CAUSATION: THE INTERFACE BETWEEN THE SCIENTIFIC AND LEGAL METHODS

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The mechanism of causation is used to attribute legal responsibility. Both factual and normative legal questions are involved. As to factual causation, complex forensic issues may arise. And it is here where science and the law intersect. Now their methods are different, but such differences must be appreciated in order to adequately assess scientific evidence. How does one engage with such evidence? What type of scientific evidence is relevant to causation? Are there new frontiers in science that have produced new causation questions in the law? This article provides a trial judge's perspective on some of these questions.

I INTRODUCTION

My purpose is to discuss the interface between the scientific method and the legal method concerning the causal connections between facts. But in order to address that topic the broader contextual differences between science and the law concerning their objectives and methodologies need first to be appreciated. These can conveniently be described in eight overlapping ways.

First, the objectives of legal and scientific inquiry differ. Science searches for increased knowledge, with truth as its ideal; its coverage is more comprehensive and not time sensitive. But in the legal context, the law's objective is to resolve conflicts; its coverage is limited and time sensitive.

Second, in expounding legal principle, the law is concerned with regulating human affairs in accordance with values and objectives. It deals with what ought to be. But as Korn explains,[1] science is descriptive of the natural world. Scientific laws do not control or judge. Rather they describe, explain and predict. But at the

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[1] Harold L Korn, 'Law, Fact, and Science in the Courts' (1966) 66(6) *Columbia Law Review* 1080, 1093–4.

general level of their respective theories, each discipline provides a body of 'generalized, systematized, and transmissible knowledge', [2] although their inductive processes differ.

Third, scientific theories are always at risk of being falsified. So much has been apparent since the metaphysics of medieval scholasticism was replaced by the Galilean method of natural philosophy via the agency of Italian humanism; this culminated in the Baconian method of interrogating natural phenomena. Scientific theories are contestable, revisable and consequently always tentative.[3] Contrastingly, legal principles are at risk of becoming irrelevant or re-interpreted, rather than formally falsified. Irrelevance may be produced in many ways. For example, the policy underpinning a principle may have changed or disappeared. More strictly, the principle may have fallen if it depended upon another rule which itself has fallen. But the doctrine of precedent provides a constructed constraint, a constraint alien to science which is not concerned with normative standards and practical temporal constraints on their alteration.

Fourth, in scientific theory development the facts relevant to an old theory ought not to contradict the new theory. Moreover, the new theory ought also to at least explain all the facts explained by the old theory. But for the development of a new legal principle, the facts of an older case are at most of secondary significance, and then only to understand the foundation of the principle to be superseded or modified.

Fifth, legal reasoning uses examples and a fluid if not contestable[4] notion of analogy to support a principle or its incremental development. But falsification of an example or an analogy does not of itself negate the principle. But in scientific reasoning, theory is not developed from ad hoc examples or limited analogies, although analogical reasoning does have some place such as Bohr's model of the atom. A scientific hypothesis is usually constructed by induction

[2] Ibid 1101.

[3] Foster and Huber have stated that 'the ultimate test of the 'validity' of a theory or of some data is time'. Science is cumulative and self correcting: Kenneth R Foster and Peter W Huber, *Judging Science: Scientific Knowledge and the Federal Courts* (MIT Press, Cambridge, 1997) 17.

[4] I say contestable for the reasons partly explained in Shivprasad Swaminathan, 'Analogy reversed' (2021) 80(2) *Cambridge Law Journal* 366. How do you decide whether the source analogue is similar? Is that choice made by little more than an intuitive notion of similarity? And is true analogical reasoning what a judge does? It might be said that some judges reach a firm view on the result first and then use analogical reasoning as a rhetorical device.

from numerous identical or similar observations, although any judgment as to similarity may require reasoning by analogy, but only in a strict sense. Moreover, one falsified deducible consequence negates the theory.

Sixth, forensic analysis in litigation proceeds by way of a dialectical process with the parties' pursuit of different epistemic objectives.[5] And the analysis takes place within the idiosyncratic constraint of how the issue is formulated and evidence is adduced. Contrastingly, participants in scientific analysis usually pursue, relative to other fields of human inquiry, a more common epistemic objective.[6] And the inquiry is not so issue or evidence constrained, although there may be practical or paradigm constraints for scientists conducting normal science within the bounds of their discipline.

Seventh, the scientific method of reasoning is, relatively speaking, more linear. Conveniently represented, it usually involves: *Observation → Hypothesis → Testing → Theory → Further confirmation or attempted falsification.* But this is simplistic for various reasons. A prior theory may have suggested the making of the initial observation. Further, the initial observation may only be interpreted in the light of prior theory. Further, a flash of insight divorced from an observation may have been the true starting point.

Moreover, there is no one scientific method. There are different scientific approaches to how one draws conclusions from observations. The physicist may only be satisfied if accurate predictions from a theory can be verified by critical confirmatory evidence. The epidemiologist may be satisfied by the accumulation and coherent organisation of sufficient observations. And indeed for some, controlled experiments are necessary; for others, this may not be possible or consistent with their methodology.

Further, there are differences even in the study of the same subject matter. Take the study of cancer.[7] The surgical oncologist treats the individual. The cancer biologist works with cells. The pathologist analyses and diagnoses the cancer. The epidemiologist gathers and analyses statistics to detect patterns in

[5] Perhaps to clothe such objectives with the label 'epistemic' confers an exaggerated respectability. After all, '... the object of the parties is always victory, not abstract truth': Sir Owen Dixon, 'Science and Judicial Proceedings' (Address, Medico-Legal Society of Victoria, 13 September 1933) 8.

[6] Susan Haack, 'Irreconcilable Differences? The Troubled Marriage of Science and Law' (2009) 72 *Law and Contemporary Problems* 1, 8, 9, 12.

[7] David Kriebel, 'How Much Evidence is Enough? – Conventions of Causal Inference' (2009) 72 *Law and Contemporary Problems* 121, 124.

population data. As a result, their methods and paradigms hypothesising on causation are different.[8]

More generally, across scientific communities there is considerable variability as to the standards by which evidence is considered to be stronger or weaker in determining the truth of a hypothesis. Such variability may be explained by location, the field of science, differences between qualitative and quantitative research and differences between normal science and work at the boundaries of a particular paradigm.

Eighth, at the forensic level in litigation, truth is static. Using for the moment the concept of truth loosely, it is whatever the judge rules it to be based upon the evidence which has been adduced and circumscribed by how the relevant issue has been identified and fought. Moreover, to so rule is an imperative imposed upon the judge. But it is not the imperative imposed upon the scientist. The judge must decide the case one way or the other. But the scientist has the privilege of a 'wait and see' approach. But then it is the privilege of the judge to be able to assert truth, which the scientist cannot. As has been pointed out:[9]

> In fact, it may be said that under the maxim *res judicata pro veritate accipitur* courts have an advantage over other seekers after truth. For by their judgment they can reduce to legal certainty questions to which no other conclusive answer can be given.

But there is a rider. If the burden of proof is satisfied by the party positing the scientific hypothesis, the judge can assert it as established even though the scientist would not. But if the burden is not satisfied, both the judge and the scientist would be in the same position. Neither would be asserting truth for the hypothesis,[10] although their reticence would have different but overlapping rationales.

Now I do not want to linger on the concept of truth, which I have used informally, let alone dwell in the philosophy of propositional truths involving the elucidation of correspondence or coherence theories. But I should make some

[8] Indeed, some might not even hypothesise on a cause. So, a medical specialist undertaking a differential diagnosis may only be seeking to identify the disease or condition rather than its underlying cause.

[9] *Bank of New South Wales v The Commonwealth* (1948) 76 CLR 1, 340 (Dixon J), approved in *Amaca Pty Ltd v Ellis* (2010) 240 CLR 111 ('*Amaca v Ellis*'), [6] and [37] with the maxim translated as 'a thing adjudicated is received as the truth'.

[10] *Adelaide Stevedoring Co Ltd v Forst* (1940) 64 CLR 538, 569 (Dixon J), approved in *Amaca Pty Ltd v Booth* (2011) 246 CLR 36 ('*Amaca v Booth*'), [69].

short observations. In the legal context, there are factual truths and legal truths as Haack points out.[11] A factual truth might engage with the question: did you drive through a red light? But strictly the legal process is not to discover independent objective truth, but rather whether the proposition has been established to the required standard. Further, evidentiary exclusionary rules based upon policy objectives that are not epistemological in terms of only being truth indicative may impact on the assessment. Further, a legal truth, for example, that one who intentionally and unlawfully kills commits murder, is socially constructed.[12] Contrastingly, as Haack explains it, scientists do not make scientific truths. Rather, 'it is not scientists' intellectual work, but the nature of the phenomena and events in the world that those claims and theories describe, that makes these scientific truths true.'[13]

Now accepting differences between the legal method and the scientific method does not deny that when lawyers analyse scientific evidence, the epistemic values that need to be applied correlate with values underpinning the scientific method. A lens must be used which provides a sophisticated picture of the content and reliability of the scientific evidence. And not to analyse scientific evidence through the appropriate lens can lead to outcomes 'determined by intuitive perceptions of the weight of authority rather than by reasoning from evidence'.[14]

Of course, the language, premises and analytical styles between the scientific method and the legal method have their differences. But when the latter is required to evaluate the former, significant correspondence of epistemic values arises. Lawyers must 'retrace and evaluate the technical analysts' logic, from empirical data to subjective judgment'.[15] And complex issues of science require reasoning among purely technical facts analysed through complex models, statistical inference and mathematical instruments that have no comparator in legal reasoning.

[11] Susan Haack, *Evidence Matters: Science, Proof and Truth in the Law* (Cambridge University Press, 2014) 305.
[12] Ibid.
[13] Susan Haack, 'Of Truth, in Science and in Law' (2008) 73(3) *Brooklyn Law Review* 985, 995.
[14] Joel Yellin, 'High Technology and the Courts: Nuclear Power and the Need for Institutional Reform' (1981) 94(3) *Harvard Law Review* 489, 491.
[15] Ibid 520. Yellin's context encompassed US judicial review of regulatory decision-making, but his comment applies *a fortiori* to direct factual adjudication between competing scientific evidence.

Now there are different modes for judicial analysis of scientific evidence.[16] The judge might hear expert evidence in the usual way or through the mechanism of concurrent evidence sessions, which is my preferred option in patent litigation.[17] The judge might appoint his own expert. The judge might obtain assistance from sitting with an assessor who may assist with any explanation of the scientific evidence. The judge might receive a report on the scientific question from a special referee, although this last option is remote from any direct judicial analysis of the scientific question. But whatever the procedural mode, this does not change the epistemic values and methods needed to assess scientific evidence. Moreover, judges are not obliged to accept the *ipse dixit* of the expert. They are obliged to test propositions, to assess one expert's opinion against another, and to synthesise a position taking into account all expert, lay and documentary evidence.

Let me now introduce the topic of causation. Scientific evidence concerning causation assessed within a legal framework can be divided into two categories. The first category can be described as the inquiry of historic cause. What was the cause of the disease? What was the cause of the explosion? The second category arises in regulatory litigation. It involves risk assessment and predicting likely consequences. Is this chemical a likely carcinogen or mutagen? How should it be classified and regulated?

