3-2011

A Common Law View of Causation, Science and Statistical Evidence in the Courtroom

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Case Note

A COMMON LAW VIEW OF CAUSATION, SCIENCE AND STATISTICAL EVIDENCE IN THE COURTROOM

Amaca Pty Ltd v Ellis
[2010] HCA 5

In March 2010, the Australian High Court in *Amaca Ltd v Ellis* [2010] HCA 5 ("*Amaca*") moved assertively to clarify the approach of the Australian courts to causation in cases of lung disease involving multiple pathogens. The court demonstrated sensitivity to both the scientific and legal inquiries while reaffirming the obligation of plaintiffs to prove causation based on the balance of probabilities. In examining the plaintiff's statistical evidence, the court established important guideposts regarding the proper use and interpretation of epidemiology in the courtroom, highlighting both the relevance and limits of such proof regarding causation and the satisfaction of the plaintiff's evidentiary burden. While *Amaca* dealt with lung cancer, asbestos and cigarette smoking, the court's careful approach to the statistical evidence and reaffirmation of the common law standard of "but for" causation are likely to resonate beyond the asbestos field to cases involving other complex diseases arising from a range of low-level occupational and environmental exposures. The High Court has established a practical and useful road map for the manner in which courts should integrate scientific proof into the inquiry while preserving the fundamental aspects and related application of the common law doctrine of causation.

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[*] The author wishes to acknowledge the valuable research assistance of Ms Stefanie Chan Yuet Ling, LLB candidate (class of 2011), School of Law, Singapore Management University.

I. Introduction

1 In *Amaca Pty Ltd v Ellis as Executor of the Estate of Cotton (deceased); South Australia v Ellis; Millenium Inorganic Chemicals Ltd v Ellis*[1] ("*Amaca*"), a decision rendered by the Australian High Court on 3 March 2010, the justices moved assertively, and, indeed, unanimously to reaffirm the relevance and precise application of the common law "but for" standard for determining causation in cases of complex multifactorial pathogenesis. The case, which involved an individual's development of lung cancer following the cumulative exposure to asbestos and cigarette smoke, is noteworthy to the extent that it (a) clarifies the standard of causation in such cases within Australia's federal system; (b) underscores the importance of examining and interpreting the scientific evidence adduced; (c) highlights the challenges, potential and limits of the dialogue between the medical and legal spheres; and (d) suggests a more conservative or traditional approach to the issue of causation, where diffuse risks cannot be definitively quantified or parsed. The exposures brought about by industrial society may be many and varied, but the justices in *Amaca* have now reaffirmed that the complexity and overlapping nature of these exposures cannot be used indiscriminately to relax the evidentiary burden where causation is at issue. *Amaca* is likely to resonate beyond the asbestos field as courts in Australia and elsewhere deal with diffuse, but low-level risks arising from rapid growth and the development of new industries.

II. Facts

2 Paul Cotton was a regular cigarette smoker of between 15 to 20 cigarettes *per* day for 26 years. He was also exposed to asbestos during two periods of employment, one with the Engineering Water Supply Department of the State of South Australia from 1975 to 1978, where he worked with asbestos-lined pipes manufactured by Amaca (formerly James Hardie & Coy Pty Ltd), and later in his work with Millennium from 1990 until his death from lung cancer in 2002.

3 Mr Cotton's executrix secured a favourable judgment against his two employers and Amaca at the trial court level in the Supreme Court of Western Australia and later prevailed on appeal in a majority opinion issued by the West Australian Court of Appeal, with Martin CJ dissenting.

1 [2010] HCA 5.

III. Issue

4 Amaca and the other original defendants were granted special leave to appeal to the High Court on the issue of causation. Issues of breach of duty and foreseeability of injury were not in question. Accordingly, the question at issue can be framed as whether, in the presence of a breach of duty where injury was reasonably foreseeable, the balance of probabilities concerning causation justified a finding that Amaca's specific breach and/or that of the other defendants led to the plaintiff's illness?

5 How should the court interpret the causative role of the decedent's exposure to respirable asbestos, an acknowledged risk factor for lung cancer, where the plaintiff's history also revealed a long and substantial history of cigarette smoking also closely associated with the pathology at issue? The case therefore essentially involved the analysis of a complex multifactorial, or synergistic, disease process within a legal setting.

