

Norwegian University of Life Sciences  
School of Economics and Business



# Evidence Based or Person Centered? An Ontological Debate

Rani Lill Anjum

Working Papers No. 7 / 2016

ISSN: 2464-1561

# Evidence Based or Person Centered? An Ontological Debate

Rani Lill Anjum

## Abstract

Evidence based medicine (EBM) is under critical debate, and person centered healthcare (PCH) has been proposed as an improvement. But is PCH offered as a supplement or as a replacement of EBM? *Prima facie* PCH only concerns the practice of medicine, while the contended features of EBM also include methods and medical model. I here argue that there are good philosophical reasons to see PCH as a radical alternative to the existing medical paradigm of EBM, since the two seem committed to conflicting ontologies. This paper aims to make explicit some of the most fundamental assumptions that motivate EBM and PCH, respectively, in order to show that the choice between them ultimately comes down to ontological preference. While EBM has a solid foundation in positivism, or what I here call Humeanism, PCH is more consistent with causal dispositionalism. I conclude that if there is a paradigmatic revolution on the way in medicine, it is first of all one of ontology.

## Introduction

There is a move within the medical paradigm: from evidence based medicine (EBM) and practice towards a more person centered healthcare (PCH). The debate concerns a number of issues related to the scientific foundation of medicine, including methods, models, concepts and practice. According to Kuhn (1962: 88)<sup>1</sup>, it is a sign of a paradigm in crisis when its members start participating in philosophical discussions, which many of these debates seem to involve. But we should not conclude from this that what we are witnessing here is primarily a medical revolution.

If we consider these discussions in unison, we can detect a fundamental ontological disagreement between the two approaches to healthcare. While EBM seems to have a solid positivist or Humean basis, PCH emphasises features and values more consistent with the ontology of causal dispositionalism. I will show here that it matters which of these ontological frameworks we choose, and that this is what makes us favour one approach to healthcare over the other.

---

<sup>1</sup> "It is, I think, particularly in periods of acknowledged crisis that scientists have turned to philosophical analysis as a device for unlocking the riddles of their field. Scientists have not generally needed or wanted to be philosophers. Indeed, normal science usually holds creative philosophy at arm's length, and probably for good reasons. To the extent that normal research work can be conducted by using the paradigm as a model, rules and assumptions need not be made explicit." Kuhn 1962: VIII, p. 88.

### **Person centered healthcare: reform or revolution?**

How revolutionary is the shift to PCH? Is it primarily a matter of practice: of how to meet and treat the patient once they enter the health system? If so, PCH seems detached from the scientific context of medicine and constrained to the realm of ethics. While the scientific concerns are focused on theory, methodology and evidence, PCH introduces more social and moral language, using terms such as value, empowerment, participation, respect, collaboration, community and autonomy (WHO 2007). Perhaps this is the most important contribution of PCH to medicine.

A question still remains whether PCH should be understood as an alternative or supplement to EBM. Could we simply add PCH as an extra ethical dimension on top of what we are already doing; or do we need a more radical change in medical thinking? Advocates of PCH disagree over this matter. Philosophically speaking, however, there are good reasons to see PCH as a more radical move away from the evidence-based framework. But this is not a typical Kuhnian scientific revolution, primarily concerned with replacing medical theory. Rather, the growing interest in the person centered approach should be seen as a symptom of a more fundamental change; a change in philosophical perspective, methodological priority and conceptual interpretation.

I here argue that if there is a paradigmatic change within medicine, then this is an ontological – not a scientific – revolution. To demonstrate this, I briefly mention some of the concerns that have been raised against various aspects of EBM, before I move on to show how EBM and PCH are committed to a number of philosophical assumptions, most of which are never explicitly discussed. I conclude that the real debate is between two conflicting ontologies, placing PCH and EBM within opposing camps.

### **Debating EBM**

While the evidence-based approach is gaining popularity within other disciplines (e.g. education, management, criminology, politics), a number of concerns are expressed by philosophers, practitioners and medical researchers contesting some of the features of EBM. These debates are often disconnected and constrained within disciplinary boundaries, but for our purpose here, it is useful to arrange them into broad categories. Hickey et al. (2012: 69) divide the debate into *statistics*, *philosophy* and *practice*, but since I take all of these debates to have a philosophical source, I will instead use the categories *methods*, *models* and *practice*. Under each, I will list and explain the points of contention.

#### *Methods*

One type of debate addresses problems with the use of randomised controlled trials (RCTs) and other statistical methods in medicine and healthcare. The following methodological approaches of EBM are all contested.

- The quantitative approach: The highest-ranking methodologies of EBM are quantitative, using statistical tools. Evidence from qualitative studies are disregarded in meta-analyses and even excluded from the evidence hierarchy. So while few disagree that medicine should be based on evidence, this is restricted in EBM to evidence of a certain type.