These two categories provide a different perspective on causation. If one is considering researching and regulating a new drug, one is considering *ex ante* possible causal pathways and undertaking a form of risk assessment. One is seeking to predict likely effects. But in litigation concerning historic cause, a different inquiry is being undertaken. One is carrying out a back-solving forensic investigation. And indeed, such an after the event inquiry may take place where the risk is known or suspected by reason of epidemiology, an advantage that may not be available for the *ex ante* context. Let me say something further about this category which more usually arises in litigation.

[16] I have discussed the various options available to Australian judges at least in my paper 'The Use of Assessors in Class Actions' (2015) 129 *Precedent* 15.

[17] I do not propose to discuss conditions for the admissibility of such evidence and s 79 of the *Evidence Act 1995* (Cth) ('Evidence Act'), but will assume such admissibility. I am also not discussing the jury trial context.

There are many intersections between the scientific method and the legal method when one is dealing with factual causation concerning historic cause. And science can assist in addressing the following questions.

First, one has the question of what is a necessary condition for causation, in other words the 'but for' or *causa sine qua non* test, although such a test has its problematic properties. How do you deal with multiple sufficient causes? And what about where you have a factor that is neither a necessary nor sufficient condition for causation? And if a factor is neither a necessary nor sufficient condition, does it materially contribute to the injury or disease? So, as Stapleton explains, where you have an indivisible injury, the defendant's breach may have made a contribution to bringing it about but may have been neither necessary nor sufficient.[18] This may be contrasted with a divisible injury where the defendant's breach has made a positive contribution to part of the injury and the 'but for' test can be satisfied with respect to that part,[19] as distinct from the entirety of the injury.

Second, one has the factual distinction between what can be described as general causation and specific causation, although this is not a legally formalised distinction under Australian law. The former may pose the question of whether, for example, a suspect agent can cause adverse effects in humans. The latter may pose the question of whether such an agent in fact caused the plaintiff's injuries. This latter question may also give rise to other factual inquiries. Was the plaintiff exposed? Was such an exposure and consequent dose of a degree significant enough to cause his injury? Were there confounding factors that otherwise caused the injury?

Now I will avoid using the label 'common sense' in my discussion of causation. If all that is meant by this is countenancing an ability to infer facts from common experience, then such a label is unremarkable but irrelevant to my context which is dealing with science and therefore matters beyond common experience or lay intuition. And if one is applying the label to scope of liability questions or, as Stapleton described it, truncation questions,[20] then that context is also not

[18] Jane Stapleton, 'Unnecessary Causes' (2013) 129 *Law Quarterly Review* 39, 40, 43. An 'indivisible' injury is one 'where the relevant step in the mechanism by which the injury is known to occur requires a certain amount of an element, but does not require more and is not affected if there is more'.
[19] Ibid 52.
[20] Jane Stapleton, 'Factual Causation' (2010) 38 *Federal Law Review* 468.

relevant to my discussion. Conveniently, subject to later making a few brief points on normative questions, I will avoid being drawn into the conceptual miasma identified by Stapleton, [21] where factual causation has been inappropriately infused with normative principles and policy choices.[22]

I will return to causation later, but let me now discuss the scientific method more generally so that the scene for the later discussion is set. Without an appreciation of that method one has an insecure foundation on which to assess scientific evidence concerning causation.

II SCIENTIFIC THEORIES AND OBSERVATIONS

It is appropriate to make some observations concerning the tentative and falsifiable nature of any scientific theory, the sociological framework within which such theories are constructed and how scientific models and mathematical components may be used. I will also address the theory laden quality of observation.

A *The Tentative Nature of Any Scientific Theory*

Any scientific theory, no matter the degree of its acceptance, is necessarily tentative and revisable. That proposition is established by what defines science. Now the demarcation between science and other fields of human inquiry is contentious. But there is some common ground among lawyers, scientists and philosophers of science. The scientific method involves the positing of testable hypotheses based upon empirical observation. But any theory formed is only tentative. Any theory gives rise to the problem of under-determination. For any set of empirical observations, there is always more than one theory which can

[21] Ibid 468–472. It is also not necessary to discuss relevant provisions of the civil liability legislation in the various Australian States. I am not concerned with 'scope of liability' questions. And as for factual causation, in essence the 'but for' test as developed and applied by the common law has been enshrined.
[22] Indeed, as Stapleton explains (ibid 469), the notion of 'common sense' causation has also been inappropriately blended with proof of breach and foreseeable result. It has been said in some authorities that a common sense approach would allow that one could couple breach with an event of the kind that might thereby be caused, which may be enough to justify an inference, in the absence of evidence to the contrary, that the event did occur owing to the act constituting the breach. But as Stapleton explains, breach and foreseeability of result are different concepts to proof that the breach was a factual cause.

explain them or is empirically adequate.[23] All scientific theories are tentative and revisable, as the history of science demonstrates. Indeed, history provides support for the pessimistic meta-induction argument. The induction that all scientific theories today will be replaced by later truer theories is supported by what has occurred for all past theories. As each present or past theory is itself an induction, the global statement is a meta-induction, although the hypothesised meta-induction is not itself a scientific theory; otherwise the meta-induction would be self-contradictory. And as to this meta-induction, it has no temporal boundaries for the replacement of a particular theory, thereby permitting of a particular theory only being replaced over a long time horizon, and then perhaps only being replaced by a broader theory which reduces the superseded theory, say, Newton's inverse square law, to a special case of the successor theory, namely, Einstein's field equations underpinning general relativity.

The US Supreme Court in *Daubert v Merrell Dow*[24] considered what the scientific method involved in the context of the admissibility of expert evidence in litigation involving the effects of a prescription drug known as Bendectin / Debenox, which allegedly caused serious birth defects in children of mothers who had ingested the drug. The Court endorsed Popper's means to delineate the scientific status of a theory by the concept of falsifiability, although some of the scope of his insight seems to have been lost in translation.

Popper's propositions that contextualised and explained the falsifiability phrase cited by the Court were:[25]

1 It is easy to obtain confirmations, or verifications, for nearly every theory – if we look for confirmations.
2 Confirmations should count only if they are the result of *risky predictions;* that is to say, if, unenlightened by the theory in question, we should have expected an event which was incompatible with the theory – an event which would have refuted the theory.
3 Every 'good' scientific theory is a prohibition: it forbids certain things to happen. The more a theory forbids, the better it is.

[23] Some philosophers would say that there is an infinite set, but with a spectrum of plausibilities across the set.
[24] *Daubert v Merrell Dow*, 509 US 579 (1993).
[25] See Karl R Popper, *Conjectures and Refutations: The Growth of Scientific Knowledge* (Routledge, 5th ed, 1989) 37.

> 4 A theory which is not refutable by any conceivable event is non-scientific. Irrefutability is not a virtue of a theory (as people often think) but a vice.
>
> 5 Every genuine *test* of a theory is an attempt to falsify it, or to refute it. Testability is falsifiability; but there are degrees of testability: some theories are more testable, more exposed to refutation, than others; they take, as it were, greater risks.
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> 6 Confirming evidence should not count *except when it is the result of a genuine test of the theory*; and this means that it can be presented as a serious but unsuccessful attempt to falsify the theory ...
>
> 7 Some genuinely testable theories, when found to be false, are still upheld by their admirers – for example by introducing *ad hoc* some auxiliary assumption, or by re-interpreting the theory *ad hoc* in such a way that it escapes refutation. Such a procedure is always possible, but it rescues the theory from refutation only at the price of destroying, or at least lowering, its scientific status ...

One can sum up all this by saying that *the criterion of the scientific status of a theory is its falsifiability, or refutability, or testability.*[26]

But falsifiability at best is only a necessary rather than sufficient condition for the characterisation of an hypothesis as scientific. Moreover, it is an imperfect test. First, scientific theories are usually only testable in combination with the utilisation of auxiliary hypotheses, theories and a metaphysical framework. So, Newton's law, F = MA, is not testable without a metaphysical theory for two of the variables, a theory of testing and a theory of measurement. In other words, it is an idealisation to talk of a freestanding theory that you might attempt to falsify. Second, if an observation falsifies the deducible consequences of the combination, which component of the combination has been falsified? It may not be the theory being tested, but rather the auxiliary hypotheses, testing procedures or initial conditions. Third, falsifiability is not a sufficient test for demarcation of a scientific claim from a religious claim. Many claims in religion are in principle falsifiable, such as Noah's flood. Fourth, normal science and its development does not proceed in the linear manner suggested by Popper, as

[26] Ibid (emphasis in original).

Kuhn later explained.[27] Fifth, much of scientific practice relates to seeking confirmatory evidence rather than falsifying evidence. Sixth, in any event, many theories have been accepted even though they were apparently falsified. For example, Newton's inverse square law continued to be accepted even when it was apparently refuted by the orbit of Uranus. Later it was discovered that the description of the initial conditions was false, rather than the theory, because of the non-inclusion of Neptune's effect. Finally, there are many theories that you would consider to be scientific and accepted as such, but which have not been able or likely to be verified or falsified. String theory is a non-classical example. Many physicists have persisted with the mathematical elegance of string theory, and its numerous variants including membrane theory, despite there not being foreseeable realistic experiments available to either verify or falsify such theories.

It is useful to adopt one description given by Edmond and Mercer that recourse to Popper's philosophy of science is appropriate 'as a legal literary technology capable of assisting with strategic articulations of science (and non science)'.[28] Now not only is it useful for such general articulations. It is also useful when considering an individual hypothesis. Take the hypothesis that breast implants caused connective tissue disease. One perspective on the science was that such a hypothesis involved vague and shifting definitions of effect. When precise definitions of symptoms were used, but shown not to be linked to breast implants, either more vaguely defined or shifting symptoms were used, making falsifiability of the hypothesis difficult to say the least.[29] The insight provided by the falsifiability indicia justifies circumspect scrutiny of such shifting hypotheses, although its application as a test has its limitations. Further, another dimension to falsifiability is its important emphasis in overcoming confirmatory bias. Scientists may have a tendency to settle on a theory quite early, and by doing so then look for confirmatory rather than discrediting evidence.[30] An emphasis on potential falsifiability counter-balances that bias. Falsifiability is, accordingly, both a test and a check.

[27] Thomas Kuhn, *The Structure of Scientific Revolutions* (University of Chicago Press, 3rd ed, 1996).
[28] Gary Edmond and David Mercer, 'Conjectures and Exhumations: Citations of History, Philosophy and Sociology of Science in U.S. Federal Courts' (2002) 14(2) *Law and Literature* 309, 339.
[29] See Marcia Angell, *Science on Trial: The Clash of Medical Evidence and the Law in the Breast Implant Case* (W W Norton & Co, 1997).
[30] Foster and Huber (n 3) 44–5.