6 How does one assess and parse risk in such a situation and can liability be assessed and apportioned before causation itself is conclusively proved or otherwise plausibly established? To what extent can the court infer causation? It is the answer to these questions that highlights the difference between the nature of the scientific and the legal inquiries and that further yields the relatively "bright line" test of "but for" causation that separates the two.

IV. Applying the standard and interpreting scientific evidence in the courtroom – Causation and material contribution

7 The history of *Amaca*[2] through the Australian court system highlights the collision, or evolving co-existence, of legal concepts with scientific, probabilistic analysis. While the case nominally dealt with the application of the "but for" or balance of probabilities standard to determine causation, it is clear that the disparate interpretation of the scientific evidence by the trial, appellate and high courts conditioned the interpretation and application of the relevant standard. The courts' understanding of the pathology and related interpretation of the scientific evidence adduced in support of the plaintiff's claim resulted in the operational application of different tests at the trial and appellate levels as compared to that of the High Court.

8 In cases of complex disease processes involving lung carcinogens, Australian courts in certain states had witnessed a qualified

2 *Amaca Ltd v Ellis* [2010] HCA 5.

relaxation of the standard of proof, relying, *inter alia*, on the House of Lords decision in *Bonnington Castings Ltd v Wardlaw*[3] ("*Bonnington Castings*"). With regard to complex disease processes involving lung pathology, the court in that case asked whether the plaintiff's exposure to silica dust was a cause or otherwise made a material contribution to the plaintiff's development of pneumoconiosis, adding that any exposure above a *de minimis* level could be deemed to be material.[4] In *Bonnington Castings*, the plaintiff's exposure to silica dust, a principal cause of pneumoconiosis, came from two sources, a pneumatic hammer and swing grinders, only one of which involved a breach of duty potentially triggering legal liability. Moreover, it was well established as a medical matter that the pathology develops over time from the cumulative exposure to silica dust. Accordingly, in a case where (a) silica's role as a cause was well accepted and (b) the cumulative nature of the disease process was understood, the court found it logically and legally plausible to ascribe legal liability to the defendant whose breach of duty exposed the plaintiff to only one source of the contaminant.

9 As a predicate to the analysis of *Amaca*,[5] it is important to note the following. The plaintiff's exposure in *Bonnington Castings*[6] dealt with two sources of exposure to the same contaminant. Silica's role as a cause of the pathology in question was beyond medical question. Accordingly, there was no issue concerning the point at which to draw a causal inference from statistical data. The role of cumulative exposure was also well established though here there was perhaps a question as to what constituted a material contribution. Given the strong causal connection between silica and the development of pneumoconiosis, the court felt justified in adopting the principle that any exposure above a *de minimis* level could be deemed to be a material contribution. It is important to note, however, that material contribution was not used to establish causation *per se*, but rather to define the level at which legal responsibility would be triggered, the causative role of silica as a medical matter having already been established. Material contribution was therefore used to inform and clarify the effects and application of a finding of causation (*ie*, to establish the scope of legal responsibility), not to substitute for proof of same.

3 [1956] 1 AC 613.
4 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613 at 621, *per* Lord Keith.
5 *Amaca Ltd v Ellis* [2010] HCA 5.
6 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.

V. *Amaca* distinguished and clarified

10 *Amaca*[7] constituted an opportunity for the court to clarify the relevant standard in cases of complex lung pathogenesis. It raised considerations that resonated with those of *Bonnington Castings*,[8] including a potentially deserving plaintiff, work place exposure, a complex disease process, and the interpretation of scientific evidence in the courtroom.

11 It did not, however, constitute a situation involving exposure to a single lung pathogen known to "cause" the condition at issue. Nor was the exposure to the pathogens at issue solely the product of the work place. Rather, *Amaca*[9] concerned the plaintiff's exposure to two different lung pathogens, cigarette smoke and asbestos, the former of which was voluntary and the latter "imposed" from the plaintiff's perspective due to the conditions of the work place. Moreover, the cumulative exposure to a single pathogen was not the sole issue, but rather the separate and combined exposure of the plaintiff to two different pathogens constituted the focus of the inquiry.