- Use of statistical averages: In clinical decisions, patients are consistently treated as statistical averages of the relevant patient group(s), usually defined by basic demographic data. But different patient groups have different averages and each patient necessarily belongs to more than one group: woman, 40+, high education, vegetarian, heart condition in the family, low blood pressure, and so on. It seems unlikely that a patient can reasonably be treated as the sum of all these averages.
- Ecological fallacy: In calculating prevalence and risks of outcomes, individual probabilities of a patient are directly derived from statistical probabilities. Such inferences from frequencies to propensities is known as the ecological fallacy, which is a logically invalid inference from group average to the individual. If half of all smokers die from it, it does not automatically follow that the probability of this outcome is exactly 0.5 for each individual smoker.
- Testing single factors: Many illnesses are recognised as complex and multifactorial, but RCTs and other comparative studies are best suited for testing single factors and interventions. This is to avoid the problem of overdetermination: a causal situation where more than one present factor is sufficient to produce the effect.
- External validity: Many medical interventions are tested on relatively young, healthy and homogeneous groups of patients, but the results are applied universally. This is a problem when caring for patients with multi-morbidity and individual variations, which is often the case with chronic and older patients (Wyller 2011).
- Limited application: RCTs are seen as the gold standard but because of rigid study design and strict exclusion and inclusion criteria (Rothwell 2005, Hjelmæsæth 2014), they are unsuitable to test many types of intervention, such as health effects of pollution, physical abuse or mental wellbeing. A criticism of RCTs and their privileged status within medicine is that it gives a methodological advantage to pharmacological interventions, since these are easily tested against a placebo.

From these debates, a new trend is emerging in medicine and healthcare that emphasises the importance of a wider notion of scientific evidence. Methodologies should also accommodate evidence from qualitative methods (Berkwits and Aronowitz 1995), medical and health humanities (Greaves and Evans 2000, Macnaughton 2011), patient stories (Broom 2000, Getz et al. 2011, Greenhalgh and Hurwitz 1998, Kirkengen 2001) and mechanistic (theoretical) knowledge (Glennan 1997, Russo and Williamson 2007, Clarke et al. 2013 and 2014). So instead of the strict evidence hierarchy, it should be acknowledged that relevant evidence is complex, comes in various forms and from different types of sources.

### *Models*

Many debates within healthcare and philosophy of science are related to the limitations of the bio-medical model. This model is not a part of EBM as such, but it is nevertheless seen as paradigmatic to current medical thinking and practice. Nevertheless, all the following features can be contested.

- Reductionism and dualism: While most illnesses are complex, including biological, psychological, historical and social elements, the bio-medical model is reductionist or at best dualist. Health

complaints that cannot be linked to a physiological abnormality remain medically unexplained. Mental and psycho-somatic illnesses are excluded from medicine and treated separately, unless a clear pharmacological treatment can be found.

- **Medicalisation:** Complex psycho-social phenomena are simplified in the pursuit of biomedical causes. On one hand, this involves narrowing the scope of human wellbeing and suffering, for instance by offering clinical diagnoses of what might be natural responses to an unbearable life situation. Grief can for instance be treated as depression, ultimately interpreting a social and interpersonal circumstance as a psychological problem of the individual that may or may not be improved by a chemical intervention. On the other hand, medicalisation involves an expansion of the medical domain and mandate into most aspects of life, including sexuality, parenthood, ageing and dying.
- **Fragmentation:** When focusing on biomedical explanations, illness is typically treated as belonging to a certain physical part of the patient, such as the back, the liver or the heart. Each body-part belongs to different medical specialisms. Many practitioners have noted, however, that a number of physical health complaints must be seen in a wider context of the patient's personal and relational situation (see for instance Thornquist and Kirkengen 2015).
- **Single disease approach:** While co- and multi-morbidity is the norm in medicine, clinical guidelines are for individual illnesses. We then get a situation that has been referred to as "silo medicine" (Getz 2006, Parekh and Barton 2010, Vogt et al. 2014), where each diagnosis has its own expert groups, patient organisations, industry sponsors and clinical guidelines. Diseases are then treated as wholes ("disease holism"), while patients are treated as composed of parts ("patient compositionality").

The bio-psychosocial model, developed by Engel (1977), was introduced as a better alternative to the biomedical model. This model is anti-reductionist and allows all aspects to be taken into account. In addition, the need for multifactorial causation seems acknowledged. Still, holism remains a problem, since genuine complexity cannot be inferred from a number of separate studies testing biological, psychological and social factors (Butler et al. 2004, Eriksen et al. 2013).

### *Practice*

A further discussion in medicine, to which PCH is an important contribution, is about clinical practice and moral values. Practitioners and therapists have raised concerns about the effects that evidence based policies have on the way in which healthcare is provided in clinical settings. The following are the contentious features of EBM that concern medical practice.

- **Lack of autonomy:** Evidence-based policies tend to downgrade clinical expertise and phronesis (practical judgement), leaving little room for practitioners to judge which treatment is best for the patient. Practitioners are encouraged to use computational tools to diagnose and treat the patients and new technologies are developed: from simple smartphone apps that can measure blood pressure to highly advanced virtual doctors, such as *Ellie*, a virtual interviewer designed to detect depression and post-traumatic stress disorder (DeVault et al. 2014).