Generally, any scientific theory must be testable and consequently falsifiable, but regardless, is only ever tentative and contestable. Scientific conclusions are subject to revisions in perpetuity. A recent example is the revision of the theory that mesothelioma can be caused by a single fibre of asbestos and the acceptance of the cumulative effect mechanism.[31] The certitude of any scientific witness for a particular theory must consequently be viewed sceptically. At most, what can be said of any scientific theory is that it is empirically adequate, temporarily. And the high point for the empirical adequacy of a theory is only that it is consistent with and explains the observed or observable data in that same class. But no monopoly of truth is conferred. There can be multiple theories of equal or greater empirical adequacy discoverable. And so the evolution of scientific theories, reflective of a Darwinian pattern. But that is not to say that at a particular point in time one cannot take a theory as 'fact' in Gould's sense that it is 'confirmed to such a degree that it would be perverse to withhold provisional assent'.[32]

B *Science: A Purely Rational Objective Activity?*

But not only is science always tentative and revisable. Science is also not solely a rational objective activity infused with Mertonian values such as universalism, disinterestedness and organised scepticism.[33] Theory selection is intertwined with the subjective frameworks of the scientists and their communities. Theories are formulated and selected utilising values that are partly subjective in content, prioritisation and application. As Edmond explains, the 'extra-social image of objectivity' is untenable;[34] 'what is considered objective depends on the stance, commitments and assumptions of the observer (or adjudicator)'.[35]

Values, talents, circumstances and the particular field guide a scientist's preference between qualitative or quantitative methodologies. Further,

[31] See *Amaca v Booth* (n 10) [21], [52], [72], [79]–[82].
[32] Stephen Gould, 'Evolution as Fact and Theory' (1981) 2 *Discover* 34 reprinted in Gould, *Hen's Teeth and Horse's Toes* (W W Norton & Co, 1994) 254.
[33] See the discussion of the views of the sociologist Robert Merton in Gary Edmond, 'Merton and the Hot Tub: Scientific Conventions and Expert Evidence in Australian Civil Procedure' (2009) 72 *Law and Contemporary Problems* 159, 170.
[34] Gary Edmond, 'After Objectivity: Expert Evidence and Procedural Reform' (2003) 25 *Sydney Law Review* 131, 135. Moreover, if scientific evidence has specifically been gathered for litigation or a theory formulated or developed in that context, objectivity develops an illusory quality.
[35] Ibid 136.

predispositions of the scientist may change over time as experience is accumulated.[36] More generally, value-laden or self-interested reasons guide prevailing research agenda and how questions are framed.[37] Indeed, research agenda that seek to challenge the prevailing wisdom in an area, for example, string theory,[38] may not be funded or the contrary scholars disadvantaged in obtaining research positions.

Indeed, the recent COVID-19 episode demonstrates how science can be infused with or affected by social values. First, some scientific experts have turned into advocates for risk assessment and public policy positions. Indeed, some seem to have developed a taste for 'rule by experts'. Second, some experts qualified in one field have held themselves out as experts in other fields in which they have no necessary expertise. Third, scientific debate has been skewed by impediments to publishing research and the filtering processes of digital service providers. Fourth, some scientists have lacked independence because of their connections with sources in the country of origin of the outbreak. Others have lacked impartiality because of their connection with pharmaceutical companies associated with vaccines. Fifth, some scientists have pushed their own statistical modelling, with little peer review let alone general acceptance. Of course, the very nature of a pandemic can skew approaches in understandable but nevertheless problematic ways.

Let me broaden the scope again. Facts in science are socially constructed. They are produced 'by human agency through the institutions and processes of science'.[39] And prior agreements about the correctness of theories, methods, instrumentation, validation and review are necessary. But these are socially derived 'through continual negotiation and renegotiation among relevant bodies of scientists' as Banks explains.[40] The consequence is that truth becomes contingent on experimental or interpretative conventions.

[36] J Morgan Kousser, 'Are Expert Witnesses Whores? Reflections on Objectivity in Scholarship and Expert Witnessing' (1984) 6(1) *The Public Historian* 5, 14.
[37] Ibid 13.
[38] Lee Smolin, *The Trouble with Physics: The Rise of String Theory, the Fall of a Science, and What Comes Next* (Mariner Books, 2007).
[39] N Kathleen Banks, 'Trials and Tribulations: Social Science Evidence, Expert Witnesses, the Voice of Authority and the Discourse of Ideology in the Courts' (1999) 6(4) *Murdoch University Electronic Journal of Law* 42, [19].
[40] Ibid.

Moreover, as scientific facts are presented and accepted in a social context, they may have to fit the social objective at hand.[41] Scientific concepts may have to be transposed into social concepts or structures, including hybrid legal and scientific criteria or terminology, as elucidated by the Honourable Robert French.[42] A statute may directly borrow from a scientific field an express evaluative standard or classificatory boundary. Alternatively and more indirectly, a statute may use a concept, say, a state of mind, the application of which may be informed by a term, evaluative standard or classificatory boundary from a scientific field. Generally, the boundary between legal and scientific concepts may be permeable. Moreover, even on the science side of the boundary, 'evaluative and normative considerations may intrude into scientific taxonomy'.[43] So, not only may there be osmosis across the boundary, but separate normative judgments on both sides of the boundary may come into play.

It is important to be clear about this when dealing with scientific testimony in terms of how normative or classificatory considerations may intrude into the evidence of a witness. Now debates about the application of a classification do not usually turn on disputes about primary facts. They are about their labelling. So if an expert has applied a label, what normative or classificatory considerations were involved? In other words, one needs to separate out the expert's characterisation based upon such matters from any scientific inference drawn from the primary uncontested facts. The former in truth dwells in the realm of the argumentative rather than the factual. Let me return to scientific theories as distinct from classifications.

At an even broader level, Kuhn[44] expounded the concept of paradigms to explain how scientific theories come to be accepted and further evolve. A paradigm in a particular subject area embraces the carrying out of what is described as normal science in that field and its then temporarily embedded theories. But when a crisis in theory or application occurs, with new competing theories put forward to solve that problem, there may be a paradigm shift.

[41] See Foster and Huber (n 3) 24–7.
[42] Robert French, 'Science and Judicial Proceedings: Seventy-Six Years On' (2010) 84 *Australian Law Journal* 244, 252; see also Robert French, 'Judging Science' (Lecture, Kos conference, 30 May 2011) 6–7.
[43] French, 'Judging Science' (n 42) 7.
[44] Kuhn (n 27).

Alternatively, there may be different paradigms at work in the same subject area at the same time within different scientific communities. So-called accepted scientific theory used by a witness is a function of a particular paradigm, itself a function of the particular sociological conditions applying to the scientific community within which that scientist works. One example of this is in the field of epidemiology which displayed opposing views on the standards for epidemiological analysis to be reliable in the Bendectin / Debendox litigation; the various knowledges also came with 'no 'neutral' means of determining their appropriateness, comparative advantages and limitations'.[45] Another example is the feuding between frequentist statisticians and Bayesian statisticians which has been said to rival that between Shia and Sunni in its intensity.[46]

Kuhn's philosophy, which posited a more sociological picture of theory development, may be contrasted with Popper's philosophy, which posited theory development as more homogenous and rational through repeated linear applications of falsification, with each new successor theory falsifying its predecessor (or reducing its predecessor to a special case) whilst increasing the scope of its empirical adequacy, and in turn subjecting itself to being falsified. Moreover, Popper's notion of the practice of attempted falsification of scientific theories as being a continuous and standard practice of the scientific method is in tension with Kuhn's paradigmatic approach, which portrays a picture of a tendency to immunise theories used within a particular paradigm from attempted falsification until a crisis in normal science develops.

Further, evaluation and acceptance of competing theories is complicated by problems with incommensurability, translation and understanding. When different theories purport to explain the same data, conceptual differences may manifest themselves at the semantic level. Incomparability may arise where different theories posit different unobservables or posit the same unobservable but define or use it differently.[47]

[45] Gary Edmond and David Mercer, 'Litigation Life: Law-Science Knowledge Construction in (Bendectin) Mass Toxic Tort Litigation' (2000) 30(2) *Social Studies of Science* 265, 298–9.

[46] Jerome Ravetz, 'Conventions in Science and in the Courts: Images and Realities' (2009) 72 *Law and Contemporary Problems* 25, 38.

[47] Most scientific definitions are precise as was drummed into me when majoring in chemistry. Necessary and sufficient conditions are specified. But some definitions of unobservables consist of a collection of properties with debate about the number of properties which must exist for a particular

Generally, scientific theories ought to be seen in a relativistic context rather than in some objective immutable framework. No scientific theory has been chosen and accepted on a purely rational objective basis. Its formulation and acceptance is significantly affected by its sociological context. Now this is not to say that theory selection is not substantially a rational process. Undoubtedly, rational epistemic values are at work in theory selection such as internal consistency, simplicity, unification, coherence with other theories, explanatory power and predictive power. But it is to say that theory selection and acceptance is affected by non-epistemic contextual factors, notwithstanding the certitude that scientific experts assert as to the purely rational objective foundations of their theories. Indeed a particular science can be as fragmented and pluralistic as other fields of intellectual inquiry.

Moreover, if any consensus does exist, it may be rationally undermined by the adversarial process of litigation which deconstructs representations of historic facts, exposes contingencies and hidden assumptions which underlie claims, and 'privileges scepticism over consensus'.[48] And in the litigation context, experts may differ not just on theoretical grounds, but also because of the mode in which their expertise is being used. A scientist may have theoretical knowledge which is not in dispute, but differences of opinion may arise in its application to litigation facts which are 'multifarious, haphazard and often unique', incomplete or require 'close questions of judgment'.[49]

C *Scientific Models and Mathematical Components*

Scientists use theoretical models, including representational entities to reflect aspects of the world. Models either constitute the scientific theory or they mediate between the theory and the world.[50] But models and their corresponding theories only describe idealised, manufactured or controlled situations. And theories relying upon such models only apply in situations resembling their models.

entity to fall within an embodiment of the definition. See also Howard Sankey, 'Incommensurability, Translation and Understanding' (1991) 41 *The Philosophical Quarterly* 165, 414.

[48] Banks (n 39) [18].

[49] Korn (n 1) 1092.

[50] Sharon L Crasnow, 'Models and Reality: When Science Tackles Sex' (2001) 16(3) *Hypatia* 138, 142–143.

Theoretical models can use abstract objects which are unobservable such as quarks, although their existence may be inferred. There is a debate between scientific realists and anti-realists about the truth of theories which posit such unobservables. Scientific realists posit the approximate truth of such theories and the ontology of such unobservables. So-called anti-realists accept the empirical adequacy of theories, that is, that they are adequate to explain empirical observations, but are agnostic about the truth of theories and the ontology of unobservables. Such a philosophical question underlies any scientific theory, although most scientists are scientific realists. I will not linger on precisely what is meant by an unobservable which I have discussed elsewhere using van Fraassen's counter-intuitive example of light.[51]

Further, to bridge the gap between a high level theory and the lower level phenomena, you may have multiple models, namely, the high level theory and its attendant model, an experimental theory with its model, and a theory of the base phenomena with its model. Moreover there may be shared structural components between the layers.[52]

So when a scientific theory is being discussed or analysed, it is based upon a model. But such a model will only deal in an idealised way with empirical phenomena, and may also posit theoretical entities or unobservables, which the history of science shows are usually replaced or reconceptualised in each successive theory, the latest being quantum field theory which is well accepted. There may, however, be some continuity in the structural elements of these unobservables and their relationship with each other in each successive theory. Moreover, some unobservables may change their status to observables with new or enhanced measuring techniques.

