this question) is difficult to justify. The methods used by epidemiology are not perfect (what in science is?), and some poor-quality epidemiologic studies can be seriously flawed. Assessing the problems with the epidemiologic evidence about the linkages between the drug Bendectin and congenital anomalies, Lasagna and Shulman outlined the difficulties that can affect the validity of epidemiological data. These included recall bias (i.e. mothers of deformed babies were more likely to remember correctly the use of drugs during pregnancy), the fact that it is easy to miss modest increases in rates of congenital anomalies unless very large numbers of subjects are studied, and the fact that repeated dredging up of epidemiologic data will predictably turn up spurious correlations by chance.<sup>791</sup>

As Gold puts it: ‘Advancing scientific understanding can assist in legal fact-finding even if science will not provide law’s longed-for, conclusive post hoc answer to the question of what did make a particular plaintiff sick. But the law must understand how science can best contribute. This understanding begins with acceptance of the fact that bringing toxicological understanding to the molecular level will not bring causation to the individual level. Even toxicogenomics and molecular epidemiology produce data that ultimately are group-based, statistical, and probabilistic...Thus, finding that a plaintiff does or does not have a genetic susceptibility to the disease-causing effect of a substance to which the plaintiff was exposed will provide probabilistic but not deterministic evidence of causation or its absence.’<sup>792</sup> Although Lord Nimmo-

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<sup>791</sup> Lasagna and Shulman (n 138) 102-103

<sup>792</sup> SC Gold, ‘When Certainty Dissolves Into Probability’ (2013) 70 Washington and Lee Law Review 237, 276

Smith opined that there is a "fallacy of applying statistical probability to individual causation",<sup>793</sup> Tavares points out that there are situations in which epidemiological evidence can be sufficiently honed to provide conclusions in individual cases, and that combined with clinical experience may well be determinative.<sup>794</sup> Given the high legal regard the law has for medicine, it may be useful for lawyers to understand how the medical profession manages evidentiary uncertainty, and the extent to which evidence-based medicine (EBM) is founded on the application of inconclusive population evidence to individual cases, as the section below explores.

#### ***4.2: Lessons from evidence-based medicine: the pragmatic use of population evidence in medical decision-making***

As we discussed earlier in Chapter 1, UK courts appear to accord greater weight to testimony from doctors rather than epidemiologists, when assessing causation, and medical evidence seems much less subject to judicial scrutiny or criticism. This is ironical, because much of the testimony medical experts provide about a specific claim is often itself based either on epidemiological or population studies, or on subjective opinion (since, as we have noted earlier, there is often no specific evidence about what caused a disease: a tumour caused by a toxin looks no different to a tumour caused by genetic factors). Thus, 'particularistic' evidence such as Wright seeks may not only be elusive for a very long time to come in disease litigation, but where adduced, may often contain

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<sup>793</sup> *McTear* (n 32) at [6.184]

<sup>794</sup> N Tavares, 'Case Comment: *Young v AIG Europe Ltd*' (2016) 1 *Journal of Personal Injury Law* C 34, 36

substantial elements of subjectivity and room for error (arguably, sometimes even more *more* than epidemiological testimony). This does not, however, make medical decision-making invalid: clinicians simply follow the pragmatic principle of using the best evidence they have.

This thesis attempts only to make a plea for is for the law to recognise these issues, so that evidence can be weighed appropriately. Medicine, as doctors will themselves usually be first to point out, is an uncertain pursuit.<sup>795</sup> In the course of their professional training it is vital for physicians to learn how to deal with uncertainty in their diagnostic and treatment decisions. The attribution of causality in medical science follows the same principle, and as Feldschreiber, Mulcahy and Day note, is 'both an art and a science.'<sup>796</sup> A common medical aphorism often repeated to medical students exhorts: "When you hear hoof beats on the cobblestones of London, think of horses, not zebras."<sup>797</sup> That is, in conditions of uncertainty, clinicians are encouraged to base their decision-making on the best evidence suggested by the general population, or on the most likely hypotheses. A medical textbook explains the probabilistic reasoning that lies at the heart of the Zebra Rule: "As you find abnormalities, you will need to decide the likely diagnostic possibilities so that you can search for clues to support or refute your differential diagnosis. Remember, common things are common so...think about the most frequent possibilities first when formulating

