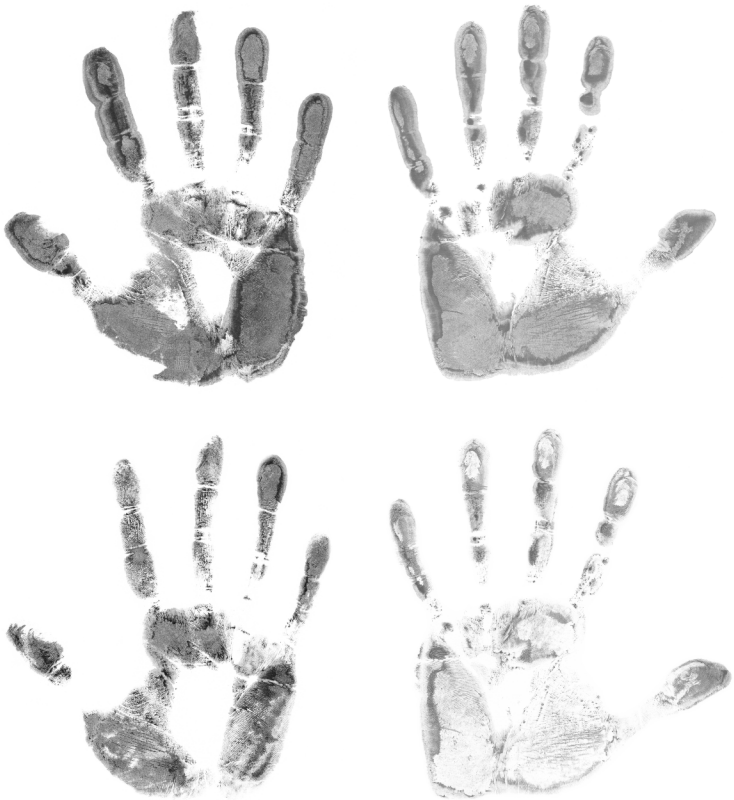
Manipulating practices

A critical physiotherapy reader

Barbara E. Gibson, David A. Nicholls, Jenny Setchell and Karen Synne Groven (eds.)



CAPPELEN DAMM AKADEMISK

CHAPTER 3

Reconceptualising causation in evidence-based physiotherapy

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Abstract

Physiotherapy practice is complex. Each interaction between a therapist and a person seeking care is unique. Physiotherapy research is aimed at providing knowledge, which can be used to inform clinical decision-making within such practice. Considering predicted therapeutic effectiveness, for example, research data should inform the process of deciding which intervention is most likely to have a causal effect on the health of the person. The growing engagement of physiotherapy practice with a framework of evidence-based practice strengthens the profession's commitment to an association between research and clinical decision-making. It would be hoped then, that the sort of causal claims arising from research methods provide precisely the sort of information needed for clinical practice, in all its complexity. This chapter presents a thesis that questions the clinical relevance of causal claims arising from our prioritised research methods. It does this on ontological grounds. The chapter proposes that the nature of causation in evidence-based physiotherapy can be understood by

the way the evidence-based framework structures itself. It then problematises this in the perspective of the complex discipline of physiotherapy with an assertion that the identified Humean nature of causation fails to relate to the context-sensitivity and complexities of a truly person-centred healthcare domain. Solutions are signalled towards a dispositional reconceptualisation of causation that would provide a more robust ontological framework on which research methods and clinical practice could be based.

Introduction

When we express what we do with patients, it is in causal terms: "this could *work* for you"; "we can help you *return* to function"; "this treatment should be *effective*", etc. These notions are increasingly influenced by a specific idea of what causation is. In an evidence-based practice framework, causation is undeniably Humean[1]. That is, causal claims derive from constantly conjoined events (observational studies) and/or from counterfactual conditions (randomised controlled trials). This is problematic, not least because many