Let me turn to the mathematical components of scientific theories and begin with some history describing how mathematics has been used in different ways.

[51] Jonathan Beach, 'Scientific Evidence: A Need for Caution in Decision-Making' (2010) 42(1) *Australian Journal of Forensic Sciences* 49, 60, citing Bas Van Fraassen, 'Constructive Empiricism Now' (Paper, Meeting of the Pacific Division of the American Philosophical Association, 2000). The observable is not just confined to what the unaided human eye can see. The use of electron microscopes, X-ray diffraction techniques etc may make something observable.

[52] See generally, Katherine Brading and Elaine Landry, 'A Minimal Construal of Scientific Structuralism' (Symposium Paper, Philosophy of Science Association Biennial Meeting, November 2004).

One use involved treating mathematics in an instrumental sense. So, mathematics was not seen as underpinning reality, but rather used as a tool to solve practical problems. Mathematical hypotheses were put forward to facilitate predictions or calculations without any assertion that they truly represented reality. Generally, Aristotelian physics relegated mathematics to this secondary role. Mathematics was not seen as underpinning natural philosophy and the causes of phenomena. So, if one considers the use of geometry by Ptolemy and his modelling for eccentrics and epicycles, geometry was not used to explain underlying causes but rather to mathematically represent physical observations on the assumption that celestial objects followed the perfection of uniform circular motion. The Ptolemaic use of mathematics was in this sense instrumental.

Another use of mathematics moved beyond the instrumental. It treated mathematics as underpinning the laws of nature, not in an idealised Platonic sense, but more in the sense that mathematical laws or relationships could be gleaned from and explained observed nature and causes. So, from observation a mathematical hypothesis could be conjectured and derived from induction. From that hypothesis, deductive inferences could be drawn and new predictions made. The testing of new predictions may then have verified or falsified the theory. If verified, a mathematical law was produced. So mathematics reflected reality, not in the sense of any metaphysical idealisation underlying the universe, but what could actually be observed. If the mathematical theories worked in making accurate predictions or according with observations, it was because the theories were true.

Yet another and idealised use of mathematics involved Platonism, which involved the 'symbolism of numbers and the spiritual imagery of geometrical figures'.[53] As Carré described it, '[g]eometrical axioms [were] representations of divine beings, or emanations from the ineffable unity';[54] reality was hidden behind appearances. Geometry was considered to have mystical properties. But the mathematics with which Plato was concerned did not apply to the physical world. Platonism and its mathematics dealt with idealised and eternal objects of

[53] Meyrick H Carré, 'Platonism and the Rise of Science' (1955) 30(115) *Philosophy* 333, 335–6. I will not linger on later schools such as neo-Platonism or Renaissance Platonism.
[54] Ibid.

perfection and not the imperfection of observed objects and their position or motion in the physical universe. So, Platonism did not principally involve the use of mathematics to explain or derive predictions about the non-idealised physical world. It was abstracted. It was idealised. An example of the Platonic approach relates to its tenet of uniform circular motion. Uniform circular motion for celestial objects was the pure position even though the appearances of planetary motions reflected otherwise. So, to save the appearances, astronomers were set the problem of finding and tweaking the appropriate combinations of uniform circular motions, leading to the Ptolemaic system. Now Platonism was itself derived from Pythagorean philosophy. But although Pythagoreans attributed mystical properties to numbers in the sense of geometrical units as Burtt explains,[55] Pythagorean mathematics also sought to derive mathematical truths and principles about the real world system.

Let me turn to the present. Scientific evidence adduced in litigation may use mathematical formulae. But several observations need to be borne in mind so that one is not mesmerised by their notations and apparent precision.

First, in the forensic context, a formula is usually used to explain an apparent regularity between empirical observations. But a formula and the underlying data does not of itself demonstrate any necessary causal connection. From a correlation between the movement in two variables, sometimes such a link is inferred. But philosophically this is always contestable in a Humean sense, and practically may be contestable in many individual cases. The correlation may be due to a separate but common cause. Movement in one variable preceding movement in the other is only the starting point for any analysis.

Second, a formula may only reflect a regularity observed from present or past data. And its function may simply be to explain that data rather than as a predictive tool. But even as an explanation for past data, the formula may have its limitations. Past data upon which the formula is built may only be a limited sub-set of the available data. The complete data set, if available, may change the apparent pattern of that regularity.

[55] As Burtt says: 'The famous Pythagorean doctrine that the world is made of numbers is apt to appear quite unintelligible to moderns till it is recognised that what they meant was geometrical units i.e. [a] sort of geometrical atomism ... They meant that the ultimate elements of the cosmos were limited portions of space.': Edwin Arthur Burtt, *The Metaphysical Foundations of Modern Science* (Dover Publications Inc, unabridged reprint of 2nd rev ed, 2003) 42, 44–5, 55.

Third, and relevantly to the predictive case, a formula may be a useful explanatory tool to explain observed phenomena, yet as a predictive tool it may have little utility. Take 10 empirical observations which are plotted on a graph showing for each observation variables *x* and *y* measurements. Assume that this graphing shows that the 10 points can be connected by a straight line. You might conclude that there is a linear correlation between variables *x* and *y* and posit a straight line function. So you have a good explanation of the relationship between your 10 observations. But how good is the formula for predictions? Theoretically there are an infinite number of lines including curved that could have been drawn to join the 10 points, with different formulae. And a further measurement may show the linear function not to hold. More generally, for any set of empirical data there are multiple potential theories to explain the same, which gives rise to the under-determination problem. Generally, one theory consistent with the data may usefully explain observed data but may be a questionable predictive tool. Whether it is also a successful predictive tool may only be ascertained by the longevity of its success. But even longevity does not establish truth; consider the longevity of Ptolemaic astronomical methods of calculation that were superseded by Newtonian mechanics.

Fourth, a mathematical formula takes its subject matter and their measurement as idealised and precise. But empirical data may be imprecise in quality or measurement. At most, any formula is only an idealised representation of the apparent regularity consistent with the model underpinning the theory. Moreover, what happens when there is a soft variable where no measurement can be made? It may be omitted from the formula or its applied computer algorithm with a corresponding distortive effect.

Generally, mathematical equations should be seen in their limited context as imperfect tools. Their apparent elegance ought not to be taken as giving a greater air of verisimilitude to a scientific theory or its applied results than is warranted considering all qualitative and quantitative aspects.

Now scientific debates are not about mundane computational questions. Scientific debates are usually about either the validity of a formula or its utility. Now assuming validity for a moment, what do I mean by utility? First, the mathematics may only be a small part of a predominantly qualitative theory. If so, the use of the mathematics ought not to be seen as adding greater weight to the theory than is warranted. Second, the mathematics may only be useful for understanding a static state, whereas an understanding of a dynamic state may be required. In the Longford Royal Commission, which investigated the cause of

a gas plant explosion, what needed to be investigated was the dynamic state of the chemical processes within the plant in the four hours preceding the explosion. But the then mathematics only allowed for static or snap shot exercises which were of limited utility. Dynamic mathematic models are now deployed more readily. But where what is being modelled involves numerous variables which are interdependent, such virtual reality exercises can produce significant errors within very short time runs, no matter the integrity of the independently verified initial conditions or how well calibrated the operation of the model is against historical data runs.

Further, the broader context must be considered. As Tribe posed the question: is there any acceptable way of combining mathematical with non-mathematical evidence?[56] And how do you integrate mathematics into the trial process? Such questions have no answers that would satisfy the purist. Nevertheless, judges have their workable solutions tailored to the individual case that seem to work nicely.

D *Probability*

Any physical phenomenon is intrinsically probabilistic. And so the dominance of the Copenhagen interpretation of quantum mechanics[57] and the rejection of any hidden-variable thesis by the proof of Bell's theorem and the violation of Bell's inequalities.[58] Uncertainty is intrinsic to such phenomena as Heisenberg explained,[59] whether his principle is expressed in terms of momentum / position or energy / time. Further, Schrodinger's wave function is best thought of as a probability wave, rather than a real wave, as Born explained.[60]

But irrespective of whether probability is intrinsic to nature, scientific assessments and judgments are probabilistic because of complexity in the systems being studied and imperfect knowledge of all relevant scenarios,

[56] Laurence H Tribe, 'Trial by Mathematics: Precision and Ritual in the Legal Process' (1971) 84(6) *Harvard Law Review* 1329.
[57] Manjit Kumar, *Quantum: Einstein, Bohr and the Great Debate About the Nature of Reality* (Icon Books, 2008) 376.
[58] Ibid 344–350; see also the entry for 'Bell's Theorem' in the *Stanford Encyclopedia of Philosophy* (online).
[59] Kristian Camilleri, *Heisenberg and the Interpretation of Quantum Mechanics* (Cambridge University Press, 2009) 85.
[60] Ibid 69.

observations and parameters. It is for this reason that scientific inductive hypotheses are expressed in probabilistic terms. Probability is a function of uncertainty. As Jaffe described it:[61]

> Proof of probability does not create uncertainty. A statement of probability is merely an uncertain estimate of actuality.

As to probabilistic evidence, concentration on mathematical probabilities can prejudice common sense.[62] Wrong intuitions can arise. Moreover, there are a number of potential fallacies to guard against, a discussion of which is beyond the scope of this paper.

Let me say something on Bayes' theorem, which has been discussed in the legal context including in Tribe's well-known paper.[63] This is a theory reflected in a mathematical formula which takes the prior probability of a hypothesis based upon certain evidence and then adjusts that probability based upon the addition of new evidence. That is, does the new evidence make the hypothesis more or less probable than the hypothesis without that evidence? Another way to view the theorem is as a means to update one's belief system, rather than as an objective measure of the probability of a hypothesis.

Unhelpfully, such a theorem has serious forensic problems in its application, although its underlying tenets have intuitive appeal. First, you need estimates of all prior probabilities, which may not be available or calculable because either the data is lacking or the data does not render itself to a probabilistic analysis. Moreover, if the particular prior probability of the new evidence is certainty, the utility of the theorem is limited. Second, in any event, any calculation of prior probability estimates is substantially subjective. There are few rigorous objective standards to assess them except in the simplest of cases which are not relevant to the usual forensic context. Third, such estimates can only be set against background knowledge that may be contestable. Fourth, the compartmentalised assessments of each of the relevant probabilities, whether prior or posterior, is necessarily artificial and their utility questionable. More generally, although

[61] Leonard R Jaffee, 'Of Probativity and Probability: Statistics, Scientific Evidence, and the Calculus of Chance at Trial' (1985) 46(4) *University of Pittsburgh Law Review* 925, 934.
[62] See David H Hodgson, 'Scales of Justice: Probability and Proof in Legal Fact-Finding' (1995) 69(9) *Australian Law Journal* 731, 736.
[63] Tribe (n 56) 1351.

Bayes' theorem has some application in particular scientific contexts such as DNA analysis, it is not useful as a separate tool for lawyers when considering how probabilities of a particular hypothesis are affected by the addition of new evidence. Qualitatively, you may be able to draw relevant conclusions, but the quantitative tool has little direct application.