- Universal treatment: It is generally agreed that each patient is different and with different needs, but EBM is motivated by the idea that the same treatment should be given to all. Still, there is no intervention that benefits everyone, meaning that at least some patients will have no effect, suffer side effects or get worse from the intervention that benefits most.
- New Public Management: It is required that health service delivery is time- and cost-efficient, specifying for instance how much time should be spent on different types of jobs (food, medication, washing). Resources are allocated from generic standards, e.g. type of diagnosis, instead of assessing each individual patient's needs. Behind this practice is the idea that one size fits all, where treatment is given as standard packages (Vogt and Pahle 2015).
- Cure versus care: Medicine has become increasingly scientific and standardised, but at the same time more de-personalised. Science and humanism are treated as separate domains (Miles 2009: 943, Miles and Asbridge 2013: 4).

There is a trend towards a more personalised, holistic and biographically informed healthcare, with focus on patient values and preferences. A number of old dichotomies are effectively challenged, including the distinction between facts and values, parts and wholes, psyche and soma and between subjectivity and objectivity (Laughlin et al. 2013).

#### *PCH as an improved paradigm*

Instead of seeing person centred healthcare as a contribution only to a debate on practice and values, it actually challenges some of the most basic assumptions within current medical thinking. To show how PCH represents such a major paradigmatic change in medicine, I will now try to make explicit some of the implicit ontological commitments that seem to lie behind and motivate EBM and PCH, respectively. This suggests that the ultimate choice to be made is between competing ontologies.

#### **Ontological commitments of EBM**

Science and philosophy are often treated as contrasting activities. Where science is a largely empirical enterprise, philosophy is abstract and conceptual. Still, many of our most fundamental assumptions, including those upon which science is based, are not empirically supported. In this section, I present some philosophical commitments of the evidence-based framework and show how most of these can be traced back to David Hume (1739, Book I). The aim is not to give an accurate representation of Hume's philosophy, but rather to establish that the scientific norms of EBM are not ontologically neutral but actually carry with them a number of philosophical assumptions. An ontology is a commitment, or list of commitments, to the reality of certain fundamental types of entity or entities. For instance, one might think that the world contains particular things, physical objects, properties, causes, laws of nature, substances, and so on. A further ontological matter is the nature of these things. One might disagree with respect to what causation is, for instance, or what it means that reality is complex. The following tacit ontological commitments can be detected within EBM.

*Empirical evidence trumps theory*

In the hierarchy of evidence, EBM favours empirical evidence over theoretical knowledge and observation data over mechanisms (Howick 2011: 4). The motivation for this priority is that if our scientific claims cannot be backed up by data, we have little reason to trust them (Howick et al. 2009: 189, Howick 2011, ch. 10). Another motivation is that much of our medical insight comes from observation data and not from understanding the underlying causal mechanisms, as was the case with smoking being established as a cause of lung cancer (Doll and Hill 1954). Evidence is then not primarily a matter of finding out why or how an intervention works, but instead whether and how often it works.

A justification for choosing data over theory can be found in Hume's philosophy. Hume was a strict empiricist, meaning that he would only accept as genuine knowledge that which we could experience directly through our senses. Anything that does not satisfy the empiricist criterion belongs to what he called 'metaphysical speculation' and should not be trusted as genuine knowledge (Hume 1748: XII, iii, 165).<sup>2</sup> Hume was also critical of the epistemic status of scientific theories, because any universal truth is vulnerable to the problem of induction. It is an inference beyond what has been observed to the nature of unobserved events.

Given an empiricist approach to knowledge, EBM is perfectly justified in giving epistemic priority to empirical data over theories about causal mechanisms. Empiricist philosophy, such as Hume's, has a sparse ontology in which the world is constructed from observed events, with a strict prohibition against positing anything beyond that as real. To be legitimate, any category, such as cause, has to have some demonstrable origin in an original experience or 'impression'.

*Correlations first, then possibly causation*

In epidemiological research, there is a reluctance to make causal claims on the basis of data. Instead, one might report a raised incidence of an outcome in the treatment group over the placebo group, or increased relative risk (Broadbent 2013: ch. 3). Such scepticism towards causal claims is compatible with Hume's analysis of causation. Before Hume, and also by his subsequent opponents, it was assumed that causation provides necessary connections to the world (Aristotle *Metaphysics*  $\theta$  5: 264, Spinoza 1677: I, axiom III: 46, Kant 1781: II.ii.3 second analogy, Mill 1843: III.v.6, Mackie 1980:62). Hume, however, denies this on strict empirical grounds. No necessary connection between the cause and effect is observable. Rather, his famous analysis of causation (Hume 1739: I, iii, 14, p. 170) restricts it to a threefold observable relation.

To Hume, causation comes down to habit of expectations, based on prior observation (ibid.). When we see two types of events where one follows the other (a relation of temporal asymmetry), repeatedly (there is a relation of regularity or 'constant conjunction') and together in time and space (relation of contiguity), we have a natural expectation that the next time we see the first event, it will be followed

---

<sup>2</sup> "When we run over libraries, persuaded of these principles, what havoc must we make? If we take in our hand any volume; of divinity or school metaphysics, for instance; let us ask, *Does it contain any abstract reasoning concerning quantity or number?* No. *Does it contain any experimental reasoning concerning matter of fact and existence?* No. Commit it then to the flames; for it can contain nothing but sophistry and illusion." (Hume 1748: XII, iii, 165)

by the second. But for this expectation we have no empirical basis, Hume argues, even for what we call laws of nature. Hume urges that we think of causation as nothing over and beyond two types of contiguous events regularly following each other. So while many think of causation as correlation plus something more (e.g. necessity), Hume thought of causation instead as strictly nothing more than a special type of correlation.