1 This relates to a theory of causation given by Scottish philosopher David Hume (1711–1776). Hume's influence on our understanding of EBM is discussed in more detail later in this chapter. However, for now, it helps to understand the basic idea of Humean causation, which is arguably the most significant idea of causation throughout the last few centuries. His idea is something which now seems desperately intuitive: causation is nothing more than a belief based on observations of two (or more) discrete events, such that event *A* is the cause of event *B*. His famous example is of two billiard balls: Ball *A* rolls and hits Ball *B*, and we say that the rolling of Ball *A* was a cause of the rolling of Ball *B*. This entails three ideas: i) that there is *contiguity* (the two events are spatiotemporally close); ii) there is *temporal priority* (Ball *A* – the cause - always comes before Ball *B* – the effect); and iii) there is *constant conjunction* – if the experiment was repeated, we would notice the same observation to a degree when we come to believe that the rolling of Ball *A* is the *cause* of the rolling of Ball *B*. Critically, Hume claimed that there was nothing more to causation – no actual *act*, *substance*, or *mechanism*. Causation is simply one thing followed by another. The reader is directed to Mumford and Anjum (2013) for further accessible information on causal theories.

evidential elements are excluded from the causal story, for example mechanistic studies, and the patient. A Humean account of causation also paradoxically prohibits the core business of evidence-based practice: for general causal claims from research to inform single instances of clinical decision-making. This chapter argues for reconceptualisation of causation whereby causes are complex, context-sensitive and seen as dispositions that only tend towards their effect. This has the advantage of being inclusive of multiple sources of information, as well as taking the patient as the starting point to understanding what "could work for you". Thus, the core business of evidence-based physiotherapy is better satisfied.

The chapter presents an argument for why the traditional account of causation within evidence-based physiotherapy should be considered as a Humean notion. This is then problematised in relation to the central claim of evidence-based medicine (EBM)[2], which is that "evidence from study designs higher up the hierarchy more reliably informs therapeutic decisions" (La Caze, 2008, p. 361).

The notion of causation is problematised in relation to evidence-based medicine. That is, how do causal claims, established by the scientific research methods favoured by EBM, relate to individual instances of care, or indeed policy? In sum, the chapter argues that EBM presently conceptualises causation as a Humean idea, and that this is insufficient in respect of the core activity and claims of EBM. It then proposes a reconceptualisation of the nature of causation that addresses some of the fundamental challenges to the core activity of EBM. This is based on a theory of causal dispositionalism.

2 I will make no distinction between the tenets of *Evidence-based Practice* and *Evidence-based Physiotherapy* and *Evidence-based Medicine* (EBM) for the purpose of this arguments within this chapter. The scientific rationale and logical basis are the same in each framework. The vast majority of philosophical literature regarding these themes refers to EBM, and as such, this extant literature will be used for the debate regarding EBP.

Why causation?

In health care, we are interested in knowing whether a therapeutic health intervention works, that is, whether or not it causes a desired health effect. Thinking of causal relationships in this way signals what we understand of the processes that have generated sufficient knowledge to allow such a statement to be made. These processes have changed over time. Understanding what works was once a product of experience, or wisdom from a teacher perhaps. Modern health care sees these processes as insufficient and has adopted specific research methods to generate knowledge of causal relationships. Formal observation and recording of patient behaviour as a response to interventions grew into what we now know as clinical epidemiology. A segue of observational studies into multi-condition trials was seen as a significant advancement of cause-claiming research methodologies. Randomised controlled trials (RCTs) were introduced in the 1950s with claims of epistemological superiority over other methods. This claim continues today and is witnessed by explicit notions of evidential hierarchies and structures of what constitutes quality of evidence and strength of recommendations for practice. Almost parallel to the development in research methods was the re-framing of clinical epidemiology as a formal framework of EBM[3]. This movement aimed to facilitate clinical decision-making by making best use of the evidence available. The evidence in this sense was normatively suggested – as

3 When I talk about "EBM", I am talking about the *post-1991* movement. The term and reconceptualised notion of "*Evidence-based Medicine*" was first formally introduced in the field of medical epidemiology in 1991 (Guyatt, 1991). The term does actually appear sporadically throughout literature on medical statistics from the 1930s up to the 1990s. However, for the purpose of this argument, I will consider Guyatt's 1991 paper as the formalised introduction of the term.

per Sackett's[4] much quoted definition – to be multiple-sourced knowledge relevant to the clinical question:

> *Evidence based medicine is the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. The practice of evidence based medicine means integrating individual clinical expertise with the best available external clinical evidence from systematic research* (Sackett et al., 1996, p. 71).