Now associated with the question of probability is statistical analysis. Data can be assimilated and extrapolated for explanatory or predictive purposes, with the tenets of probability theory underlying the analysis generating specific probability estimates. I will discuss statistics later when dealing with causation.

E *The Theory Laden Quality of Scientific Observation*

Scientific observation is often portrayed as an objective tool separate from theory. But it is theory-laden. Observations are usually interpreted in the light of theory. And even where the observations are being made in the Popperian sense of attempted refutations of a theory, the observations are usually interpreted through the embedded theories of the prevailing paradigm that is being challenged by the new theory.

Scientific observation is usually performed with instruments. And so for a reliable observation you need not only a theory of what is sought to be observed, but also a theory as to its means of detection and measurement, such as the theory of optics underpinning microscopes and telescopes. Indeed, the relevant phenomenon or entity may not be directly measurable but only inferred from other data, such as traces left in a cloud chamber used to infer the existence and trajectory of a particle. Further, you need a theory as to the design of an instrument which accords with such theories, the reliable operation of the instrument and a theory as to the interpretation of the results produced, including any magnitude of error involved in these elements.

And even direct or mediated visual observations can depend upon theory or the observer's perceptual experience or knowledge. Polanyi[64] gives the example of a medical student who is taught to make a diagnosis based upon an x-ray picture. Initially the student may perceive shadowy blotches and possibly

[64] Michael Polanyi, *Personal Knowledge: Towards a Post-Critical Philosophy* (Routledge & Kegan, 1973) 101.

shadows of the heart and ribs. After several lectures the student may see through the ribs to observe the lungs. Eventually, after more lectures the student may see a rich panorama of physiological variations in the lungs. Another example is provided by the Longford Royal Commission where microscopy on a fracture point in the weld of a heat exchanger[65] was open to conflicting theory-laden interpretations as to whether the crystallographical observations displayed inter-granular or trans-granular cleavage in the weld. Indeed the interpretation of these pictures displayed more of an art than a rigorous scientific method.

Problems are also exacerbated when the interpretation of observations has injected into it subjective considerations peculiar to the particular expert witness, such as the experience of the witness, including the number and diversity of occasions of making and interpreting relevant observations. Further, one may have to consider the subconscious biases of the witness in interpreting such observations, including any idiosyncratic preferences given to qualitative over quantitative techniques; the greater that preference, the more that may be read into the observation or the more dogmatic the qualitative interpretation advanced by the witness.

Generally, it is not justifiable to give scientific observation some status independent of theory. The former is usually built upon or interpreted by the latter, particularly where mediating instruments are involved. Both the quality of the observation and its theoretical underpinnings either for the making of the observation or its interpretation need to be scrutinised.

Now having set the scene, I should turn to causation.

III CAUSATION

I propose to address factual causation in the context of historic cause, and so the question of whether the act or omission of the defendant caused the plaintiff's harm. For the moment, I will put to one side likely future causation questions that may be involved in regulatory action or litigation. So, I am focusing on scenarios of past causation, which may involve a retrospective counterfactual. If the act or omission had not occurred, would the harm have been avoided?

[65] This exchanger in a lean oil absorption gas processing plant in Longford, Victoria failed, with consequential vapour release and then ignition.

Now I am not concerned with a philosopher's perspective on causation, which might range from a metaphysical interpretation giving it some form of realism through to Humean scepticism where in its purest form not even correlation can be used to infer causation. And nor am I focusing on normative legal questions derived from making cause an element of liability, although I should make some brief points.

In the context of determining legal responsibility for the causal connection between a tortious act or omission and harm, in some cases showing that the act is factually a necessary condition may be enough. But in other cases it may not be. More generally, the causal requirement in a particular case will depend upon the context, nature and form of liability being considered, including the nature of the normative standard and its breach, and the nature of the loss or damage. But given my focus I do not need to grapple with labels applied to causes such as 'direct', 'natural and probable', 'operative', 'efficient', 'proximate' and the like. Their imprecision is apparent, but their use is explicable once one understands that they are infused with value judgments designed to address the question of legal responsibility.

Causation is also relevant to the question of indeterminacy and ripple effects when considering the law of negligence. The ripple effect concerns acts or omissions that produce secondary claims. The loss of a first line victim can produce loss to a second line victim and so on. The ripple effect entails causal indirectness.[66] The law of negligence has no affection for ripple effects and victims beyond the first line.

Indeterminacy can arise not just in pure economic loss or property damage cases, but also in personal injury scenarios.[67] For example, a computer virus may cause a widespread shut down in computer systems for utility services in hospitals or buildings leading to personal injury. Another example is the diffusion of electro-magnetic radiation or a chemical which had a mutagenic effect on the germ cells of a first generation population resulting in an inherited deformity for the second generation. Claims by the second or later generation

[66] Jonathan Beach, 'Indeterminacy: The Uncertainty Principle of Negligence' (2005) 13 Torts Law Journal 129, 140, 141.
[67] Ibid 150, 151.

may entail indeterminacy.[68] For the moment I do not need to discuss this control mechanism further or its potential inverse, namely, requiring sufficient closeness and directness. Let me return to the factual question of causation.

One can consider the general factual question of whether an agent can cause harm. As Scheines[69] points out, such a focus usually looks at a population of individuals and considers relative risk rates of harm when exposed to an agent. Now this tells you little about whether an individual has suffered harm by the exposure, but it does inform the question of whether a particular agent is capable of causing harm.

Of course, in such a context causation is a hypothesis which seeks to link an agent with a particular harm. It is not a directly observable event in one time and place. Indeed, as Edelman J described it, factual causation posits a 'metaphysical relationship between an event and an outcome'.[70] I am not here dealing with physical contact scenarios such as a car driver's door hitting a passing cyclist. Further, a causal claim may not have some unique event 'that can be documented, verified, and directly observed'.[71] You may know the agent. You may know the cancer. But the causal connection is a scientific hypothesis, which may be more or less well supported.[72] Indeed, as the human body is a 'complex dynamic system'[73] where there is inherent and fundamental uncertainty in how it will react to exposure, such uncertainties may not be capable of reduction.

Further, as Weed explains, under-determination looms large in this context of causation, in that 'the available scientific evidence under-determines the choice between the various alternative hypotheses that can explain that evidence'.[74]

[68] The thalidomide episode was not such a case as thalidomide had a direct teratogenic effect on the embryos. So the second generation was exposed at the same time as the mother. For a mutagen, the effect is on the germ cells i.e. before inheritance. The second generation would not even exist in the embryonic form at the time of exposure.

[69] Richard Scheines, 'Causation, Truth, and the Law' (2008) 73(3) *Brooklyn Law Review* 959, 961. I should say here that I am not concerned with formalised demarcations between general causation and specific causation that may be made in non-Australian jurisdictions.

[70] *Caason Investments Pty Ltd v Cao* (2015) 236 FCR 322, [153] (Edelman J).

[71] Douglas L Weed, 'Truth, Epidemiology, and General Causation' (2008) 73(3) *Brooklyn Law Review* 943, 949.

[72] Ibid.

[73] Kriebel (n 7) 122.

[74] Weed (n 71) 950.

In addition to the general factual question one has to consider the specific factual question: did exposure to this agent cause the particular plaintiff's harm? The framework is individual and specific. Was the plaintiff exposed to a sufficient degree? But for the exposure, would the harm not have occurred?

Given how the 'but for' test is formulated, one poses a counterfactual world.[75] So the question is whether the plaintiff's harm would not have occurred if the exposure to the apparent agent had not occurred. Indeed, a counterfactual world may also be posed for general causation claims relevant to the causation hypothesis. [76] But it may be impossible or impractical to assess these counterfactual worlds. Hence other less ideal techniques have to be used to draw inferences from not only what has occurred, namely, the factual world, but what has not occurred but could have occurred in your retrospective hypothetical, namely, the counterfactual world.

Now as Bant and Paterson explain,[77] the 'but for' test only connects to the factual world to the extent that 'it is legitimate to draw inferences from the metaphysical world to reality'. And simulating the hypothetical world to apply the 'but for' test may be difficult. What if there are multiple conditions or factors posited as causally relevant? Which do you remove to then simulate the counterfactual? What if there are some conditions or factors that are unidentified or if identified are unknowable? What counterfactual do you use? And how is the reliability of the inference from the counterfactual to the reality to be assessed?

Moreover, the 'but for' test gives rise to problems of over-determination and under-determination.[78] For the former problem, what do you do when there are two or more independently sufficient factors to produce the harm? For the latter problem, what do you do when there are multiple conditions, each of which if taken singularly are neither necessary nor sufficient to produce say an indivisible injury but together are sufficient, that is, one dimension of the material contribution scenario as Stapleton discusses?[79]

[75] Scheines (n 69) 960.
[76] Ibid 962.
[77] Elise Bant and Jeannie Paterson, 'Statutory Causation in Cases of Misleading Conduct: Lessons From and For the Common Law' (2017) 24 *Torts Law Journal* 1, 14.
[78] Ibid 15.
[79] Stapleton, 'Unnecessary Causes' (n 18).

Bant and Paterson have posed an alternative to the 'but for' approach. Their solution is said to avoid the problems with the over-determination and under-determination of the 'but for' test. Indeed, it is said to avoid a consideration of any hypothetical or counterfactual world altogether. They posit the concept of 'a factor' contribution as a test of causation, which may have some resonance with Stapleton's suggestion concerning showing that a breach of duty resulted in a positive contribution, assuming it to be neither necessary nor sufficient, to how an indivisible injury came about. So, their approach poses the factual issue of whether an event played a role in the historical process that led to the harm. As they explain,[80] the posited cause does not need to be a 'but for' cause. One asks whether the posited cause formed one of the historical conditions that, together with the other conditions that were actually present, played a role in producing the harm. No positing or analysis of a counterfactual world is necessary, so they say. One just analyses the actual historical processes.

Now this approach has some attraction. But forensically their approach may not completely avoid a narrower form of 'but for' analysis if one is required to address the question of not just 'a factor' but 'a material factor', although Bant and Paterson eschew the latter formulation. So, to ask whether a factor played a material contribution, you might ask what might have been different if that factor had not been present. But I can pass on from any of these questions as I am more concerned with the forensic construct than the legal construct.

Let me now address some topics of contemporary interest where the law and science intersect in the causation context, concerning:

1 statistical evidence;
2 some general issues;
3 artificial intelligence; and
4 COVID-19 vaccines.

[80] Ibid 16. I should say that much of their discussion is in the context of decision causation, looking at the impact of some matter on a plaintiff's decision to act or refrain from acting in a way relevant to the plaintiff's ultimate harm. As they would have it, one cannot unpack decision-making under laboratory conditions, as one can the passage of a bullet from a gun, and see what would have happened if X had not occurred. The mind is largely a blackbox. And to create a hypothetical, such as what a person would have decided or done had X not occurred, both literally and metaphorically has an air of unreality to it.

A *Statistical Evidence*

Statistics in the forensic context involves the analysis of surveys or experiments to draw cause and effect inferences, for example, the potential mutagenic or carcinogenic effects of a chemical or radiation.