Humeans understand the world as a patchwork of unconnected events (Lewis 1986a: ix). Some events come together with, or are accompanied by, others. But there is no real production of an effect by its cause. On this view, it is perfectly rational to search for causation via correlation data. The more robust the correlation, the stronger the evidence of causation. For Humeans, there is nothing more to be found of causation than this.

#### *Homogeneity as an ideal starting point*

A central feature of Hume's regularity view, is that same cause gives same effect. This assumption can also be seen in EBM, where we often start from the perspective of homogeneity (Eriksen et al. 2013). For instance, we could imagine a pair of identical twins who lived exactly the same life, so that all the biological, social and psychological factors would be the same for both. Ideally, then, one could infer from what happens to one of them what would also happen to the other.

This is what we do when we try to find the relevant statistical data for our patients: we try to find their most relevant sub-group, which ideally would consist of the patient's "identical twins". The information about the patient group can then be used to inform predictions and clinical decisions about individual patients. It follows from this assumption that if there is a difference in outcome between the patient and the patient group, then this must be because there is a causally relevant difference between the patient and the group. Any prediction of effect is therefore made with a *ceteris paribus* clause: all else being equal.

We find the same background assumption in another aspect of EBM too. While individual variation is acknowledged, through randomisation over a large enough population, the aim is to produce two test groups that are effectively "identical twin" populations. So if a trial intervention then works on the treatment group, the assumption is that it would have worked equally well, and to the same degree, on the placebo group, had they received it. And if one further added an assumption that the treatment group was representative of the general population, then it ought to work on them too.

If same cause gives same effect, then starting from homogeneity when testing causation seems the most rational choice, even if heterogeneity between patients is acknowledged.

#### *More data gives better evidence*

We saw that EBM gives epistemic priority to large-scale correlation data and systematic reviews in providing the best proof of causation (Howick 2011: 4-5). The emphasis on quantitative methods and large amounts of data in EBM fits well with Hume's idea that causation is a law-like matter. He thought that causation can be known to happen in the particular instance only insofar as it occurs universally or at least generally. We can call this the covering law theory of causation.

The view that causation is inferred from the general to the particular, is also seen in EBM. If we want to know whether an intervention caused an outcome in a particular patient, we perform a study to see whether the same happens to others. Since Hume takes causation to be correlation or constant conjunction, the more data we gather, the closer we get to causal knowledge. A total data set would then give us complete knowledge, also of causal laws, since we avoid any problem of generalising beyond the sample.

If causal knowledge is derived from the general to the particular in this way, then EBM is justified in giving priority to population studies over qualitative studies and clinical experience.

### *Probability as statistical frequencies*

EBM is premised on the idea that what works on group level can and should inform clinical decisions about the individual patient (Hickey et al. 2012: 69-70). If a treatment cures 70 percent of a patient group, then one might say that a patient who gets this treatment has a 0.7 probability of being cured. This fits with a Humean and empiricist view on probability. The view of probability inspired by the empiricist view is frequentism (Venn 1866). This is the view that the probability of a certain outcome is given by the proportion of successful outcomes in a sequence of trials. The higher the number of trials, the more reliable the result.

As already mentioned, problems of extrapolation, external validity and the ecological fallacy have been raised (Broadbent 2013, ch. 7, Fuller and Flores 2015). But if we assume frequentism about probabilities, EBM cannot be criticised for applying statistical results directly to individual patients. As long as the results come from a sufficiently large sample and the relevant patient group, this is exactly how probabilities are determined, for empiricists.

### *Complexity and wholes are studied through their parts*

Since no quantitative or physiological study is tailored to one individual, a way to deal with heterogeneity and variation could be to look at results from different studies, testing different factors. This is one way to deal with multi-morbidity and causal complexity that could fit the bio-psychosocial model. We could then first test the effects of some biological factors, then of some psychological and social factors. When adding these results together, we might have a better idea of the complexity of the illness. Causally complex must then mean the same as multi-factorial. Otherwise, we should expect that the different factors interacted and affected each other, rather than just adding up in a simple linear fashion.

There is, however, a view in philosophy that wholes can be treated as sums of their parts, which don't themselves interact. This is called classical extensional mereology. From such a perspective, it seems less problematic to treat health and illness as belonging to various parts or aspects of the patient, rather than to a person as a whole. Given the unconnectedness of the elements in the Humean ontology, this mereological view of wholes is considered natural.

### *Summing up*

We see that at least some of the criticisms that have been posed against the evidence-based framework would instead be more appropriately targeted at the underlying ontology. If one thinks that the world is fundamentally this way, some of the methodological assumptions found within EBM follow naturally. The biomedical model is justified in the assumption of reductionism, for instance, and statistical methods are appropriate for generating individual probabilities if we assume frequentism. Mereological composition, against genuine holism, seems warranted and the regularity theory of causation supports universal treatment and finding causes through homogeneity.