Depending on the nature of the clinical question, "systematic research" would mean the best research for that particular question purpose. Questions of causation (i.e. does this work?) would appeal to methods of causation – that is, RCTs, or ideally systematic reviews of RCTs. Thus, research methods are an inherent and immanent part of EBM, and findings from research should be used to directly inform clinical practice.

The majority of philosophical concerns in this area have been associated with epistemological issues of research methods. Causation is explained as a product of research methods, and philosophy has aimed to understand the best methods for producing causal claims. The issue of observational epidemiological studies versus RCTs is often used as a demonstration of epistemological differentiation, (for example, Vandenbroucke, 2008). Most commonly, conclusions appeal for continued commitment to the experimental type comparative studies exemplified by RCTs. Appreciation of the ability of statistical analyses to compare group means against each other is linked to beliefs about the method's ability to differentiate correlation from causation. Observational studies can suffer from a

4 David Sackett was a founder of the contemporary EBM movement. He was founding chair of the first department of Clinical Epidemiology and Biostatistics in the world at McMaster University, Canada. During the early 1990s, he was a key member of McMaster's *Evidence Based Medicine Working Group*, a group that led the way in transitioning the seemingly stale notion of *clinical epidemiology* into a more "shop-floor" idea of *evidence-based medicine*.

lack of a comparable control group: a group so similar to the intervention group save one factor that any inferences drawn must be considered coincidental, not causal. The ability of RCTs to create and control homogenous groups and manipulate interventions appeals to the scientist. If the groups are similar save one thing (the intervention), and a between-group difference in outcome is observed, then it is argued that that difference must be due to the intervention. Causation – rather than some other association – can been claimed. Thus, when we state, "it works", we are saying something about the epistemological qualities of the research methods embedded in this framework of EBM. Despite what has just been said, I will eventually claim that EBM can be satisfied with causal claims from certain types of observational studies too, and I contend that this in fact is the key to understanding the nature of causation in EBM, as it stands.

Constraining philosophical analysis to epistemological concerns entails significant limitations. "It works" may well say something about epistemology, but it might also say something about what we understand of the nature of causation. Thus if we propose that epistemological concerns are of urgent priority to understanding best care, then ontological concerns also need to share that priority. Here is an example: I say, "it works" because I have attended to outcomes of epistemologically superior research methods (for causation at least). This exposes my appreciation of how knowledge is generated. However, I am also saying that what I mean by causation is that it is something inherently related to those methods in its nature. For example, I might genuinely believe that a causal relationship did not exist before it was *produced* by the RCT, or whatever; or I might believe that the causal relationship did always exist but I did not know of it and it took the RCT to *expose* it; or I might say that I always strongly suspected a causal relationship but the findings of the RCT better *justify* my use of the intervention in some procedural sense. However viewed, what I am saying is that the nature of causation seems to be dependent on the research methods at hand.

This then presents some problems. If evidential hierarchies are to be taken seriously, (i.e. evidence from study designs higher up the hierarchy more reliably informs therapeutic decisions), as is the normative stance, then causal ontology can be read from the hierarchy itself. That is, causation is something that is inherently related to the fact that groups are compared against each other, but not something that is part of "lower level" evidential sources. This is a clear position that exposes how health science understands causation. I will use this normative stance as the basis of the majority of the analysis to follow.

The normative stance alone is complex and challenging. However, the broad problem is exaggerated further by the descriptive stance. In reality, causal claims are, made from multiple sources of evidence that may or may not include RCTs, for example, smoking causes cancer. Nevertheless, there is a problem: health science states that causation should exclusively be the domain of certain types of studies, and causation is dependent on, and is characterised by, that epistemology. However, causal claims are made otherwise. Therefore, what causation is cannot be sustained on epistemological grounds. Further, epidemiology does not have a "fall-back" epistemological position to widen the nature of causation. It is therefore necessary to include attention towards the most fundamental aspects of an activity if progress is to be made regarding both scientific and humanistic directions. The focus of this chapter shall now be on these ontological concerns.