Issues of substance concerning collected data and its context are intertwined with statistical issues.[81] Intertwining may also occur between a particular scientific theory and statistical issues. For example, a theory may be mathematically posited as to the relationship between variables *x* and *y*. Such a theory is deterministic.[82] But data on *x* and *y* may be imprecise or difficult to generate. Statistics may need to be gathered and assessed, and assumptions made as to their distributions.[83] The combination of the theory with the statistics then provides the explanatory or predictive tool. Now any mathematical formula used in statistics may be derived by reasoning from a priori principles or experimentally, as Imwinkelried explains.[84] But whichever way, various questions may arise. Is the formula generally accepted by scientists / statisticians in the field? Does the formula omit factors that are relevant but too soft to quantify? Are the sources for, or measurement of, the variables of the formula unreliable?

One common use of statistics in the forensic context is in epidemiology. Epidemiology may be used to infer a cause and effect relationship between an exposure to an agent, say, a chemical or radiation, and subsequent disease or death. But some general points should be made.

First, epidemiology can only directly establish that there is a correlation between exposure to a particular agent and disease. But correlation is not causation. Correlation may assist with drawing an inference about causation, but that is all. And even if correlation shows that there is an increased risk, that does not necessarily establish causation in terms of material contribution.[85]

[81] Stephen E Fienberg and Miron L Straf, 'Statistical Evidence in the US Courts: An Appraisal' (1991) 154(1) *Journal of the Royal Statistical Society* 49, 53.
[82] *Australian Gas Light Company v Australian Competition and Consumer Commission* (2003) 137 FCR 317, [495] (French J).
[83] Such an assumption may involve a stochastic conjecture about a probability distribution, say, a normal distribution curve, for the data concerning a particular variable.
[84] Edward J Imwinkelried, *The Methods of Attacking Scientific Evidence* (LEXIS Law Pub, 4th ed, 2004) Chapter 6.
[85] *Amaca v Booth* (n 10) [41]–[43].

Second, although epidemiology may show a statistically significant correlation between agent and effect, that may only show correlation for a population. But to draw an inference about causation in an individual case, you need to demonstrate that that case is 'conforming to the pattern described by the epidemiological studies'.[86]

Third, even if the specific case conforms to that pattern, showing by the epidemiology that a small percentage of cases of disease were caused by the agent does not, without more, establish that for the specific case. To show that an agent can cause disease does not entail that it did in the specific case.[87]

Fourth, if a disease can be brought about by multiple different agents, but the epidemiology only deals with a sub-set of those agents, such studies with their relative risk ratios and attributable fractions calculations based only upon the sub-set may have little utility. All results from all realistic possible causes must be considered.[88] If studies for other potential agents have not been carried out, the utility of the epidemiology which has been carried out is diminished.

Fifth, and correspondingly, the fact that epidemiology may not establish a positive link between agent and effect does not entail that such a link does not exist, although it would have to be established by other evidence.[89] In other words, epidemiology which establishes a positive link between agent and effect is neither a necessary condition nor, as stated earlier, a sufficient condition for establishing causation.

But let us assume that you have epidemiological results that show a correlation between agent and effect. And let us suppose that the results justify the conclusion of an increased risk between exposure to the agent and effect. Can you move closer to establishing causation?

One way to do this is to consider the matters raised by Bradford-Hill relevant to cause and effect inferences, including the following questions.[90]

[86] *Amaca v Ellis* (n 9) [62].
[87] Ibid [68] and [70].
[88] Ibid [56].
[89] *Amaca v Booth* (n 10) [23], [51] and [88].
[90] See generally *Amaca v Booth* (n 10) [44]–[46]; Austin Bradford-Hill 'The Environment and Disease: Association or Causation' (1965) 58(5) *Proceedings of the Royal Society of Medicine* 295; Alfred S Evans, 'Causation and Disease: The Henle-Koch Postulates Revisited' (1976) 49(2) *Yale Journal of Biology and Medicine* 175.

What is the strength of association and its degree? The higher the relative risk ratio, the less chance of a confounding element or bias, and the more likely the ratio is to support an inference of causation.[91] But as Bradford-Hill points out, one should not dismiss a cause and effect connection merely because the observed association is slight.[92]

Is any dose response effect demonstrated? If the level of risk is directly proportionate to the level of exposure, a causal link is more plausible. For example, if exposure increases by, say, 10% and the risk correspondingly increases by that order of magnitude or greater, then that is consistent with enhanced plausibility for a causal link; however, the rate of increase of the risk as exposure increases may start to level off at high levels of exposure.

Is the time sequence correct? The exposure or risk factor must precede the disease. But sometimes this is not easily resolved. For example, does a particular diet lead to a particular disease or does that disease cause the dietary habit, in other words, reverse causality?

What other potential confounding factors have been considered in the epidemiological study?[93] Have all realistic hypothesised associations been tested? If not, you may not be able to exclude other causal agents as being involved apart from the litigated agent.

Is there consistency of result over a meta-analysis of two or more epidemiological studies? If similar results apply over similar populations, places and circumstances, well and good. If similar results apply over diverse populations, places and circumstances, there is a much stronger foundation for an inference. But one has to be careful of publication bias. Positive results studies are usually written up more than negative results. This can bias any meta-analysis.

[91] The relative risk ratio is the ratio of the disease incidence in the exposed group to the disease incidence in the unexposed group. Say for a chemical which is speculated to produce a particular cancer the ratio is 50. Underlying that ratio may be that on average 50 people in one hundred thousand of an exposed group contract the disease as compared with an average 1 person in one hundred thousand of an unexposed group. A relative risk ratio of greater than one may be said to imply a causal relation. US courts have favoured ratios of 2 or more as supporting a causal inference. I am not, of course, talking about absolute risk but only relative risk.

[92] Bradford-Hill (n 90) 296.

[93] Anthony M Graziano and Michael L Raulin, *Research Methods: A Process of Inquiry* (Harper Collins, 2nd ed, 1993) 170–182 in which the major confounding variables are listed.

Are animal toxicology studies consistent with the epidemiology? And from such studies, what is the relevant dose exposure necessary threshold level? After all, according to Paracelsus' first law of toxicology, it is the dose that makes the poison.

Is there a plausible or demonstrated human biological pathway such as to provide a micro-biological foundation for the causal inference suggested by the epidemiology? So for example, is there any agent potentially caused mutation in any DNA sequence, which mutation has the capacity to affect gene expression relevant to the particular disease? Such plausibility may involve the testing of human cells. But plausibility may also be established by animal or other mechanistic studies with perhaps many mice martyred in the process. A related area to biological plausibility is the question more broadly of whether there are any genetic biomarkers indicative of exposure or causation.

Is there any cause and effect analogy between another analogous agent, say because of structural reasons involving a similar class of compound or stereochemistry, and an analogous disease?

Now it should be apparent that none of these factors, other than time sequence,[94] are necessary conditions for a causal inference to be drawn; and of course none of them are sufficient conditions.

Epidemiology is often favoured over, say, toxicology evidence by judges assessing causation. After all, the former is based on statistics with respect to humans. But as Goldstein[95] observes, such a preference may in some situations not be deserved. First, when testing new drugs, you may be able to meet the gold standard of a randomised double blind control trial. But you can only use observational studies when considering, say, the exposure to an agent in the workplace or other environment. Second, the environment for assessing animal toxicology can be rigidly controlled, unlike observational studies of a workplace; in that latter context, only a very small percentage may have suffered the disease in question; moreover, some in the cohort may not have been exposed to the agent. Third, any mechanism of action conclusions drawn from the toxicology may diminish or even negate the causal inference otherwise suggested from the

[94] National Research Council, *Reference Manual on Scientific Evidence* (The National Academies Press, 3rd ed, 2011) 601.

[95] Bernard Goldstein, 'Toxic Torts: The Devil is in the Dose' (2008) 16(2) *Journal of Law and Policy* 551, 582.

epidemiology; alternatively, it may assist in understanding the latency period between exposure and disease.[96]

Let me say something more about the problem of extrapolation.

For animal toxicology studies, the extrapolation of results to other species, including humans, may be problematic. Further, although one might realistically extrapolate general trends of toxicity across mammals in terms of general causation, nevertheless there may be specific dose, mechanical or metabolic issues such that different mammalian classes may give rise to different issues. I mean by dose the amount entering the body. This differs from exposure, which is the concentration of a chemical in, say, air, water, or food to which the body is exposed; exposure may need to be looked at in terms of frequency and duration as well as pathways and routes of entry.[97] Further, there may be different thresholds and different dose response relationships[98] for different classes and even different individuals within the same class.

For epidemiology, there may also be extrapolation difficulties. There are various dimensions. The starting point is to extrapolate from individuals in a particular setting to a class. Then you may seek to extrapolate from one class of persons to another class of persons who have different characteristics or are in a different setting. But can you generalise from women to men, young to old, one ethnic group to another and so on? Now some of these differences may be cancelled out by any randomisation process or through statistical techniques. Nevertheless the basis for extrapolation needs to be carefully assessed.

Now before proceeding further, let me return to a topic that I just flagged.

Genetic data may also assist in more directly determining the question of causation as Marchant has explained.[99] Such data may take you beyond statistics and general causation. There are various dimensions.

First, genetic biomarkers such as DNA mutations, chromosomal re-arrangement or gene expression patterns, may be indicators of exposure to a particular agent.

[96] Ibid 586.
[97] David Eaton, 'Scientific Judgment and Toxic Torts – A Primer in Toxicology for Judges and Lawyers' (2003) 12 *Journal of Law and Policy* 1, 11.
[98] Ibid 15, 16.
[99] Gary Marchant, 'Genetic Data in Toxic Tort Litigation' (2016) 45(2) *Brief* 22.

Second, such biomarkers may more directly provide evidence of causation in an individual case. So, if genetic mutations or gene expression are correlated with a particular condition linked to a particular agent, the presence of such biomarkers in the plaintiff may assist his case on causation. Equally, a defendant may rely on the absence of biomarkers in a particular case as evidence tending to disprove causation.

Third, biomarkers in the form of genetic variations[100] may be evidence of genetic susceptibility of a particular individual to a particular condition. This may be useful evidence for a plaintiff who has to show individual causation, but has little more than general epidemiological evidence showing a favourable relative risk ratio. If the plaintiff can also show individual gene susceptibility, this will assist in establishing individual causation.

Now I have said something about the subject matter of statistics, but I should also say something on statistical significance, which determines the probability of an observed outcome being due to chance rather than real association on the assumption that the null hypothesis is true. If this probability (*p*) is equal to or less than 0.05, the result suggesting a real association is said to be statistically significant.[101] The figure 0.05 means that the relevant result is expected to happen by chance one in 20 times. But this significance level of 0.05 is only a convention. Nothing enshrines it. There is room for some flexibility at the lower end of the number so long as the consequence of adopting a p value higher than 0.05 is understood, that is, the higher the number, the less the significance to be given to any statistical correlation.

Statistical significance is relevant when you want to examine two variables where co-variance is observed. Is there a causal relationship? If there is statistical significance, the null hypothesis is rejected. You guard against a type I or alpha error of concluding a relationship where there is none (a false positive). But sometimes because of a low sample or where the relationship is weak, there may be no statistical significance. You then accept that the null hypothesis is true. But

[100] There are genetic differences between individuals produced by inter-alia DNA polymorphisms. Further, genes that code for enzymes involved in the metabolism of an agent may vary among individuals. Such variations can be correlated with susceptibility to particular conditions.