12 The factual situation in *Amaca*[10] was therefore decidedly more complex than the one at issue in *Bonnington Castings*[11] as both a medical and legal matter. There were more permutations or causal scenarios to separate and analyse than in *Bonnington Castings*. Moreover, once the inquiry began in earnest, it was clear that epidemiology would play a significant role. The plaintiff brought at least six scientific experts to bear on the case, several of whom "had longstanding interest and experience in the study of asbestos-related diseases".[12] Although the plaintiff's experts developed probabilities for asbestos alone causing the decedent's lung cancer, smoking alone causing it, and some combination of the two causing it, the trial court took a broad-brush approach to the epidemiological evidence, stating essentially that if each carcinogen posed a risk then it was virtually axiomatic that the two acting together would result in some type of synergistic or cumulative impact of the two substances, acting in concert. The trial judge therefore moved directly to the issue of causation, framing the plaintiff's burden in a manner echoing *Bonnington Castings* as:[13]

> If the evidence establishes that it is more probable than not that Mr Cotton's lung cancer was caused by asbestos arising from one or

7 *Amaca Ltd v Ellis* [2010] HCA 5.
8 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.
9 *Amaca Ltd v Ellis* [2010] HCA 5.
10 *Amaca Ltd v Ellis* [2010] HCA 5.
11 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.
12 *Amaca Ltd v Ellis* [2010] HCA 5 at [17].
13 *Ellis v The State of South Australia* [2006] WASC 270 at [641].

> both periods of occupational exposure to that mineral or if it supports the conclusion, on the probabilities, that his cancer was caused to a material extent by the combined effects of his periods of asbestos exposure with the effects of his chronic smoking [then the plaintiff would succeed].

13 As the High Court pointed out, no attempt was made to delineate among the defendants in terms of their respective legal responsibility, if any, arising from the plaintiff's exposure to asbestos and subsequent development of lung cancer. Moreover, the trial court's approach to the epidemiology was summary, essentially amounting to the proposition that if both asbestos and cigarette-smoking were known lung carcinogens, then it was logical to infer that Mr Cotton's condition was the result of a synergistic or cumulative interaction between the two in which asbestos had made a not insignificant, read "material", contribution. The trial judge reaffirmed this view by stating in language highlighted by the High Court that:[14]

> [T]here was 'really no answer to the evidence of Professors Musk, Wan, Dr Kendall, Professor de Klerk, Dr Leigh, and Professor Berry that a not insignificant contribution to the combined causative effect was due to this asbestos exposure'. The conclusion that both periods of occupational exposure to asbestos made a 'not insignificant' or 'material' contribution to the onset of the development of Mr Cotton's cancer (or both its onset or development) was said to be supported by 'the probabilities [being] that the toxic effect of the carcinogens of tobacco and asbestos had synergistic effect, and that their effects were also cumulative upon previous exposures'.

14 As the disposition of the case by the High Court demonstrates, it does, indeed, appear that there was an answer to the plaintiff's experts or at least an alternative interpretation of their findings. Before turning to the High Court's reasoning, however, it is necessary to review certain fundamentals of epidemiology and also the manner in which this evidence was subsequently treated (or not) by the Court of Appeal in upholding the trial court ruling.

VI. Epidemiology and the legal standard of causation

15 As has been seen, the trial judge in *Amaca*[15] tended to view Mr Cotton's environment and circumstances in the aggregate. Mr Cotton incurred various work place exposures, together with others arising from his personal habits, in the context of a complex disease process that certain experts had described as synergistic. In the trial judge's view, it was therefore legally sufficient in ascribing liability to

14 *Ellis v The State of South Australia* [2006] WASC 270 at [689].

15 *Ellis v The State of South Australia* [2006] WASC 270.

each of the defendants to describe how Mr Cotton's cancer might generally have been caused rather than to establish the specific cause as a function of each defendant's actions, and, indeed, of Mr Cotton's own actions as a smoker. A clinical review of Mr Cotton's medical case and risk profile was substituted for a legal analysis of specific considerations of duty, breach and causation. Moreover, this process was aided by the judge's substitution of the "material contribution" test for a situation in which strict adherence to the "but for" standard of causation was more apposite. In an effort to avoid "rough" justice, the judge had come up with a "rough" estimate, construing the uncertainty against the defendants and, conversely, drawing positive inferences regarding causation for the plaintiff.