Since someone could consistently commit to each of the ontological principles that we here refer to as Humean (e.g. Lewis 1986), there is nothing intrinsically wrong with the evidence-based methods or practice. From a Humean ontological perspective, much of the criticism against EBM is simply unwarranted.

Only from a very different ontological perspective, therefore, do we seem justified in our criticism: for instance, that EBM cannot accommodate holism, complexity, heterogeneity, individual propensities or causal mechanisms.

### **Causal dispositionalism and PCH**

I now move on to present an ontology that represents a radical departure from Humeanism, and which can accommodate exactly those features that EBM is said to ignore or devalue. This ontology is called causal dispositionalism and is developed in detail in Mumford and Anjum (2011), based on previous work by Harré and Madden (1975) and Molnar (2003). From this perspective, the scientific norms and methods of PCH seems a preferable alternative to EBM. The following features illustrate this.

### *Science deals with causation*

On causal dispositionalism, causation is one of the most basic features of reality. Among other things, this means that it cannot be reduced to the observable relations that Hume suggests; indeed, it cannot be reduced to anything at all. Causal production is fundamental, on this view, so it gives us an ontology in which particular causes and effects are genuinely connected rather than merely conjoined. Hume describes the elements in the world as 'loose and separate', but the causal dispositionalist instead allows that the features of particular situations have a natural tendency towards certain effects. They often succeed in producing those effects, though they needn't do so always. They will tend to do so. Regularity is not constitutive of causation, though it might be understood as a symptom of it. Approximate regularities are produced by the action of the natural tendencies of things.

In contrast, EBM is not primarily concerned with causal mechanisms, but simply with whether an intervention works. But can one really get by without real causation? It seems not. Even if one avoids explicit causal language, such as "cause", "prevent" or "produce", one cannot avoid invoking implicit causal notions (Broadbent 2013, ch. 3). When we say that an intervention "works", this might just mean that it increases the incidence, or reduces the risk of, the outcome. But the only coherent way it can do so, according to the causal dispositionalist, is by somehow affecting the outcome. "Work", "affect",

“increase”, “reduce” and “outcome” are all causal concepts, suggesting that the intervention has some real causal power to contribute to or counteract a certain effect.

From this perspective, science cannot be conducted with data alone, especially if we want to generate knowledge for explanation, understanding and prediction. If all we were interested in were the data themselves, we would have no reason to apply our results beyond the tested sample. Any attempt to make medical prognoses, policy recommendations or clinical decisions based on a medical study would then be meaningless, since nothing in the intervention is thought to do any actual causal work. But what then is the purpose of science? To generate the data is only part of science. If we aim to make a generalisation from the data sample to the general population, we need a causal hypothesis to even motivate it.

### *Establishing tendencies and causal powers*

A dispositionalist would not link causation to perfect regularities, but to tendencies. On this view, a cause is something that tends towards its effect with stronger or weaker intensity. Still, tendency does not here mean simply a statistical regularity, but one for which there is a real disposition or causal power. It is for instance a statistical fact that married men live longer than unmarried men. But a dispositionalist is primarily interested with what exactly it is that is doing the causal work. Is it the marital care, or the legal document, that has the power to prolong life in men? Or is the causal work instead done by something else related to marriage, such as a diet, social networks, companionship and emotional security? Is it simply that healthier men are more likely to marry?

Placing causal powers correctly is thus a major scientific task, and one that is taken seriously in medicine. Since the placebo effect is known to be strong, it is a moral matter as much as an epistemic one that the causal power to cure is in the drug and not only in the patient. From the dispositionalist perspective, the reason for performing RCTs is to establish that an intervention actually has the causal power to produce the anticipated outcome, and this is what it means to say that an intervention “works”.

The person centered approach seems *prima facie* interested in the questions of “why” and “how”, in its emphasis of understanding, meaning and sources. It is also acknowledged that there are a number of things that have causal power to affect our health, not all of which are biomedical. Causal relevance for health is attributed to a whole range of social, personal, spiritual, moral and existential parts of life. The insistence on replacing “patient” with “person” also fits the dispositionalist ontology. By using the term “patient” we metaphorically strip a person of their causal powers, leaving them causal patients rather than causal agents with respect to their own health: passive rather than active (Tyreman 2006).

### *Causation happens in the particular*

PCH gives epistemic priority to the individual person and their unique context. Placing the individual at the centre in this way is consistent with causal singularism, which is one feature of causal dispositionalism. What this means is that causation happens in the particular case, without an assumption of a corresponding causal law. Since each individual person represents a unique

combination of causal factors and dispositions, any generalisation from a population sample will necessarily involve an abstraction from individual variations, with the risk of missing something crucial.

In PCH many practitioners urge the importance of using “patient stories” for understanding the true sources of an illness, such as childhood trauma, sexual abuse or other traumatic experiences or loss of relations (Kirkengen 2010, Broom 2007). Failure to discover these, they argue, places the individual at risk of receiving inadequate healthcare and becoming chronic patients, focusing primarily on the immediate health complains. From a singularist perspective, it makes perfect sense for PCH to search for causes by looking into the background situation, uncovering as much as possible of the unique context that could be causally relevant for the illness and recovery.