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<sup>795</sup> G Cooke, 'A is for Aphorism' (2012) 41 *Skin Cancer* 534, 534

<sup>796</sup> Feldschreiber et al (n 86) 179

<sup>797</sup> It is difficult to ascertain clearly source of this common medical aphorism that is frequently found in many medical textbooks it is attributed to Sir William Osler by KP Ryan, *When Tumour is the Rumour and Cancer is the Answer* (Author House 2014) 297. Regardless, it is generally accepted as a core principle of medical decision-making.

your differential (hence the Zebra Rule above).<sup>798</sup> To most medical practitioners, the assertion that population studies tell us *nothing* about an individual case would be an absurd one, as they routinely rely on population studies to aid individual clinical decision-making. Inferences about specific causation often require extrapolation from the evidence, using strategies such as inference to the best explanation. Courts that are unaware of this fact may tend to be overly impressed by ‘specific’ rather than ‘general’ evidence.

Medicine is highly reliant on ‘population’ epidemiological evidence, and epidemiologists are highly trained to respond to the many multidisciplinary issues that must be considered in this general assessment of disease causation, in addition to statistics. Notes McIvor: ‘While statistics are certainly an important tool used by epidemiologists, they are no more than a tool... Epidemiologists are trained in statistics, research methodologies, and also in medicine. The highly specialised techniques that they use for drawing causal inferences from empirical data are informed by all three disciplines.’<sup>799</sup>

Evidence based medicine (EBM) is generally defined as ‘the conscientious, explicit and judicious use of the current best evidence in making decisions about the care of individual patients’, and rests on the integration of clinical expertise and the best external evidence.<sup>800</sup> This decision-making process, as professor of primary healthcare Dr. Trish Greenlagh observes, heavily involves the use of mathematics (or what lawyers tend to refer to as statistics): “EBM is the use of

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<sup>798</sup> NJ Talley, S O’Connor, *Pocket Clinical Examination* (2<sup>nd</sup>ed Elsevier 2004) 23

<sup>799</sup> McIvor, ‘Debunking...’ (n 34) 570

<sup>800</sup> DL Sackett, WM Rosenberg et al, ‘Evidence-Based Medicine: What It Is and What It Isn’t’ (1996) 312 *British Medical Journal* 71, 71

mathematical estimates of the risk of benefit and harm, derived from high-quality research on *population samples*, to inform clinical decision-making in the diagnosis, investigation or management of *individual patients*.”<sup>801</sup> (Emphasis is added). Thus, stresses Professor Greenlagh, ‘If you follow an evidence-based approach to clinical decision-making, therefore, all sorts of issues relating to your patients... will prompt you to ask questions about scientific evidence, seek answers to those questions in a systematic way, and alter your practices accordingly.’

Justice Robert Jay points out that it is vital to recognise that scientific decision-making, just like legal decision-making, involves exercising judgment and extrapolating from existing data: “...in so many cases a medical expert will be expressing an opinion in the individual case on the basis of clinical judgment. That, no doubt, may be a compelling amalgam of observation and experience, but it certainly does not entail the recruitment of the claimant to any randomised controlled trial. In the same way perhaps as the good judge, the good doctor is applying intuition honed by experience to a particular evidential matrix and conjuring a common-sense conclusion out of the mix. Outside the courtroom, that good doctor will be expressing himself or herself in terms of degrees of confidence (or lack of it); inside the courtroom, those possibilities are ironed out and become transformed from an expert expression of probability into a judicial expression, once the judgment is delivered, of synthetic certainty.”<sup>802</sup> As Miller points out: ‘Science, however complex it might appear, is a... pragmatic activity –

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<sup>801</sup> T Greenlagh, *How To Read A Paper: The Basics of Evidence- Based Medicine* 5<sup>th</sup> ed. (Wiley/BMJ Books 2014) 1