16 It is to the issue of estimates that we now turn. When one enters the field of epidemiology, it is important to realise that we are dealing with just that – estimates, with some results being more robust or probative than others. Stated succinctly, epidemiology deals with the collection, collation and statistical processing of observational data concerning the disease incidence in human populations between exposed and unexposed groups in respect of a given factor or factors. In comparing the disease incidence between exposed and non-exposed groups, epidemiology yields a figure known as relative risk, which describes the probability of the risk in the exposed group as compared to the unexposed group.[16]

17 Translating a relative risk into a legal context can, however, be a tricky matter as the parameters and nature of the inquiries differ. From a numerical standpoint, a relative risk of less than 1 does not describe a meaningful association as it suggests that there is no difference between the exposed and unexposed group in terms of disease incidence. Relative risks between 1 and 2 are deemed to be statistically significant, but evidence of only a weak association. Finally, statistical relative risks of more than 2 have assumed a certain status in the courtroom as they suggest that the risk of the exposed group in contracting the pathology at issue is twice as great as that of the unexposed group. Moreover, the notion of a 2 to 1 ratio seems to accord nicely, on the surface at least, with civil litigation standards of the "preponderance of the evidence" and its corollary of "more likely than not".[17] However, it should be noted

16 Michael D Green, D Michal Freedman & Leon Gordis, "Reference Guide on Epidemiology" in *Reference Manual on Scientific Evidence* (J Cecil gen ed) (Federal Judicial Center, 2nd Ed, 2000) at pp 363–369, 389, 395.

17 *Siharath v Sandoz Pharmaceutical Corporation* 131 F Supp 2d 1347 (ND Ga, 2001). See also *Seltsam Pty Ltd v McGuiness* [2000] NSWCA 29 at [137]. For varying interpretations by the UK courts of the significance of a relative risk of 2 or more for establishing legal liability, see *Novartis v Grimsby* [2007] EWCA Civ 1261 and *Sienkiewicz v Greif(UK) Ltd* [2009] EWCA Civ 1159; [2010] 2 WLR 951. The author is grateful to an anonymous reviewer for highlighting these two cases.

that even a 2 to 1 ratio is at the lower bound of what constitutes a meaningful association from the scientific perspective.[18]

18 With regard to the nature of the scientific, as compared to the legal, inquiry, it is important to note that the former seeks to establish associations, describing what could have caused a given pathology. Moreover, from a scientific point of view, an association is not tantamount to causation *per se*. Through observational data, scientists begin to establish probabilities that are related to certain risk factors. Epidemiology does not shed light on the mechanism of the disease etiology or process nor does it establish causation *per se*. However, as a statistical association between a given risk factor and a given disease becomes greater and more robust in terms of the magnitude of the relative risk, there will be a point at which a causal inference can be drawn. Scientists differ over this threshold, a point which fuelled the controversy over lung cancer and tobacco smoking for many years, but in any event, the epidemiological threshold for a finding of "causation" is generally above the relative risk ("RR") of 2, that has become a type of gold standard for the courts.[19]

19 When more than one risk factor is in operation in a so-called multifactorial disease, such as lung cancer, the situation becomes complex and the differences between the nature of the scientific and legal inquires even starker. In a multifactorial setting, the scientific inquiry will seek to evaluate the nature of the risk from each factor independently and/or to assess whether there is a significant interaction or synergy between the two in the onset of the pathology. The inquiry is geared toward gaining a better understanding of the disease and the role of various risk factors within a constellation of environmental and lifestyle factors. The goal is therefore to gain increased understanding of the disease over time rather than to arrive at a final conclusion concerning cause or causation within a specific context. The legal inquiry, of course, seeks a definitive answer to a specific question within a fixed time frame, using scientific data that have evolved in a distinctly different intellectual context and field of inquiry. It is this interaction, a conflict of contexts or paradigms, that renders the competent handling of epidemiological evidence in the courtroom so difficult.

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18 Wayne Roth-Nelson & Kathey Verdeal, "Risk Evidence in Toxic Torts" (1995-6) 2 Envtl Law 405 at 425–426. See also Basil C Bitas, "Probability in the Courtroom" in *Handbook of Probability: Theory and Applications* (Tamás Rudas gen ed) (Sage Publications, 1st Ed, 2008) at p 429.