#### *Complexity and multifactorial causation is the norm*

PCH acknowledges that the mono-causal model is limited for understanding health and illness. Instead, one must look to all aspects of life, including relational, environmental, cultural, moral and spiritual aspects. Complexity seems unavoidable in medicine, considering the large number of complex illnesses, co- and multi-morbidity and multi-factorial health complaints.

While multifactorial causation and complexity remains a challenge for medicine, a causal dispositionalist sees it as an essential feature of causation, where any outcome is produced by a number of causal factors in conjunction. Even for bacterial infections, the bacteria alone cannot cause the infection. They require the right “manifestation partner”; a body that can host it and a receptive environment. A body in stress might for instance have a weaker immune system, making it easier for the bacteria to spread. No ontological distinction is drawn, therefore, between cause, trigger, stimulus or background conditions within the dispositionalist understanding of causation. They are all causes that contribute to the outcome, and should therefore be taken into account.

#### *Individual variations should be expected*

Once we allow multifactorial causation and heterogeneity, some degree of medical uniqueness is to be expected. This fits well with dispositionalism, according to which causation is highly sensitive to contextual variations. Causal dispositionalism urges that any minor change in causal set-up might result in a vast effect in the outcome. This is because interaction of causal factors is often nonlinear. One example of nonlinear interaction of causes is the risk of lung cancer from the combination of smoking and asbestos exposure, which is 50 to 90 times higher than for smokers without such exposure.

Causal dispositionalism also explains why it is possible that different people respond differently to an intervention. On this ontology, one should not expect that there is a standard way to express a disease (Eriksen et al. 2013). Since no two patients will have exactly the same genetics, life-style, values, history, etc., any assumption of homogeneity is an abstraction from reality. Instead, one should expect that individual variations affect disease expression, prevalence and treatment response.

#### *A qualitative approach to causal evidence*

Causal dispositionalism reveals the importance of tailoring a treatment to the individual by looking at their total situation. Each person meets the treatment with a unique set of causal factors from their lifestyle, biology and medical history. The treatment that is best for most might still be harmful for some. The more we know about the individual, therefore, the better position we are in to understand their illness, its causes and to find the best treatment. But then we also need to understand causal powers, processes and mechanisms, and how an intervention affects the person.

To understand how – not only that – a causal factor is productive of an effect, gives evidential priority to individual propensities and mechanistic understanding over statistical frequencies and correlation data (Kerry et al. 2012). For this, qualitative, experimental or other interactive methods are more appropriate.

### *Summing up*

I have tried to show that the dispositional ontology is better suited for PCH. It provides the holism and complexity that is emphasised in the person centered approach. It also stresses context-sensitivity and causal singularism, thus motivating PCH's aim to accommodate heterogeneity and the unique needs of the individual. Methodologically, dispositionalism gives epistemic priority to qualitative studies, and to single propensities over statistical frequencies.

### **EBM or PCH? A choice of ontology**

I have now presented some features of two ontologies: Humeanism and causal dispositionalism. But how different are the two? Is it possible to accommodate both at the same time? Could one for instance change the practice and ontology while keeping the same methods? I would say no. Many of the criticisms directed at EBM reveals a dissatisfaction that goes beyond practice and methods and target our most fundamental ontological assumptions; of causation, probability and the very nature of knowledge.

While some argue that it is possible to make EBM more person-centred (cf. “the EBM Renaissance Movement”, see Greenhalgh et al. 2014), I would argue that PCH requires something that is quite dissimilar to EBM, not only an addition. A genuine consideration of an individual's health cannot easily be accommodated within a methodology that ultimately reduces uniqueness and complexity to the sum of various averages, or derives individual propensities from statistical frequencies. If one assumes instead that complexity is simply a compositional matter and that individual propensities are generated statistically, EBM is the way to go.

In contrast to this, causal dispositionalism gives us an ontological foundation for a more radical change within medical thinking. Figure 1 sums up the ontological commitments that justify a choice of PCH over EBM: holism over reductionism and dualism, complexity over mereological composition, context-sensitivity over regularity, causal singularism over universal laws and propensities over frequencies.

Empowering the individual and giving back professional autonomy to the practitioner to consider the total situation of the subject before deciding on a treatment, seems preferable from a dispositional point of view. On this singular ontology, universally applicable truths in medicine are a

misrepresentation of reality, which is multifactorial, heterogenic and highly contextual. To assume that the statistical generalisations carry more scientific force than the particular instances from which they are abstracted, would thus be a mistake. Causal singularism teaches us what PCH already knows: that each person is unique, and that one size does not fit all.