<sup>802</sup> Jay (n 35) 3

a theory is only as good as the rigour of the last (unsuccessful) attempt to refute it. And no branch of science is more deliberately pragmatic than that concerned with the cause and distribution of disease, viz epidemiology... Politicians are not philosophers; they cannot wait for certainty before, for example, banning a drug which appears to have a harmful side-effect... Epidemiologists can assist both politicians and lawyers by devising criteria that indicate when the inductive leap – from statistical association to causation – might be justified.<sup>803</sup> Just like politicians (and doctors), the law does not have the luxury of being able to wait until certain and perfect evidence appears out of scientific journals, or to operate on the belief that intuitive conclusions about factual matters is a better substitute until that happens. As Miller notes elsewhere, ‘civil courts cannot commission laboratory experiments or epidemiological studies, but nor can they suspend a case until someone else does.’<sup>804</sup> In any case, as the Bayesian philosophers of science point out, even though perfect, ultimate truth may exist, it is usually impossible to know when we have found it.<sup>805</sup>

Rejecting useful, reliable evidence from a discipline that has proved its value by its many achievements in healthcare is an illogical approach. Tavares notes that it was the impossibility of applying epidemiological studies to determine causation in individual cases which was cited as the principal reason the tobacco litigation failed, but the law needs to learn to apply the best available evidence

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<sup>803</sup> Miller, ‘Causation in Personal Injury’ (n 59) 547

<sup>804</sup> C Miller, ‘Coal Dust, Causation and Common Sense’ (2000) 63 *Modern Law Review* 763, 769

<sup>805</sup> Rothman et al (n 39) 26-30

(even where this is overtly probabilistic) to aid factual questions, for: 'if only everything could be reliably determined on "common sense"!'<sup>806</sup>

Probabilistic epidemiological methods can be refined and combined with other evidence to formulate robust and justifiable inferences of causation. Where 'gold standard' methodologies are impossible to implement, alternative research methodologies such as observation can offer valuable- and robust- alternatives, even if they require further corroboration through larger sample sizes, better statistical techniques and more replication before findings can be accepted as valid. The Law Commission Report emphasises the importance of improving applications of scientific evidence in UK law. However, as Heffernan and Coen<sup>807</sup> note: 'The success of the Law Commission's proposals turns in no small measure on the education and training of judges and lawyers in forensic science. The Consultation Paper recognises the benefits that would flow from judges and criminal practitioners receiving practical training on the methodology of science...and how to determine the reliability of experience-based expertise,<sup>808</sup> but stops short of making a formal recommendation in this regard.' Lasagna and Shulman recommend that an expert panel may help the court to subject the validity of all scientific evidence to standardised critical evaluation, rather than leaving it to juries to correctly weigh and contextualize scientific testimony may be useful.<sup>809</sup> Despite age-old controversies about the

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<sup>806</sup> N Tavares, 'Case Comment: *Young v AIG Europe Ltd*' (2016) 1 Journal of Personal Injury Law C 34, C36

<sup>807</sup> L Heffernan and M Coen, 'The Reliability of Expert Evidence' (2009) 73 Journal of Criminal Law 488, 506

<sup>808</sup> Law Commission, *The Admissibility of Expert Evidence in Criminal Proceedings in England and Wales: A New Approach to the Determination of Evidentiary Reliability*, Consultation Paper No. 190 (April 2009) at para. 6.72

<sup>809</sup> Lasagna and Shulman (n 138) 116



epistemological foundations of science, and the endless debates regarding the law-science interface, confidence in scientific method is not misplaced.<sup>810</sup> The legal approaches towards causation of have enormous practical implications for vast numbers of people (claimants and defendants) involved in toxic tort litigation demands that courts use all available evidence to arrive at the fairest and most informed solutions possible, despite the inherent uncertainties around disease causation. This cannot be possible without due consideration of scientific and epidemiological evidence wherever available. Perfect answers may be elusive, but this does not justify us therefore turning our backs on the best answer that is available. The philosopher Simon Blackburn points out, ‘...there may be rhetoric about the socially constructed nature of Western science, but wherever it matters, there is no alternative’.<sup>811</sup>

The eminent epidemiologist Sir Austin Bradford Hill noted in 1955:

‘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, or to postpone the action that it appears to demand at a given time.’<sup>812</sup> UK tort law cannot justify continuing to dismiss an evidence-based approach to decision-making even six decades later, in a futile longing for perfect proof.