19 Basil C Bitas, "Probability in the Courtroom" in *Handbook of Probability: Theory and Applications* (Tamás Rudas gen ed) (Sage Publications, 1st Ed, 2008) at p 429. See also Wayne Roth-Nelson & Kathey Verdeal, "Risk Evidence in Toxic Torts" (1995–6) 2 Envtl Law 405 at 425–426.

VII. Courts and scientific evidence

20 In *Bonnington Castings*,[20] a statistical approach was not necessary as it was well established from both a medical and "common sense" perspective that silica dust could both cause and aggravate the condition known as pneumoconiosis. Causation had been established as a medical and biological fact. However, in *Amaca*, epidemiology and statistics were required at the trial court level[21] to support the speculative conclusions or causal inferences concerning the relationship between two different carcinogens alleged to function both independently and cumulatively in the onset of the disease. Epidemiology was necessary to fill a gap in the corpus of direct biological proof. As has been seen, the trial court took a broad-brush approach to the epidemiology, taking the expert opinion regarding the possible cumulative effect of the two risk factors at face value and applying a watered down standard of "material contribution" to find each of the defendants liable.

A. Court of Appeal – Accepting the trial court findings without reference to the epidemiology

21 The Court of Appeal's approach to the interpretation of scientific evidence in the context of legal causation harkened back to that of *Bonnington Castings*,[22] albeit in a factual context that was far more complex. The issue with which the House of Lords in *Bonnington Castings* and the trial and appellate courts in *Amaca*[23] were essentially grappling was the notion of causation regarding a so-called indivisible disease potentially brought about by at least two separate sources of exposure to a potentially causative agent. Again, *Amaca* introduced an additional element of complexity as it dealt with multiple sources of exposure and two different agents. Where the state of medical knowledge was such that an exposure at issue could be considered to be a general cause of the disease, even if not established to be the sole cause in the particular case, Australian courts had been inclined to let plaintiffs jump the "evidential gap"[24] by making a causal inference at least in cases where exposure to the same causative agent, albeit from two different sources, was involved. The *Amaca* trial court extended this reasoning to a situation where exposure to two different causative agents was at issue. Rather than disregard the epidemiology, the court took the view that the statistical evidence, coupled with expert opinion, having established the synergistic nature of the disease where two causes act

20 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.
21 *Ellis v The State of South Australia* [2006] WASC 270.
22 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.
23 *Ellis v The State of South Australia* [2006] WASC 270; *The State of South Australia v Ellis* [2008] WASCA 200.
24 *Amaca Pty Ltd v Hannel* [2007] WASCA 158 at [420].

interdependently and cumulatively to produce the pathology, it was appropriate to consider causation as established and to move to the lesser standard of "material contribution".

22 The majority opinion in the Court of Appeal took a somewhat more radical approach to the epidemiological evidence. Accepting the trial judge's finding that "the medical evidence reflected in the acknowledged synergistic effect of tobacco smoke and asbestos [showed] that tobacco smoke and asbestos fibers operated interdependently and thus cumulatively to cause Mr Cotton's lung cancer", the majority concluded that it was no longer necessary or appropriate to investigate the role of the asbestos and cigarette smoke as independent causative agents even if the plaintiff had adduced proof to this effect.[25]

> Once it is accepted that smoking and asbestos are not independent, but rather cumulative causes of lung cancer, the epidemiological evidence has no direct application to the question of causation at law because it is based on a false assumption. Once it is determined that tobacco smoking and all asbestos exposures operated cumulatively, the only remaining issue is whether each asbestos exposure made a material contribution.

23 The Court of Appeal was essentially stating that direct proof in the form of expert opinion, particularly from that of Dr Leigh, the only specialist physician and epidemiologist among the plaintiff's cadre of expert witnesses, rendered the epidemiological analysis moot or at least irrelevant. In a manner similar to the trial court, but in more unequivocal terms, the Court of Appeal was asserting that a finding of synergy trumped any specific numbers. In contrast, the trial court had seemed to suggest that the numbers, together with expert opinion, suggested a certain synergy. Both courts arrived at the lower standard of "material contribution", but with different levels of deference to the specific statistical findings. The trial court relied on a general inference of causation based on its interpretation of the aggregate findings, thereby giving rise to application of the lower standard. The Court of Appeal relied on what it perceived to be conclusive expert assertions concerning the nature of the disease, thereby giving rise to application of the lower standard and the consequent disregard of the specific statistical findings. Indeed, the Court of Appeal appeared to be treating as "medical" fact a situation that was actually rife with uncertainty.[26] Their approach reflected a wholesale and inapposite use of *Bonnington Castings*,[27] treating the science as established without regard to the statistical findings in a situation that was susceptible to many different

25 *The State of South Australia v Ellis* [2008] WASCA 200 at [336].
26 *The State of South Australia v Ellis* [2008] WASCA 200 at [319] and [336].
27 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.

interpretations due to the complexity, nature and number of exposures at issue.