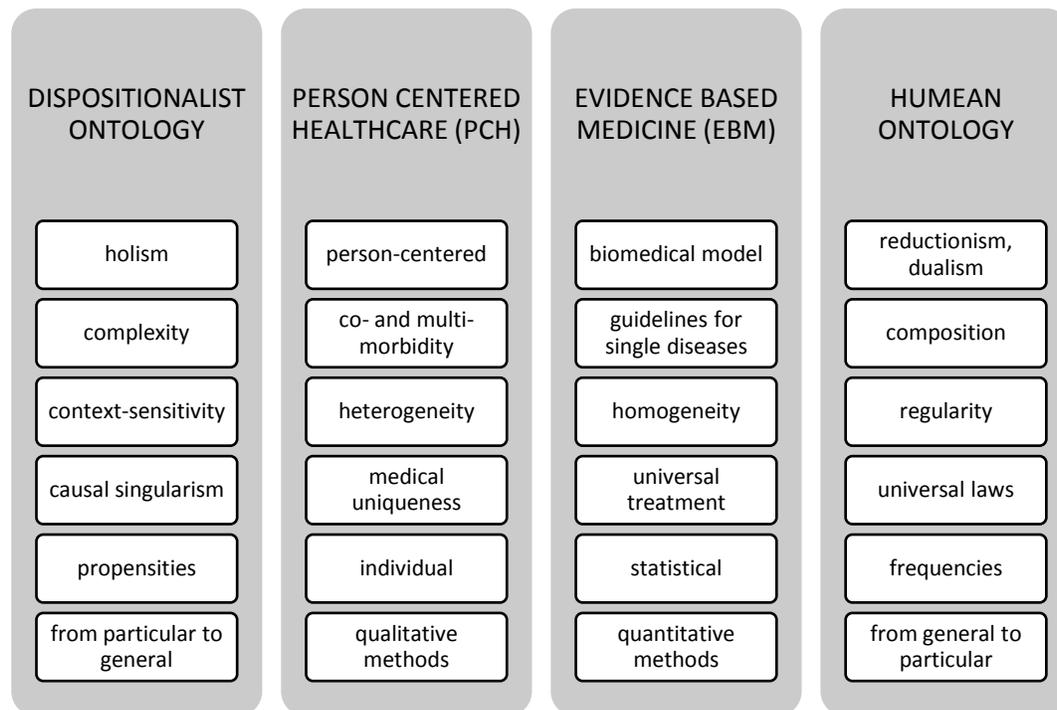


Figure 1: Ontological assumptions of PCH and EBM

## References

- Aristotle, *Metaphysics*, H. Lawson-Tancred (trans.), London: Penguin, 1998.
- Berkwits M., Aronowitz R. (1995) 'Different questions beg different methods', *Journal of General Internal Medicine*, 10 (7): 409-10.
- Broadbent, A. (2013) *Philosophy of Epidemiology*, New York: Palgrave Macmillan.
- Broom, B. C. (2000) *Somatic Illness and the Patient's Other Story*, London: Free Association Books.
- Broom, B. C. (2007) *Meaning-full Disease. How Personal Experience and Meanings Cause and Maintain Physical Illness*, London: Karnac.
- Butler, C. C., Evans, M., Greaves, D., & Simpson, S. (2004): 'Medically unexplained symptoms: the biopsychosocial model found wanting', *Journal of Royal Society of Medicine*, 97: 219-22.
- Clarke, B., Gillies, D., Illari, P., Russo, F., & Williamson, J. (2014) 'Mechanisms and the Evidence Hierarchy', *Topoi - special issue on 'Evidence and Causality in the Sciences*, 33(2): 339-360.

Clarke, B., Gillies, D., Illari, P., Russo, F., & Williamson, J. (2013) 'The evidence that evidence-based medicine omits', *Preventive Medicine*, 57 (6): 745-747.

DeVault, D., Artstein, R., Benn, G., Dey, T., Fast, E., Gainer, A., ... & Lucas, G. (2014, May) 'SimSensei Kiosk: A virtual human interviewer for healthcare decision support', In *Proceedings of the 2014 international conference on Autonomous agents and multi-agent systems*, International Foundation for Autonomous Agents and Multiagent Systems, pp. 1061-1068.

Doll, R. and Hill, A. B. (1954) 'The mortality of doctors in relation to their smoking habits; a preliminary report', *British Medical Journal*, 1: 1451-5.

Engel, G. L. (1977) 'The need for a new medical model: a challenge for biomedicine', *Science*, 196 (4286): 129-136.

Eriksen, T. E., Kerry, R., Mumford, S., Lie, S. A. N., & Anjum, R. L. (2013) 'At the borders of medical reasoning: aetiological and ontological challenges of medically unexplained symptoms', *Philosophy, Ethics, and Humanities in Medicine*, 8(1), 1-11.

Fuller, J. and Flores, L. J. (2015) 'The Risk GP Model: The standard model of prediction in medicine', *Studies in History and Philosophy of Biological and Biomedical Sciences*, 54: 49-61.

Getz, L. (2006) *Sustainable and Responsible Preventive Medicine*, Doctoral thesis, Trondheim: Norwegian University of Science and Technology, Faculty of Medicine, Department of Public Health and General Practice.

Getz, L., Kirkengen, A. L., & Ulvestad, E. (2011) 'The human biology – saturated with experience', *Tidsskrift for den Norske Lægeforening*, 131: 683-687.

Glennan, S. S. (1997) 'Probable causes and the distinction between subjective and objective chance', *Nôus*, 31: 496-519.

Greaves, D. and Evans, M. (2000) 'Medical humanities', *Medical Humanities*, 26: 1-2.

Greenhalgh, T. and Hurwitz, B. (eds.) (1998) *Narrative Based Medicine. Dialogue and Discourse in Clinical Practice*, London: BMJ Books.