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<sup>810</sup> Klinkner (n 1) 129

<sup>811</sup> S Blackburn, *Truth: A Guide for the Perplexed* (Penguin 2006) 196

<sup>812</sup> AB Hill, ‘The Environment and Disease: Association or Causation’ (1965) 58 Proceedings of the Royal Society of Medicine 295, 300

## CONCLUSIONS

This thesis has aimed to advocate reform in the UK judicial approach to epidemiological and scientific evidence in claims for negligently caused diseases. It has outlined the issues that hinder the effective application of such evidence in UK tort law, and has contended that poor legal understanding about scientific reasoning as a whole, as well as lack of awareness about the complexities of disease causation, lie at the heart of the problematic approach towards epidemiological evidence. The thesis first outlined the problematic beliefs about the discipline that hinder its effective application in the law. In order to show why these judicial beliefs are unjustified, the thesis then addressed the wider legal misconceptions about science, and the mismatch between legal and medical views of causation. After clarifying the fundamentally probabilistic nature of scientific reasoning (and how the law has misunderstood it); as well as the complexities of disease causation (and why the existing confused and deterministic legal principles of causation cannot deal effectively with these), the thesis then argued for a more probabilistic and scientifically informed approach to causation in complex disease litigation. It also made a case for the crucial role for evidence in the factual enquiry.

Despite many judicial attempts to resort to normative and subjective approaches to factual causation, it is becoming increasingly obvious to lawyers that it is impossible to escape the need for scientific evidence in disease litigation. However, this thesis has argued that such evidence cannot be appropriately applied or utilised unless the law reevaluates outdated legal approaches to

causation, and to science in general. The thesis highlighted the legal factors that hinder the ability to use such evidence coherently, and concluded by making suggestions for a better use of scientific, particularly epidemiological, evidence in the law.

Chapter 1 introduced epidemiology and the UK legal approach to epidemiological evidence. It evaluated some strengths and limitations of the discipline, and then outlined the judicial view of such evidence, as indicated by some key judgments. This discussion highlighted the concerns that courts have about the alleged epistemic deficiencies of epidemiology, including its reliance on population data, statistical tools of analysis and observational methodologies. It also highlighted substantive errors around the importance of measures such as relative risk thresholds.

Chapter 2 highlighted *the* probabilistic nature of science and the scientific method. It explored the deeply erroneous view that the legal profession has of the ideal science as being one that possesses the quality of certainty, and about ideal scientific testimony as being one that provides conclusive proof to the legal inquiry. This view is not only fundamentally erroneous, but is dangerous, as it has occasionally led to erroneous legal outcomes due to wrong applications of scientific evidence. Criticisms of epidemiological evidence on such grounds reflects a mistaken judicial belief that probabilistic reasoning is unique to epidemiology as a scientific discipline, and the corollary that other sciences are capable of providing certainty. In fact, this chapter illustrated, scientific evidence is almost always probabilistic in nature, and very rarely supplies definitive

certainties: now more so than ever before. Discoveries of the twentieth century, particularly in the world of physics, have shaken many of the older, deterministic conceptualisations of science fundamentally. Even physics, traditionally that most deterministic of sciences, is now being fundamentally challenged by realisation of insurmountable uncertainties. Our ability to know and predict things, it appears, is much more limited than once thought. All sciences now seem to be converging towards an acceptance of their limited knowledge. The chapter contended that lawyers have failed to appreciate this, which has led to unrealistic expectations about scientific evidence, and inflated perceptions of its probative value. These unrealistic expectations can quickly turn to disenchantment when the legal longing for certainty is not fulfilled, or when excessive deference to scientific testimony leads to wrong legal outcomes. Courts, this thesis contended, consequently go to the other extreme and dismiss probabilistic scientific evidence as having no meaningful role to play in the factual enquiry. The sceptical judicial in UK courts attitude to epidemiology is an example of this.