A Humean account of causation for EBM

On causation, Hume stated:

> *[W]e may define a cause to be an object, followed by another, and where all the objects similar to the first are followed by objects similar to the second. Or in other words where, if the first object had not been, the second never had existed.* (Hume, 1748, Sect 7, Part 1, Para 60).

This should be read in two parts: first Hume states that a cause is a form of regularity: one object regularly followed by another. He then asserts a condition that the regularity should be confirmed by the fact that the second event did not occur when the first object did not exist. This aligns to a counterfactual condition. The counterfactual account is developed by many, but perhaps David Lewis offers a comprehensive modern philosophical treatment of the conditional (Lewis, 1973a). In sum: "A $\gamma\rightarrow$ C is nonvacuously true if and only if C holds at all the closest A worlds"[5] (Lewis, 1973a, p. 561). Cartwright also represents counterfactual conditions in experimental-like trials by saying:

[I]f two groups have identical distributions, save one (T) and a probabilistic difference obtains (O occurs in "T" group only) then T is causally related to O (Cartwright, 2007, p. 46).

Accordingly, the counterfactual state (control or comparison group) is in fact the truthmaker of causation: that is the proposition cannot be true in itself – it is the counterfactual that is making it true. We can observe a series of events following each other, but we only read causation into the observation if the same regularity is absent in another condition. Causation in health science is then in some part counterfactually dependent. This is a secure position if elements below RCTs are to be considered non-evidentiary. However, as suggested, this seems not to be a favoured position and evidential claims for causation do exist in relation to non-controlled (non-counterfactual) observational studies. Therefore, if the rules of evidence differentiate controlled methods as being

5 Lewis uses the symbol "$\gamma\rightarrow$" as the counterfactual operator. For Lewis then: "*Given any two propositions A and C, we have their counterfactual A* $\gamma\rightarrow$ *C: the proposition that if A were true, then C would also be true.* ***The operation*** $\gamma\rightarrow$ ***is defined by a rule of truth****, as follows. A* $\gamma\rightarrow$ *C is true (at a world w) iff either (1) there are no possible A-worlds (in which case A* $\gamma\rightarrow$ *C is vacuous), or (2) some A-world where C holds is closer (to w) than is any A-world where C does not hold*" [emphasis added] (Lewis, 1973a, p. 560).

constitutive of causation due to their structural characteristics, but causal claims still arise from non-controlled methods, then counterfactual dependency cannot be a sufficient account for the theory of causation in healthcare nor any science.

The structure and function of observational studies requires some expansion if the essence of causation is to be considered further. For the purpose of this step, observational studies will be thought of simply as that collection of methodologies embedded in the tradition of epidemiology, in which the intention is to investigate associations between determinants of health and health outcomes. They do this by observing large groups of patients in various ways. The differentiating characteristics of observational studies from RCTs are that "investigators neither allocate patients to receive an intervention, [nor] administer an intervention" (Howick, 2011, p. 40). As such, observational studies suffer from nearly intractable problems of "confounding by indication" (Vandenbroucke, 2008, p. e67), or as Howick summarises:

> *The main problems with observational studies are that they suffer from (i) self-selection bias…(ii) allocation bias, and (iii) performance bias* (Howick, 2011, p. 40).

For the purpose of searching for their causally evidential basis, then, we can surmise that although observational studies do indeed follow-up and identify patterns of association in large groups of people over time, controlling for confounding and systematic biases are absent. The closest possible A world does not exist. How then can we read causation into these elements given the counterfactual condition asserted by Hume? In fact, Hume allowed that causation could be wholly represented in fact by adherence to three criteria: temporal priority, contiguity, and constant conjunction:

> *Every object like the cause, produces always some object like the effect. Beyond these three circumstances of contiguity, priority, and constant*

> *conjunction, I can discover nothing in the cause.* (David Hume, 1740, Para 9).