B. The High Court – A return to the "but for" standard

24 The High Court sought to parse the approaches at the trial and appeals court levels by distinguishing between direct biological proof as reflected in accepted medical knowledge or expert opinion and general causal inferences drawn from aggregate, statistical proof and by further highlighting the role of specific causation, that is the link to the plaintiff, within that mix. Behind the High Court's analysis was a desire to delineate not only among the responsibility of the respective defendants but also a desire to limit application of the material contribution standard to cases involving multiple exposures to a single, known pathogen emanating from different sources. Again, *Amaca* dealt with multiple exposures to asbestos from two different workplace situations, together with a separate non-workplace exposure to cigarette smoke. In looking at the evidence adduced at trial, the High Court first asked whether causation had been established by direct proof as reflected in specific biological evidence and expert opinion, concluding that none of the experts, including Dr Leigh, the only specialist physician and epidemiologist in the group, had adopted the position that smoking and asbestos must work together to cause cancer.[28]

25 With the causal connection not having been satisfied by direct proof or expert testimony, the High Court then explored the strength of the causal inference that could be drawn from the epidemiology. This type of thoroughgoing exploration of the issue of causation marked the High Court's moving back to the strict application of the "but for" standard.

26 Looking at the epidemiology, the court noted that the relative risks established by the experts for contracting lung cancer from smoking were 7.7, 20, 15 and 8 respectively. It is worth noting that these thresholds were all well above the relative risk of 2, that is twice as more likely, often seen by courts as an important threshold in establishing "but-for" causation. The relative risks established by the experts for the risk of contracting lung cancer from exposure to asbestos were 1.3, 1.1 to 1.2, 1.1 and 1.16, placing these figures in the statistical nether land between 1 and 2 where the association is weak at best. Using these relative risk figures, the experts went on to assign probabilities to Mr Cotton's condition being caused by smoking alone, by asbestos alone or by the interaction of the two. With regard to the former, no estimate was lower than 67%. For asbestos, no estimate was higher than 23%.

28 *Amaca Ltd v Ellis* [2010] HCA 5 at [53].

With regard to the combined exposure to both, the highest figure was 20%. The expert consensus was that the risk of contracting lung cancer from cigarette smoking was many times higher than the one arising from asbestos. Moreover, on the issue of the proposed synergy, the highest figure was many times lower than the similar probabilities ascribed to smoking alone.[29]

27 The High Court placed emphasis on the interpretation of Dr Leigh's expert opinion wherein he stated that "while the precise mechanism of interaction between asbestos and tobacco smoke in causing lung cancer is not known, it is not possible in my view to separate their effects in the individual case when both have acted and it is thus more probable than not that in this situation, the lung cancer was the singular result of the two factors acting together. It is, however, true that exposure to either factor alone is capable of causing lung cancer."[30]

28 Given the probabilities in question, including the much lower values ascribed to asbestos, and, indeed, Dr Leigh's admission that the mechanism of interaction was not known and that either factor alone could cause lung cancer, the High Court found that the threshold for drawing a causal inference was not met. Moreover, in making this point, the court also touched on the issue of general versus specific causation, noting that the plaintiff had confined her argument to a discussion of asbestos and tobacco smoke whereas the universe of potential risk factors could be much greater. As the High Court put it: "But if conclusions are to be drawn from population studies, all of the results obtained for all possible causes of the cancer must be considered. To consider whether one of two circumstances is more dangerous than another must not be permitted to obscure examination of the relative danger of all causes."[31] The court therefore took issue with the scope of the inquiry and its potential relationship to Mr Cotton's illness.