Chapter 3 highlighted the complexities of disease causation, and the growing mismatch between legal and medical approaches towards disease. *It* argued that confused principles of causation in tort law, and an excessive infusion of normative considerations into the factual enquiry, are a further barrier to an objective and evidence-based assessment of factual causation. This normative approach is even more likely to be resorted to in disease claims because of evidentiary uncertainties that make legal tests for causation often impossible to satisfy. The determinism of the 'but for' test, and its emphasis on 'necessity' of

the causal factor in bringing about the harm, is particularly unsuited to assessing disease causation. This is due to the very complex, multifactorial and stochastic nature of disease, making this one of the most uncertain areas of science. The chapter explored current biomedical evidence about disease, in order to highlight the increasingly probabilistic medical conceptualisations of disease. This chapter argued strongly for greater legal coherence with scientific and medical views of causation. It highlighted the urgent need for legal approaches that can better align with probabilistic empirical evidence.

Chapter 4 outlined the 'but for' test and the alternative exceptional tests that have been applied to the factual causation assessment. It discussed why the 'but for' test proves inadequate so often in complex disease claims, which has led to the development of a number of somewhat haphazard, piecemeal exceptional tests for causation. There is currently little clarity about when these exceptional tests will apply. The chapter highlighted that the exceptional tests are now being widened to many different disease scenarios, and contended that this is unsurprising, since all diseases are characterised by fundamental causal uncertainties. Overdetermination, this chapter argued, may be the norm rather than the exception in disease litigation. Simpler, less rigid tests for factual causation, that take due account of probabilistic causation, would be a much better fit in this area of the law than rigid, necessity-based tests such as the but-for test. The more principled solution, this chapter proposed, would be to replace the 'but for' test in complex disease claims with a more practical, flexible, probabilistic test for causation which assesses the *contribution* of a tortious factor to the disease, on the balance of probabilities, rather than its *necessity*.

This should be applied on a proportionate liability basis. This would also allow for appropriate and realistic assessment of scientific, medical and epidemiological evidence in disease litigation.

Chapter 5 then returned to epidemiology, and re-examined the judicial objections to epidemiology in the light of all the preceding discussions about science and about the complexities of disease. This chapter also suggested some general strategies for improving the use of scientific evidence in the law. *It is imperative for UK courts to recognise that science very rarely supplies definitive certainties.* Scientific research methods inevitably possess methodological limitations and flaws, which scientists refer to as their error rates. Good scientific research clearly defines the potential for error or confounding variables in the research, and these error rates must be taken into consideration when interpreting research evidence for any purpose (including legal purposes)<sup>813</sup>. Thus, experts who provide their testimony in qualified and tentative terms, and are transparent about their potential for error, may often be providing more scientifically credible testimony than experts who make excessively confident and sweeping assertions.

Uncertainty about one's conclusions, this thesis tried to contend, does not imply that the conclusions are baseless or unsound. If that were so, then almost all human scientific knowledge would be worthless. The fundamental quality that distinguishes reliable from unreliable inferences is that reliable inferences are

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<sup>813</sup> D Faigman, 'Judges as "Amateur Scientists"' (2006) 86 Boston University Law Review 1207, 1220-1225

arrived at only after all relevant *available* evidence (whether supportive or contrary to our hypothesis) has been objectively evaluated and considered. Legal decision-making must be informed by all available relevant evidence that exists (especially in this area of the law, which is deeply challenged by evidentiary gaps and confusion). The central argument this thesis has made is that especially when it comes to disease causation, probabilistic evidence is the best that science currently has to offer, and epidemiology is the scientific specialism that can provide the best probabilistic evidence about the determinants and causes of disease.

Epidemiological research, for all its methodological caveats, has made an invaluable contribution to our knowledge about the causes of disease, and through illuminating questions of causation, has led to enormous improvements in public health. There is no justification for the law to ignore such evidence where it is available. Neither is it justified to believe that it is better to assess factual issues entirely on the basis of subjective values or personal preference, just because the available evidence is not conclusive. This thesis has argued that used judiciously, epidemiology can help develop a fairer and clearer legal approach in this challenging and complex area of the law. The uncertainties and imperfections of epidemiologic evidence may find a more honest fit in a system that reflects the subtleties of causation, noted the American authors Lasagna and Shulman.<sup>814</sup> This thesis has attempted to make the plea that it is now imperative for the UK tort system to do so.

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<sup>814</sup> L Lasagna and SR Shulman, 'Bendectin and the Language of Causation in KR Foster, DE Bernstein and PW Huber (eds), *Phantom Risk: Scientific Inference and the Law* (MIT Press 1993)

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