Thus, if an observational study can demonstrate that the cause always precedes the effect (*a* precedes *b* in time), that the effect is consistently close to the cause (*a* and *b* are spatiotemporally contiguous), and that the association is repeatedly and constantly observed (events like *a* are invariably followed by events like *b*), we can in fact still claim causation in a Humean sense (*a* causes *b*), but not counterfactually. Indeed, this is the position of GRADE[6] regarding the potential for "upgrading" observational studies to the status of RCTs (Guyatt et al., 2011). This regularity view of causation offers a better philosophical stance for supporting causal claims from observational studies, in the sense of capturing how evidential frameworks view causation. The regularities view is still embedded with a counterfactual make-up: trials simply compare the difference between two or more regularly occurring events. In this sense, the counterfactual condition can thus be seen as some sort of "add-on" to strengthen a belief in the observer's impression of the comparative rates of events. However, that the *truthmaker* to a causal claim is the counterfactual group is an unstable position to hold. It is clear that the *causes* we are interested in are actually in the factual group (the intervention group, say).

Let us consider a further dimension to understanding what causation might be here. This relates to EBM's de-emphasis of evidence from mechanistic science and mechanistic reasoning[7]. This, I say, further supports the notion of causation in EBM being considered

6 Grading of Recommendations, Assessment, Development and Evaluations Working Group

7 These terms express a concept that relates to the scientific activity *and associated inferences* of understanding and explaining how parts of a system relate to each other. The conflation of "scientific activity" and "associated inferences" is purposeful and facilitates clarity. In this case then, what I mean by *mechanisms* seems similar to what others mean by it, for example (Clarke et al., 2014).

as something Humean. Take *On the idea of a necessary connexion* from Hume's *Enquiry*:

> *The impulse of one billiard-ball is attended with motion in the second. This is the whole that appears to the outward senses. The mind feels no sentiment or inward impression from this succession of objects: consequently, there is not, in any single, particular instance of cause and effect,* ***any thing*** *[sic]* ***which can suggest the idea of power or necessary connexion*** *[emphasis added]* (Hume, 1748, Sect 7, Part 1, Para 50)

So, Humeans have a strict interpretation of this. Briefly, for the present purpose, we can use Lewis' *Neo-Humean Supervenience* thesis as a helpful example of such Humeanism:

> *[A]ll there is to the world is a vast mosaic of local matters of particular fact, just one little thing and then another. ... We have geometry: a system of external relations of spatiotemporal distances between points. Maybe points of space-time itself, maybe point-sized bits of matter or aether* (sic) *or fields, maybe both. And at those points we have local qualities: perfectly natural intrinsic properties that need nothing bigger than a point at which to be instantiated. For short: we have an arrangement of qualities.* ***And that is all. There is no difference without difference in the arrangement of qualities. All else supervenes on that*** *[emphasis added]* (Lewis, 1986, p. IX)

Thus, the world here is simply a vast collection of local matters of facts with all else supervening on the mosaic of facts. The facts themselves, however, are "devoid of any intrinsic nomic, causal, or modal character" (Jacobs, 2011, p. 81). In these terms then, EBM seems to have no concern with understanding causes as anything more than regularities of facts. That is, there is nothing internal to the causal process that relates to a cause producing an effect, for example, some sort of real force or compulsion. Causation is just

one thing followed regularly by another. EBM seems not to claim that causation is itself observable in either RCTs or observational studies. It is only the regularitiy of one event being followed by another that is observable.

Although Humeans (and EBM) might be happy with this stance as a complete account of causation, health science and healthcare practice might not be. Health science in one sense seems rich with a history of informative science ranging from laboratory studies through to large scale clinical trials. Further, healthcare itself is constructed of clinical experiences, patient values and social contexts. Indeed all of these elements are explicitly embraced and showcased in EBM's manifesto. If a Humean stance is to be taken, then what is there to be said of the *other* discounted knowledge, experiences, patient input, and contexts? The EBM framework determines that clinical decisions – entailing causal intentions – should integrate as much of this knowledge as possible. A Humean commitment seems not to allow such background conditions a role in the understanding of a precise nature of causation in health care, thus an ontological tension seems to exist. This tension exists in at least two places: First, tension within the research methods themselves. Although broadly Humean, there are some difficulties in understanding how comparative trials (counterfactual dependency) relate to a *difference making* theory of causation, whilst observational studies relate more closely to a pure *regularities* view of causation. Even within a Humean world, it is therefore difficult to understand what causation is. If we do not understand what causation is, then how do we know we have found it? This makes it troublesome to understand how observed facts can remain stable through their transition to spatiotemporally removed environments. What is holding the facts together in order for them to remain stable during their transference and operationalisation in the complex and context-sensitive situations of clinical decision-making for individuals and populations?