29 On the issue of the use of epidemiological studies and specific causation, the court added that: "To draw an inference about causation from what was established by the epidemiological studies, it would be necessary to decide whether the particular case under consideration should be treated as conforming to the pattern described by the epidemiological studies … Absent evidence which suggests that the individual may stand apart from the ordinary, there may be sufficient reason to assume conformity, but whether or not this is so, it is important to recognize that the first step must be taken, if an inference is to be drawn from epidemiological studies …"[32] Then to close the

29 *Amaca Ltd v Ellis* [2010] HCA 5 at [29]–[30].
30 *Ellis v The State of South Australia* [2006] WASC 270 at [354].
31 *Amaca Ltd v Ellis* [2010] HCA 5 at [56].
32 *Amaca Ltd v Ellis* [2010] HCA 5 at [62].

circle, the High Court added that even if the general studies could be linked to Mr Cotton's circumstances, the probabilities reflected in the epidemiology were insufficient to support a finding of causation. The "material contribution" standard for synergistic disease did not enter into the analysis because there was simply no foundational evidence regarding causation.

30 The High Court moved assertively to clarify the approach to causation, the scope and application of the "but-for" versus "material contribution" standard, and the role of direct biological proof, expert opinion and statistical epidemiological evidence in satisfying the plaintiff's evidentiary burden. The effect of this exercise was to reinvigorate the application of the "but for" test even in complex cases of medical causation, thereby arresting any further drift toward the lower standard of "material contribution". *Amaca* offered the High Court a chance to reaffirm and extend the application of *Bonnington Castings*[33] and the "material contribution" standard to cases involving a plaintiff's multiple exposures to different pathogens. This the court most resolutely refused to do. Distinguishing this case from, and criticising the plaintiff's reliance on, *Bonnington Castings*, the court stated:[34]

> The issue in *Bonnington Castings* was whether one source of an injurious substance contributed to a gradual accumulation of dust that resulted in disease. The issue here is whether one substance that can cause injury did cause injury. Or, to adopt and adapt what Starke J said in *Adelaide Stevedoring Co Ltd v Frost*,[35] was Mr Cotton's cancer 'intimately connected with and contributed to' by exposure to asbestos? Questions of material contribution arise only if a connection between Mr Cotton's inhaling asbestos and his developing cancer was established.

31 The High Court's last sentence is instructive. The material contribution test is not a substitute for proof of causation, but is a means of establishing the scope of legal liability where medical causation has been established through some plausible means, whether based on recognised scientific fact, direct biological proof, conclusive expert testimony or compelling epidemiological data. However, in *Amaca*, with multiple pathogens and sources, inconclusive scientific testimony, an incomplete understanding of the so-called synergistic mechanism, and statistical data far below the threshold at which a causal

33 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.
34 *Amaca Ltd v Ellis* [2010] HCA 5 at [68].
35 (1940) 64 CLR 538 at 567; [1940] HCA 45.

inference can sometimes be drawn, there was simply no legally sufficient manner for the court to close the "evidential gap" on causation.[36]

VIII. The limits of scientific evidence in the courtroom – Policy implications of *Amaca*

32 By reaffirming the enduring vigour of "but for" causation in complex medical cases, the judgment in *Amaca* also served to clarify the nature of the legal as opposed to the scientific inquiry and how the two should perhaps intersect. In addressing the nature of the question, the High Court stated:[37]

> [D]espite this uncertainty [re the cause of Mr Cotton's cancer], the courts must, and do, 'reduce to legal certainty [a question] to which no other conclusive answer can be given'. The courts do that by asking whether it is more probable than not that X was a cause of Y. Saying only that exposure to asbestos may have been a cause of Mr Cotton's cancer is not a sufficient basis for attributing legal responsibility. Observing that a small percentage of cases of cancer were probably caused by exposure to asbestos does not identify whether an individual is one of that group. And given the small size of the percentage, the observation does not, without more, support the drawing of an inference in a particular case. The paradox, if there be one, arises from the limits of knowledge about what causes cancer.