Greenhalgh, T., Howick, J., Maskrey, N. (2014) 'Evidence based medicine: a movement in crisis?', *British Medical Journal*, 348: g3725.

Harré, R. and Madden, E. H. (1975) *Causal Powers: A Theory of Natural Necessity*, Oxford: Blackwell.

Hickey, D. S. (2012) 'The failure of evidence-based medicine?', *European Journal for Person Centered Healthcare*, 1 (1): 69-79.

Hjelmesæth, J. (2014) 'Randomised studies – useful for whom?', *Tidsskrift for den Norske Lægeforening*, 19: 134.

Howick, J. (2011) *The Philosophy of Evidence-Based Medicine*, Oxford: Wiley-Blackwell.

Howick, J., Glasziou, P., Aronson, J. K. (2009) 'The evolution of evidence hierarchies: what can Bradford Hill's 'guidelines for causation' contribute?', *Journal for the Royal Society of Medicine*, 102: 186-94.

- Hume, D. (1739) *A Treatise of Human Nature*, L. A. Selby-Bigge (ed.), Oxford: Clarendon Press, 1888.
- Hume, D. (1748) *An Enquiry Concerning Human Understanding*, P. Millican (ed.), Oxford: Oxford University Press, 2007.
- Howick, J. (2011) *The Philosophy of Evidence-Based Medicine*, Chichester: Wiley-Blackwell.
- Kant, I. (1781) *Critique of Pure Reason*, N. Kemp Smith (trans.), London: MacMillan, 1929.
- Kerry, R., Eriksen, T. E., Lie, S. A. N., Mumford, S. D., & Anjum, R. L. (2012) 'Causation and evidence-based practice: an ontological review', *Journal of Evaluation in Clinical Practice*, 18 (5), 1006-12.
- Kirkengen, A. L. (2001) *Inscribed Bodies*, Dordrecht: Kluwer Academic Publishers.
- Kirkengen, A. L. (2010) *The Lived Experience of Violation. How Abused Children Become Unhealthy Adults*, Bucharest: Zeta Books.
- Kuhn, T. (1962) *The Structure of Scientific Revolutions*, Chicago: University of Chicago Press.
- Lewis, D. (1986) *On The Plurality of Worlds*, Oxford: Blackwell.
- Lewis, D. (1986a) *Philosophical Papers II*, Oxford: Oxford University Press.
- Loughlin, M., Bluhm, R., Stoyanov, D. S., Buetow, S., Upshur, R. E., Borgerson, K., Goldenberg, M. J. & Kingma, E. (2013) 'Explanation, understanding, objectivity and experience', *Journal of Evaluation in Clinical Practice*, 19 (3), 415-421.
- Mackie, J. L. (1980) *The Cement of the Universe*, Oxford: Oxford University Press.
- Macnaughton, J. (2011) 'Medical humanities' challenge to medicine', *Journal of Evaluation in Clinical Practice*, 17 (5): 927-32.
- Miles, A. (2009) 'On a medicine of the whole person: away from scientific reductionism and towards the embrace of the complex in clinical practice', *Journal of Evaluation in Clinical Practice*, 15: 941-9.
- Miles, A. and Asbridge, J. (2013) 'The European Society for Person Centered Healthcare', *European Journal for Person Centered Healthcare*, 1 (1): 4-40.
- Mill, J. S. (1843) *A System of Logic*, London: Parker.
- Molnar, G. (2003) *Powers: A Study in Metaphysics*, S. Mumford (ed.), Oxford: Oxford University Press.
- Rothwell, P. M. (2005) 'External validity of randomised controlled trials: "to whom do the results of this trial apply?"', *The Lancet*, 365 (9453): 82-93.
- Russo, F. and Williamson, J. (2007) 'Interpreting causality in the health sciences', *International Studies in the Philosophy of Science*, 21 (2): 1157-70.
- Spinoza, B. (1677) *The Ethics*, R. H. M. Elwes (ed.), New York: Dover, 1955.
- Thornquist, E. and Kirkengen, A. L. (2015) 'The quantified self: closing the gap between general knowledge and particular case?', *Journal of Clinical Evaluation in Practice*, 21: 398-403.

Tyreman, Stephen (2006) 'Causes of illness in clinical practice: A conceptual exploration', *Medicine, Health Care and Philosophy*, 9 (3): 285-91.

Venn, J. (1866) *The Logic of Chance*, 4<sup>th</sup> edn, New York: Chelsea, 1962.

Vogt, H. and Pahle, A. S. (2015) 'Likeverd på samleband', *Morgenbladet* 11 September 2015, <http://morgenbladet.no/ideer/2015/09/likeverd-pa-samleband>.

Vogt, H. et al. (2014) 'Getting personal: can systems medicine integrate scientific and humanistic conceptions of the patient?', *Journal of Evaluation in Clinical Practice*, 20 (6): 942-952.

WHO - World Health Organization (2007) *People-Centred Health Care: A policy framework*, Manila: WHO Regional Office for the Western Pacific, <http://iris.wpro.who.int/handle/10665.1/5420>.

Wyller, T. B. (2011) 'Evidensbasert medisin eller vulgærcochranisme?', *Tidsskrift for den Norske Lægeforening*, 131: 1181-2.