Summary of the problems with Hume

Hume offered an account of causation by which the research activity with EBM can be understood. The way causal claims are generated within the EBM framework is, in no uncertain terms, Humean. This leaves us asking whether or not this account is relevant for the central claim of EBM, which is concerned with translating such claims from their source of origin (research) to place of intended use (clinical practice). Is the sort of causation established by EBM's research methods the sort of causation we desire in either policy or individual clinical decision-making?

To sum up the Humean position, what we can say is that causation is in fact not causation at all, but something that is analysed away into a collection of discrete statistical facts. Hume and EBM explicitly demonstrate that they have no concern with the matter of causation itself, only in what can be known by the structured and systematic observation of constantly conjoined events. Taking these statistical facts to a spatiotemporally removed environment relies on assumptions and premises that must be defined by what those facts represent. Below, we can see that this is problematic if health care is to be thought of as something which embraces the complexity of human nature and the socio-cultural environment within which humans exist. Indeed, this is something which is most likely not even up for debate. To portray this, we can consider a model of person-centred health care, and try to understand how a Humean idea of causation resonates with the characteristics of a holistic health care model. Previous authors have defined what might be meant by a humanistic, holistic health care model:

> *Person centered medicine an affordable biomedical and technological advance to be delivered to patients within a humanistic framework of care that recognises the importance of applying science in a manner that respects the patients as a whole person and takes full account of*

> *his* (sic) *values, preferences, aspirations, stories, cultural context, fears, worries and hopes and thus that recognises and responds to his emotional, social and spiritual necessities in addition to his physical needs* (Miles & Mezzich, 2012, p. 219).

This model builds on earlier "landmark" casuistic framing of how EBM should be considered (Tonelli, 2006). Further, it references a historical background of care related to the evolution of a biopsychosocial framework (Engel, 1977) – namely Francis Peabody's *The Care of The Patient*, and Paul Tournier's *Medicine de la Personne*.[8] Referring to Engel's rejection of a monistic or reductionist approach to clinical practice, the sentiments of person-centred health care are clear:

> *In doing so, he [Engel] directly laid the foundations of the thinking that now recognises the importance of complexity theory in medical practice and that illustrates that clinical phenomena are generally far too complex to be understood solely through the use of linear cause-effect models* (Miles & Mezzich, 2012, p. 210).

The emergence of person-centred health care can be sketched out from a developing EBM movement and a general idea of historical "good care" referred to as patient-centred care. The difference between the *patient* and the *person* is emphasised in sympathy to a "deep respect…as unique living beings" (Epstein & Street, 2011, p. 100). Person-centred health care gives a substantial and focused account of a "crisis" of knowledge, care, compassion and costs in modern medicine (Miles & Mezzich, 2012). Its claim is that an over-emphasis of scientific medicine has resulted in the depersonalisation of care. In response, proponents of person-centred

8 See: Peabody, F. W. (1927). The Care of the Patient. Journal of the American Medical Association 88, p. 877 – 882.; Tournier P. (1940) Médicine de la Personne, Neuchatel, Switzerland: Delachaux et Niestlé.

care propose an emergent humanistic model of clinical practice grounded in holism and complexity. There is an easy and intuitive response to this, that EBM does not in fact "depersonalise" care at all. EBM proponents have indeed suggested how EBM has led to the sophisticated articulation of the proper role that patients' values and circumstances play in clinical decision-making (for example, Montori and Guyatt, 2008 and Montori et al., 2013). Furthermore, it may be that the person-centred argument sees EBM as a rigid strategy for the practicalities of clinical decision-making (always do what is supported by the best evidence). This might not be a fair characterisation of EBM because of the tension it artificially creates between facts and individual values. However, despite these intuitive responses, it is still unclear to see how the relationship between facts (data, say) and values (clinical context, patient values), might actually develop whilst maintaining the grounded principles of holistic person-centred care. This is especially the case given the Humean characterisation of causation.