[101] But that does not tell you the probability of any cause / effect inference.

this may lead to a type II or beta error (a false negative). This can be guarded against by combining multiple studies.[102]

Further, as I have intimated, you have to be careful with statistical significance testing. It tests the null hypothesis. But as Bradford-Hill said, 'far too often we deduce 'no difference' from 'no significant difference".[103] Further, one should test the significance of all hypotheses, not just the null hypothesis. Non-significance does not mean that the null hypothesis is the most likely explanation of an association.[104] Further, it is a fallacy to treat the absence of evidence of a relationship as evidence of no relationship.

Finally on statistics, I should say something about linear regression analysis, which can assist in inferring causation from the correlation of movement between two or more measurable quantities. Say it is observed that variable A moves before or at the same time as variable B. Correlation of movement between variable A and variable B may not prove that A caused B. What if there is another variable that independently changes both A and B? What if there is a variable that intervenes between A and B and acts independently on B? Correlation of movement may be a necessary condition for causation, but it is not a sufficient condition.

Linear regression analysis is not limited to scientific evidence. Statistical analysis in cartel cases to establish causation may take the form of multiple linear regression analysis that analyses the relationship between a variable of particular interest (dependent variable), say, the price of goods, and other variables that explain movements in that dependent variable over time (explanatory variables), such as the costs of producing such goods.[105] An additional putative explanatory or dummy variable is added as the proxy for unexplained factors which may influence the dependent variable. Multiple regressions are then run to see whether such a dummy variable has a positive co-efficient. If it does, the co-

[102] Joseph Sanders, 'Scientific Validity, Admissibility, and Mass Torts after *Daubert*' (1994) 78(6) *Minnesota Law Review* 1387. But one has to also be careful of data dredging.
[103] Bradford-Hill (n 90) 300.
[104] Sander Greenland, 'The Need for Critical Appraisal of Expert Witnesses in Epidemiology and Statistics' (2004) 39(2) *Wake Forest Law Review* 291.
[105] Jonathan Beach, 'Some Forensic Issues and Techniques' in Damian Grave and Helen Mould (eds), *25 Years of Class Actions in Australia* (Ross Parsons Centre of Commercial Corporate and Taxation Law, 2017) 103; Jonathan Beach, 'Class Actions: Some Causation Questions' (2011) 85 *Australian Law Journal* 579, 589, 594.

efficient of the dummy variable measures the extent to which movements in the dependent variable in a given period, say the posited cartel period, cannot be explained by the usual known influences that are measured by the other explanatory variables. The co-efficient of the dummy variable is then sought to be associated with the cartel's effect, as the only factor that could explain the movement in the dependent variable apart from the other known explanatory influences. Of course, the co-efficient of the dummy variable may capture other unmeasurable or unidentified factors not within the known explanatory variables that affect price, not just the effect of the cartel. The influence of these other factors may not be separable from the dummy variable; the positive co-efficient of the dummy variable may represent an indivisible factor, with the cartel effect an inseverable (or even non-existent) component thereof. Accordingly, the dummy variable, even with a positive co-efficient, may not be able to prove or disprove the existence of a cartel or measure its effects.

B *Some General Issues*

Let me make seven points on some general issues.

First, at the time of litigation there may be a lack of research on causation where one has a rare or new potential link between an agent and harm. It may simply not have been studied, particularly at the time of the first wave of litigation. A judgment on causation may need to be rendered on sparse information.

Second, let us say that you are synthesising different qualitative and quantitative evidence from different fields. What hierarchy or priority rules, if any, do you apply as between competing fields? And indeed in the same field, what hierarchy or priority rules do you apply? Now answering this latter question is less problematic. But even in the same field reasonable minds might differ on methodological rankings. Let me give an example of the broader issue.

Take the scenario where a drug is said to be a carcinogen. From weakest to strongest evidence, you might rank the available types of evidence in the order of molecular structural analogues, in vitro experiments, in vivo experiments and then epidemiology.

Structural analogues might be said to be the weakest evidence.[106] After all, very small differences, such as a different optical isomer of an otherwise identical molecule, can have dramatically different effects.

And as between in vitro experiments on cells, organs etc on the one hand, and in vivo studies on the other hand, extrapolation problems are more acute for the former. After all, you are seeking to extrapolate from in vitro studies to live animals, before you even get to the problem of extrapolation from live animals to humans. Of course, if the in vitro experiments concern human cells then the ultimate extrapolation pathway may be firmer.

Further, does epidemiology trump in vivo animal experiments? If the drug's effect has a very short latency period, then positive epidemiological results compared with equivocal in vivo experiments may be enough.[107] But what if the latter contradicts the former? You are then in more difficult territory, particularly if the latency period is medium to long term.

More generally, a judge has to carry out the necessary synthesis across all fields and may not be assisted in that broader synthesis by experts qualified only in their particular fields. Such experts can assist in priority or hierarchy rankings of methods within their own fields, but not across other or all fields. Now some experts have criticised judges who have not accepted their opinions on causation in their own field, but without appreciating that judges are required to draw conclusions based upon synthesising evidence across multiple fields embracing all expert and lay evidence.

Third, it may be that in a particular case there are different categories of scientific evidence, each of which establish that the posited causal pathway is a possibility. In such a case, no one category may support a causal inference on the balance of probabilities. Nevertheless, the combination of all categories may satisfy that standard of proof.

Fourth, if the science is thin in terms of positive evidence establishing the posited causal pathway, nevertheless if other possible causal pathways have been ruled out or are unlikely, then there may be a sufficient foundation to infer the posited pathway on the balance of probabilities.

[106] Joseph Sanders, 'From Science to Evidence: The Testimony on Causation in the Bendectin Cases' (1993) 46 *Stanford Law Review* 1, 19.
[107] Ibid 25.

Fifth, take a slightly different scenario. If there are multiple posited causal pathways, each of which shows causation between the defendant's act or omission constituting the breach of duty and the harm, then it may not matter that no single pathway can be established on the balance of probabilities. If all realistic causal pathways can be sourced to the defendant's negligence, then that may be sufficient to establish causation.

Sixth, I have not discussed the material increase in risk type scenario. Under Australian law, that is not sufficient to establish factual causation. But it is not out of the question that in a particular case a material contribution may be inferred from a material increase in risk where the contemplated or reasonably foreseeable harm has eventuated and there is no other realistic competing cause.

Seventh, so far I have concentrated on scientific expert evidence in the context of causation. But it ought not to be assumed that only scientific and related mechanistic experts have a role to play. In the US, historians have been called as expert witnesses.[108] So, where harm results after a long latency period from exposure to asbestos, radiation, cigarette toxins and the like, questions may arise concerning decades old industry knowledge. What was known about a particular agent, its latency and its risks to human health? When and by whom? Of course such matters go to questions of duty and breach, but they may not be irrelevant to causation to the extent that the known factual experience of a potentially harmful agent may throw light on causation questions.

Now it may be said that advocates and judges are well able to deal with such matters. Indeed, it might be said that such opinion evidence would not be admissible.[109] But historians are trained to evaluate and contextualise information including discrete or incomplete information, and to construct an objective picture. An historian may give a more nuanced picture. By contrast, an advocate's expertise may be to take advantage of incompleteness or uncertainty by deconstructing facts or, conversely, to take a so-called 'smoking gun'

[108] David Rosner, 'Trials and Tribulations: What Happens When Historians Enter the Court Room' (2009) 72 *Law and Contemporary Problems* 137, 138, 146 and 152.

[109] But under s 79 of the Evidence Act, if appropriate specialisation, experience and research is displayed opining on primary documents, say, decades old that are adduced as an exception to the hearsay rule, and given the latitude now permitted by s 80, there may not be a difficulty. And after all, relevant persons with first-hand knowledge may long be deceased. Now it may be said that historians are just presenters of abstracted data. But so too are accountants, whose evidence courts have little difficulty in receiving.

document and exaggerate its significance. But for an historian it is the accretion of information and development of knowledge in its historical context that is important.[110] Of course, I do not say that a judge cannot competently tread the same path as an historian. But for a judge only trial, if the evidence is admissible it may be of assistance but not definitive. Let me now turn away from the past.

C *Artificial Intelligence*

Novel causation questions are likely to arise from the use of artificial intelligence, which reflect both its embodiment and its application.

Artificial intelligence can be embodied in autonomous or non-autonomous systems. These systems can be mechanical or even biological with the use of nano-technology, although I will only focus on the mechanical.

A non-autonomous system is a computer that cannot move freely about the physical environment, and does not exert direct physical force on the external world in a relevant sense. But it may embody what is now described as machine-learning and in that sense may be described as semi-autonomous.

An autonomous system is a robot that has physical independence and may exert direct physical force or contact on the external world. It may also engage in machine learning and also acquire autonomy through a form of senses by exchanging data with its environment. As a result of that exchange it may adapt its behaviour and actions.

What do I mean by artificial intelligence? There are various possibilities. One could use a standard in terms of what humans are observed to do or acting humanly; and so the Turing test. One could adopt a functional test in terms of the modes of thinking that scientists and engineers program into their systems. But this may embrace, say, a brute force computational tool that one would not usually characterise as intelligence; the computer program designed to beat a chess master is of that variety. One could embrace within the notion of intelligence a capacity for deductive reasoning, inductive reasoning and pattern recognition, in other words thinking rationally, but leave out the concepts of consciousness or sense of self that need to be further defined and their source identified by philosophers and neuro-biologists.

[110] Rosner (n 108) 152, 153.

I will avoid definitional debates by equating artificial intelligence with machine learning. But what do I mean by the latter? In traditional computing, you have a set of inputs into a computer program prepared by the programmer, who usually programs for all conceivable options. An output is produced. Contrastingly, in machine learning the machine is given inputs and outputs to then develop its own algorithm(s). But this is simplistic. Let me deal with two types of machine learning.

First, one can have supervised learning. The machine is given a known training data set, which includes input data and labelled response values. The machine learns to build a model that can make predictions of the response values for a new data set. The machine may be given generic algorithms such as a classification algorithm, an anomaly detection algorithm, a regression algorithm and a clustering algorithm. But the model built by the machine will involve the creation of a new algorithm from unknown iterations and combinations of generic algorithms. But how it does this and the precise construct of the model produced may not be transparent.

Second, one can have unsupervised learning. The machine is given only input data without being given labelled response values. It uses the generic algorithms to produce its own algorithm that enables inferences to be drawn from the input data in support of an identified goal. As part of this, reinforcement learning may be involved, where the machine may be set a goal which maximises the cumulative reward set for it. In this context, artificial neural networks may be used.[111] On each run, if the desired maximum has not been reached, the process may be repeated using back-propagation until the maximum is reached.

Now either of these manifestations of machine learning may have problematic features. There may be biases in the input data selection from which the machine learning algorithm has been self-generated. Further, there may be biases in the selection of the generic algorithms in terms of their number and type, which selection will affect the compilation of the machine learning algorithm. Further, and problematically for ascertaining causation and allocating responsibility for an act or omission that has caused harm, there may be a lack of transparency concerning the boundaries and content of the machine learning algorithm and how it was created. So, you may observe a defective output that has caused harm,

[111] *Thaler v Commissioner of Patents* (2021) 160 IPR 72, [19]–[28] (Beach J).

but the source of the defects may not be known in the sense that how the machine produced the output may be a blackbox.