33 The court's reasoning aptly highlights the differences between the scientific and the legal inquiry and the approach to uncertainty in each. Science seeks a better understanding over time, whereas courts must make a judgment in the immediate. The many and varied risks of modern industrial society will prompt the courts to take recourse to science in clarifying their understanding of a given issue. Established medical facts, such as that the inhaling of silica dust can cause pneumoconiosis or that the inhaling of asbestos can cause mesothelioma, a disease far more highly correlated to exposure to asbestos than is lung cancer, can assist the courts in drawing certain

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36 As to the origin of the phrase "evidential gap" in this context, see Basil C Bitas, "Probability in the Courtroom" in *Handbook of Probability: Theory and Applications* (Tamás Rudas gen ed) (Sage Publications, 1st Ed, 2008) at p 429. See also Wayne Roth-Nelson & Kathey Verdeal, "Risk Evidence in Toxic Torts" (1995–6) 2 Envtl Law 405 at 425–426. It is noteworthy that the High Court's approach appears to reflect some of the views expressed by David Hamer in "Mind the Evidential Gap: Causation and Proof in *Amaca Pty Ltd v Ellis*" [2009] 31 Syd L Rev 465, commenting on certain aspects of the lower courts' approach to *Amaca* prior to the High Court decision; see especially pp 474 (discussing the "displacement" of the "but for" test in causation) and 475 (discussing the "scope of liability"). Some foundational evidence of causation regarding exposure to the agent in question appears necessary before the material contribution standard can be applied.

37 *Amaca Ltd v Ellis* [2010] HCA 5 at [70].

conclusions about causation and, yes, material contribution where multiple exposures to the same pathogen are at issue. Indirect proof in the form of epidemiology, if parsed and evaluated for what it can and cannot demonstrate, is also a valid source of reference. However, there are limits to what the scientific inquiry can contribute, or the extent to which it can substitute for the certainty that the legal inquiry requires.

34 By refusing to extend *Bonnington Castings*[38] and, conversely, by reaffirming the importance of the "but for" test in cases involving the medical and legal causation of complex multifactorial diseases, the court reaffirmed that while science has a role to play in settling legal questions, it cannot supersede the essential nature of the legal inquiry. Specifically, where the state of scientific knowledge promotes general understanding within a sea of uncertainty, this without more will not allow the specific plaintiff to satisfy his or her specific burden. There may be situations where this circumstance will result in difficult judgments for plaintiffs. However, the alternative in which a valid, but general scientific and statistical understanding regarding the association between certain low-level risk factors and various pathologies is used to reduce evidentiary burdens and open the way to recovery, appears hardly desirable.

35 While *Amaca*[39] dealt with asbestos and cigarette smoking, the High Court's cautious approach to the role and interpretation of scientific evidence and restraint in extending *Bonnington Castings*[40] reflect a reaffirmation of common law doctrine with an eye perhaps to curbing litigation in other evolving sectors, such as cell phones and power generation and transmission, which lie on the periphery of scientific understanding regarding risk and in which low-risk epidemiology is likely to be used to "demonstrate" causation in the onset of certain multifactorial pathologies. Life is full of risk, both real and imagined. By applying the "but for" test forthrightly and without equivocation, the court in *Amaca* appears to be stating that we must have some legitimate basis that the evidence is "real", meaning legally foundational from the standpoint of causation, before we can begin to imagine.

IX. Conclusion

36 The court's decision therefore signals a more cautious, read traditional, approach to causation in cases involving statistical evidence. Weighing the rights and interests of business, consumers, workers and

38 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.
39 *Amaca Ltd v Ellis* [2010] HCA 5.
40 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.

the public at large, the court's refusal to extend *Bonnington Castings*[41] marks a reaffirmation that scientific complexity should not be used to reduce the plaintiff's evidentiary burden. There must be evidence of causation in terms of biological fact, conclusive expert opinion, plausible statistical inference or some combination thereof before any discussion of "material contribution" can come into play. Moreover, any scientific evidence of a general nature adduced in court must be demonstrated as being relevant to the plaintiff's specific circumstances to fulfil the criterion of specific causation.

37 The High Court's careful approach to the scientific evidence, questioning what constitutes medical fact, limiting the basis on which a causal inference can be drawn, and reaffirming the "but for" standard for determining causation even, or particularly, in cases involving multiple risk factors of varying severity sets out a useful guidepost for litigants that is likely to resonate beyond the asbestos field. Claims of synergy and causal inferences drawn from statistical evidence must still be subjected to legal scrutiny carried out through a distinctly legal lens. The High Court fulfilled its role with care and legal sensitivity, thereby establishing a road map for common law courts in Australia and perhaps elsewhere regarding the appropriate manner of integrating scientific proof into the courtroom while protecting the integrity of the legal inquiry.

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41 *Bonnington Castings Ltd v Wardlaw* [1956] 1 AC 613.