We are now left with a paradox whereby the very methods prioritised by EBM to inform its own practice reveal an account of causation in which the translation of causal claims remains unfounded. So the question remains: Can there be an alternative way of accounting for causation in EBM that relates much more convincingly to person-centred, holistic health care? The final section of this chapter argues that there is, and as such sketches out, a possible re-conceptualisation of the causal account for EBM.

Causal dispositionalism as a way forward for evidence-based health care

I have drawn out an account for causation within health care by considering the way by which it structures its evidential sources. This is an unusual way to explore an ontology, but EBM gave few other

options. EBM seems not to be concerned with what causation is, only the observation of discrete, constantly conjoined events. As it stands, EBM is perhaps best considered as deontological. Although this might satisfy some (for example, Howick, 2011), it seems substantially to curtail further enquiry and progress into the philosophy and practice of health care. I suggest a position where a causal ontology is developed in light of the core values and purposes of health care, say, as per person-centred care, and that its epistemological account follows. Therefore, what we read from the outputs of the methods, far better relate to the type of causal activity desired in making clinical decisions intended to impact on the health status of individual people or inform health policy. Here, I suggest that a theory of causal dispositionalism can respond to many of the problems exposed by the Humean account. The theory in mind is one based on the work of Mumford and Anjum (Mumford & Anjum, 2011), and is one which takes causes as tendencies towards an effect, and as things which manifest in single instances. The theory considers the notion of causation as complex, and whether or not causes result in some effect is a highly context-sensitive issue. Furthermore, and of utmost importance in responding to Hume, the theory takes causes as real and primitive – that is, causation cannot be analysed away to something else, as is the case with the Humean account. Causes are the very things that make up what the person is, and the person is the most obvious source of observation to understand what causation is.

It is beyond the parameters of this chapter to lay out a fully detailed account of the theory of dispositionalism, and for this the reader is directed to supporting resources (Kerry et al., 2012; Eriksen et al., 2013; Mumford & Anjum, 2011; Mumford & Anjum, 2012). For the purpose of this chapter, causal dispositionalism will be introduced within a focussed framework of key areas relevant to the central claims of EBM. The key areas which emerge from the existing problematisation of causation and which also relate to

EBMs central claim are i) how can a theory provide detail of causal content from its methods, ii) how can it motivate a viable epistemology, iii) how can it account for individual decision-making, and iv) how can it help understand the assumptions needed to bridge the inferential gap between population level evidence and clinical decisions. These four key areas are now attended to in turn.

Causal content

A traditional Humean account of causation offers some explanation as to how causal claims are developed from research methods. The account is able to discuss such claims in terms of either frequencies of occurrence of events, the degree of differences between two frequencies, or both. Proponents of the Humean account are satisfied that this sufficiently explains the causal role of research content, specifically highlighting that this avoids unnecessary matters of ontology. The dispositionalist response is straightforward: the content that is being referred to here is not of causation, but of something else. The essence of causation has not been reached, and as such, any explanation related to causal content cannot be given. The "truthmaker" of causation within traditional accounts is removed from where causation itself is most likely to be found. What dispositionalism offers is a view that sees causation within the core of the content itself. Changes are seen within groups, and these changes occur because of multiple events tending towards and away from effects. Whereas Humeans consider single and necessary causes by proxy of frequently occurring observed events, dispositionalists see various causal factors that may or may not manifest in an effect. The causal role of these events for dispositionalism is the notion of how they manifest and how they may tend towards and away from anticipated thresholds. Dispositionalists are unsatisfied with causal explanations that relate to frequentist interpretations of probability,

as probability should be thought of in relation to the propensities held by causal factors.