This brings me more directly to causation. The attribution of fault is usually driven by the mechanism of causation. But if the essence of machine learning is the capacity for machines to learn and to make and implement independent decisions, this can lead to unpredictable outcomes.

What happens where the defect or decision cannot be traced back to human error? Where there are machine decisions based upon deterministic computer software traceable to human agency arising from defective programming, defective input data or incorrect operation, attribution of fault including the identification of the relevant state of mind may have a ready solution.[112] But what happens when the machine learning has produced the emergence of a machine generated goal and an algorithm to achieve it? What happens where the machine has acted in an emergent, unforeseeable but autonomous way which is otherwise in conformity with its design? Who is liable for the harm? The manufacturer? The programmer? The owner of the machine? And how will causation be comprehensively analysed with the blackbox problem?

And how do we treat the machine in terms of the attribution of fault? Indeed, as Chesterman discusses, should we recognise or confer any legal personality?[113] Should we treat them as a person? As the agent of the owner? As a child of the owner? As a dangerous animal of the owner? As a tool owned by the owner? As the Romans treated their slaves? As a defective product supplied by a manufacturer or reseller?

Common law or statutory concepts of causation designed to identify who should be legally liable are based on the ready identification or attribution of human knowledge, and control of the relevant design and programming. This is the foundation used to attribute liability to the relevant legal person, including a body corporate. But such concepts may be outmoded for autonomous or semi-autonomous systems using machine learning.

[112] *Quoine Pte Ltd v B2C2 Ltd* [2020] SGCA(I) 02, [97]–[99], [124] and [125] (Menon CJ) discussing problems that had occurred with algorithmic trading involving cryptocurrency, and on the defence of unilateral mistake, identifying the state of mind as to knowledge of the mistake to be that of the programmer of the relevant program at the time it was written.

[113] Simon Chesterman, 'Artificial Intelligence and the Limits of Legal Personality' (2020) 69 *International and Comparative Law Quarterly* 819, 825–827.

What happens when an element of fault requires establishing a state of mind? For a claim against a natural person there is no conceptual difficulty. For a claim against an artificial legal person such as a corporation, rules have been developed involving an attribution of the state of mind of one or more individuals to the corporation, alternatively imposing vicarious liability.

But take a corporation where the knowledge resides in an artificial intelligence system rather than one or more employees. Say the relevant 'thinking' has been done by a system without anthropomorphising its algorithms. Further, suppose the system generated the thinking in an autonomous or semi-autonomous way. How do you assess the state of mind of the corporation? There is much to be said for Diamantis' extended mind thesis.[114] He cogently argues that we should treat the notion of the corporate mind, which in any event is artificial since a corporation has 'no soul to damn, no body to kick',[115] as extending to algorithms that now fulfil rules in substitution for human employees or other agents.

I have raised more questions that I have solved. But then have I not mimicked what energises the scientific method? Let me turn to the final topic.

D *COVID-19 Vaccines*

There may be litigation concerning COVID-19 vaccines against manufacturers or suppliers, although I note that a no fault COVID-19 indemnity scheme was recently announced by the Commonwealth Government to provide claimants with an administrative option to seek compensation rather than via court processes concerning vaccines approved by the Therapeutic Goods Administration.[116]

In Australia there are no relevant statutory immunities available to the manufacturers or health care professionals who may supply or prescribe such vaccines. There may be Commonwealth indemnity schemes, but that does not prevent beneficiaries of such schemes from being sued by those who have suffered harm. Such beneficiaries could be liable in tort or under the Australian

[114] Mihailis Diamantis, 'The Extended Corporate Mind: When Corporations use AI to Break the Law' (2020) 98 *North Carolina Law Review* 893, 900.
[115] Apparently the words of Lord Thurlow in 1778; see *Australian Securities and Investments Commission v Westpac Banking Corporation* (2018) 127 ACSR 110, [1659] (Beach J).
[116] See the 28 August 2021 media announcement by the Minister of Health.

Consumer Law for supplying a defective product or for failing to advise or being misleading concerning the relevant risks.

Issues may arise as to whether benefits have been oversold, such as marketing relative risk reduction benefits rather than absolute risk reduction benefits.[117] Issues may arise as to whether various risks have not been disclosed. Let me speak hypothetically. If any vaccine directly consists of or can indirectly produce the SARS-CoV-2 spike protein or an analogue that is then used as the antigen to provoke an adaptive immune system response, and questions are raised as to whether such proteins separately[118] impair endothelial function or generally elicit cell signalling or have implications for auto-immune diseases, non-disclosure of such risks may have legal consequences.

Interesting causation questions may arise if someone suffers harm as a result of taking a vaccine.

First, take the case where risks have been inadequately disclosed. Would the person have taken the vaccine anyway if there had been full disclosure? That is a standard question to ask in negligence cases involving medical treatment scenarios.

Second, has it been shown that the vaccines can cause harm? And if so, to what class of person? And did it cause the particular plaintiff's harm? Did it materially contribute to the injury? Or did it just materially increase the risk of injury? And what is the relevance and effect of co-morbidities on such questions?

Third, to what extent does one consider the counterfactual that if the vaccine had not been taken, the plaintiff could have been infected with SARS-CoV-2? What is the likelihood of that? And if they had been so infected, would they have suffered an injury if only asymptomatic? Further, what was the chance of suffering clinical observable symptoms and so an injury? And would this injury have been more serious than the injury caused by the vaccine? And what about the case where the injuries would have been different? Generally, would the plaintiff have been in an equal or worse position by not taking the vaccine?

Fourth, if the vaccine never had the promise of avoiding an infection but only had the promise to reduce the likelihood of infection, what was the degree of

[117] Ronald Brown, 'Outcome Reporting Bias in COVID-19 mRNA Vaccine Clinical Trials' (2021) 57(3) *Medicina* 199.

[118] In other words, the spike protein does not just have the function of allowing the virus to bind to the ACE receptors of a host cell. It may in this hypothetical case have a separate detrimental effect.

reduction? And how does that play out in assessing the counterfactual? Analogous questions arise if the vaccine only had the promise of reducing the severity of the symptoms of COVID-19, rather than avoiding illness.

One can see that these factual questions involve causal chains with compounding probabilities, uncertainties and different risk calculus. But there are also broader legal questions.

Should special rules of proof be created for causation? Should there be a rebuttable presumption or reverse onus of proof in cases where a plaintiff has taken a vaccine and suffered a particular type of harm, where it is accepted that the vaccine in question could cause that type of harm? And should you do away with the counterfactual by not inquiring into what would have happened if the vaccine had not been taken? So you just compensate for the injury if it occurs, rather than engaging in a 'but for' analysis.

Further, what is the relevance of Governmental coercion or encouragement to take a particular vaccine to the question of causation? Clearly, the consequential increased vulnerability and reduced autonomy would enhance arguments for a duty of care.

And what is the relevance of Governmental coercion forcing a manufacturer to supply a vaccine or a health care professional to provide one? There may be a right of indemnity in their favour. Further, one can envisage possible defences to a suit at the hands of a victim, depending upon the nature of the cause of action. But no additional interesting causation question is likely to arise on a victim's claim.

Let me make one other point that seems topical in the context of COVID-19, albeit that its context relates to future risk assessment rather than any inquiry as to historic cause.

Debate about the relevant science is sometimes sacrificed on the altar of the precautionary principle,[119] which is not a scientific question but a public policy reaction. Simply put, it says that one should take a cautious approach where the science is uncertain and there is a risk of harm. Its allure may arise in the context

[119] This is not always the case. In *Re Dow Chemical (Australia) Ltd and Director, Chemicals Notification and Assessment* (1999) 58 ALD 179, [13], the Administrative Appeals Tribunal (Cth) was persuaded to temper such a vice when considering the carcinogenicity and mutagenicity of trichloroethylene.

of regulatory action or litigation where likely future causation and risk questions arise.

Such a putative protective coating is usually employed where there are competing positions on the science and it is easier to delegate the decision of deciding between them by adopting such a default setting. But usually when you see an analysis justifying taking step A to deal with risk X to avoid consequence Y by applying the precautionary principle, you do not see any analysis of taking lesser steps B, C and so on and their impact on X or Y or both. Nor do you see any analysis of not taking steps A, B, C and so on and the effect of non-action on X or Y or both. But all of this should be part of the decision matrix. Further, in the context of say a health crisis, Y is not only to be seen through the window of direct health consequences, but also through the economic impacts of acting or not acting that may produce even more long term and wide-spread ripple effects such as poverty, mental health issues and delays in other medical diagnoses, which may produce consequences several orders of magnitude above the direct health effects.

Of course, science may be uncertain and knowledge of risk may be inadequate, such that the magnitude of potential harm and the probability of its occurrence may not be able to be usefully calculated.[120] But the application of the varnish of the precautionary principle should not be the first resort to grappling with uncertainty.

IV Conclusion

I have set out some epistemic themes that trial judges might consider bearing in mind when assessing scientific evidence relevant to causation. Moreover, with new technical advances and consequently new causation questions arising, applying such a lens might be said to be an imperative. Of course, judges differ in their degree of technical literacy, but none should lack the capacity and motivation to come to grips with the competing scientific evidence before them.

[120] David L Bazelon, 'Science and Uncertainty: A Jurist's View' (1981) 5(2) *Harvard Environmental Law Review* 209. He also rightly emphasised the distinction between risk disclosure and risk taking. Autonomy of choice in the latter requires transparency in the former. Further, in many cases, a buffer or element of conservatism may be added to the former before the latter is justified.

I will conclude by reinforcing my perspective against the gentle chiding by Brennan and Carter[121] of Anglo-American judges. Some of us who have applied a 'but for' analysis have been labelled as disciples of corpuscularianism, that is, devotees of Newtonian mechanics where particulate motion and collision is said to be the source of causation and open ended probabilistic concepts are treated as second-class notions. And so they say:

> More explicitly, there are three facets of mechanistic science that appeal to the legal 'common sense.' First, since mechanics fits a deductive model, mechanistic reasoning is capable of identifying one liable individual act within the causal chain of 'but for' events: a single encompassing 'but for' cause. Second, since probability and statistics are second-rate forms of reasoning for a corpuscularian, a corpuscularian judge need not consider probabilistic aspects of cause which arise in scientific evidence. Third, a mechanistic or corpuscularian concept of science allows a judge to procrastinate by demanding more complete evidence when faced with any amount of scientific uncertainty.

This is entertaining stuff. But in defence of my colleagues, I would venture to suggest that judges well appreciate multiple causes and probabilistic treatments. And where they do single out a particular cause, they do so ultimately as a normative value judgment based upon assigning legal responsibility rather than just an application of physics. And as to their last point, judges do not have the luxury of waiting, and I do not wish otherwise.

[121] Troyen Brennan and Robert Carter, 'Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease in Individuals' (1985) 10(1) *Journal of Health Politics, Policy and Law* 33, 36, 53.