Epistemology

Proponents of a Humean account of causation in health care have taken it as problematic that dispositionalism sees causes as real, for example Williamson (2006). By committing to the reality of causes, the demarcation between those matters that are problematic and those that are not is compromised. As such, accepting that causes themselves may not be a real feature of the world, permits priority of epistemological enquiry – that is, we do not need a "thick" theory of causation in order to understand the world. As such, Williamson and others prefer a theory of *epistemic causality* (Russo & Williamson, 2011). In order for such a theory to advance understanding of the world, it must accept that multiple methods are used as evidential sources. However, because there is no commitment to what causation is, all that can be said is that each method must relate to its own interpretation of causation, for example RCTs relate to causes as making a difference; observational studies relate to causes as regularly occurring events; etc. Although this *causal pluralism* seems to advance from an idea on *causal monism* (that there is only one way to find one idea of causation), its inability to commit to a single idea of what causation is leads again to a Humean stalemate.

Dispositionalism offers a commitment to monism, but an *ontological* monism. That is to say, there is a single idea of what a cause is. It can align this with a notion of pluralism, but a *methodological* pluralism. This means that because causes are complex and context-sensitive, they will display many symptoms. Accepting that the outcomes of scientific methods are *symptomatic* rather than *constitutive* of causation facilitates a methodological pluralist stance, whereby information from

multiple methods and sources may reveal parts of the causal process. These sources can include indicators of causation such as mechanistic science and patient narratives. Dispositionalists do not need to worry about the cumbersome and ultimately unsuccessful reconciliation of multiple theories of causation, because causes are only one thing.

Individual decision-making

Proponents of Hume say their causal account explains inference from research to the clinic. However, the assumptions for this are unfounded. They argue for this conclusion with limited consideration of the ontological issues at stake. Indeed, their stance suggests those considerations are not even relevant. An ontological inquiry however is beneficial in unveiling some of the shortcomings of current causal models for the complexities of medical phenomena. The essence of this chapter centres on the relationship between general and particular instances of causes. A causal theory should account for causal processes in individual-level clinical decision-making. The traditional stance had limited options and utility concerning this, appealing to either a rationalised faith in probabilistic inference, or some claim to universal laws. Dispositionalism can respond with ease to this desideratum. Although it has no deep ontological commitment to the priority of either general or particular instances, dispositionalism does take the single instance as to where causes are. From this, the theory can account for general causes as being *signals* to where causation might lie. There is no commitment needed to universal laws, and the account avoids problems associated with probability and induction, as below.

The inferential gap

The Humean position is simply to assert that if prioritised methods are conducted correctly – without experimental error – then

predictions should be forthcoming that are simple, exact and unfailing. Good examples of this characterisation of EBM are found in critical responses to a dispositions account by, for example, Strand and Parkkinen (Strand & Parkkinen, 2014; 2015). However, we know this to be false; otherwise the problem of induction would not be centuries old. The gap between science and application will never be unfailing, and inferences can always be wrong. Any account of causal inferences has, therefore, to respect the obvious datum that predictions are fallible and defeasible. A Humean account is grounded in deductivism and therefore necessity. What this means is that the logical form of the traditional account dictates that all claims will be necessarily so, to a probabilistic level (note that framing necessity in terms of probability does not solve this issue). So how can cases of failed causation be accounted for? Dispositionalism avoids commitment to ever necessitating an effect, and rather offers an explanation of prediction and inference within a fallibilist's framework in which dispositions tend to produce their effects but might not always do so. With dispositionalism, the problem of induction becomes redundant.

Conclusion

This chapter has proposed that causation is an idea that is central to the essence of evidence-based physiotherapy. By understanding the structure of prioritised evidential sources within the EBM framework, a causal account related to a Humean theory of causation can be developed. This is problematic to the central claims of EBM because a world of constantly conjoined discrete events does not seem prepared to relate to a person-centred model of health. Thus, any grounds on which to bridge the space between research outcomes and clinical decision-making remain unfounded. What would better support and progress the purpose of evidence-based practice is a theory of

causation that better understands causes to be real, singular, complex, and context-sensitive. If this were the case, multiple research methods would be able to work in establishing where causes lie. However, population level research outcomes would not be constitutive of causation, merely symptomatic. The real causal matter would be found in the single, individual case.

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