GUEST EDITORIAL

CauseHealth: integrating philosophical perspectives into personcentered healthcare

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Introduction

Person-centered healthcare (PCH) represents a shift in medicine that goes far beyond practice and values. Arguably, a genuine shift away from the biomedical model and its reductionist, mechanistic, mono-causal approach, will also involve a change in the ontological framework. By urging a healthcare that accommodates individual variations and propensities, causal complexity, psychosocial influence and top-down causation, PCH effectively criticises an orthodoxy with roots back to 17th and 18th Century philosophy.

In the multi-disciplinary research network CauseHealth, based in Norway, philosophers, practitioners and medical researchers have recently joined forces to explore the relationships between ontology, scientific methodology and clinical practice. We will come back to the specific aims and discussions that took place during the first network meeting this last October.

The network has both critical and constructive objectives. The critical part is to show how the quantitative, statistical and homogeneous approach of evidence-based methodology and practice fits perfectly with certain philosophical views on causation and probability.

In a pilot project for CauseHealth [1], a connection was shown between philosophical theory and the scientific methods of EBM. For instance, uncontrolled observation studies are related to regularity theory and RCTs and other comparative methods suggesting that causation is conceptually linked to difference-making, both of which

are inspired by David Hume's analysis of causation from 1739.

Furthermore, large-scale statistical data are thought to be more or less directly applicable to individuals, something that suggests a philosophical commitment to frequentism. This is the view that probabilities in individual cases are generated by the frequency of successful outcomes in a sequence of trials.

But does it really matter on which philosophical theories we base our scientific methods? Absolutely so, we argue. This brings us over to the constructive goal of CauseHealth: to provide a philosophical framework that is better suited for PCH with its focus on the individual. For this, the ontology of causal dispositionalism, developed by Mumford and Anjum [2], was chosen because it emphasises exactly those features that PCH sees as essential, but which are ignored or not sufficiently accommodated by EBM: complexity, context-sensitivity, holism, emergence and singularism.

A shift in medicine

One criticism against EBM is the excessive trust in statistical studies and the problem of applying data from homogeneous clinical trials to the complexity of human biology in its socio-cultural environment. Moreover, the over-emphasis in following algorithms and guidelines has resulted in a policy that discourages the use of practitioners' professional experience and autonomy in clinical situations. Rather than encouraging the practitioner to adjust the treatment to the individual patient, taking into

account the wider context unique to that person, EBM is motivated by the idea that the same treatment should be given to all, independently of individual variations.

PCH offers a perspective that seems innocent at first glance, but which is actually quite difficult to integrate with the medical model of EBM. In a previous Guest Editorial of the *Journal* [3] a complementation of scientific knowledge with the subjective experience of practitioners and patients is emphasised. It seems, then, that PCH should include something *more than* EBM, not something substantially different.

Still, the move from EBM to PCH is often presented as a replacement of the biomedical model with something else. In the first editorial of the *Journal*, Miles and Asbridge [4] stressed the need for a shift from the largely anatomico-pathological focus in clinical practice toward a wider anthropocentric approach, including psychological and emotional dimensions of health and illness.

We might say that the person-centered approach represents a change in perspective, zooming out from the level of biochemistry to include the whole human being in all its complexity and context. But this involves taking in features that were not previously thought to be medically relevant: values, expectations, preferences, relationships, hopes and fears.

The problem of multi-morbidity and heterogeneity

When we consider the whole person rather than just some of their body-parts, we can already see how existing methodology falls short. To deal with the complexity of data that is relevant for assessing an individual's state and establishing an effective treatment approach, we need new models. But such models are unlikely to arise out of a framework that does not first address these complexities.

The CauseHeath group addresses how to design and implement PCH models that allow us to bridge the gap between the scientific data available and the complexity of reality. When using primarily epidemiological and quantitative methods, we get a problem with cases of complex disorders, multi-morbidity and heterogeneity. This is particularly a problem when dealing with so-called medically unexplained symptoms (MUS), which amount to about 30% of the symptoms reported to general practitioners [5]. For these conditions medicine has not been able to find a common set of causes, a definite psyche-soma division, or even decide upon clear-cut classifications. Individual medical uniqueness is common for MUS, which means that population studies are of limited use.

Not only MUS, but any complex disorder is difficult to treat because of multiple causes: genetic, environmental and lifestyle factors, many of which are yet unknown. Most medical conditions are complex in this sense [6]. And since each patient has a unique combination of biological, social and psychological factors, they are also likely to be heterogeneous [7].

If we can find models and methods that are more suitable for dealing with complexity and heterogeneity, we will also be in a better position to deal with the complex nature of human health and illness in general [8].

The CauseHealth Network

Human health is an integration of all levels in nature: physiology, biology, psychology and sociology. The biomedical model falls short because it fails to acknowledge the psycho-social dimension as anything more than a manifestation of underlying biological or physio-chemical processes. If we take seriously the idea that health and illness are more than such processes, we also need to bring in expertise on these other dimensions of human life.

The Network CauseHealth brings philosophers, practitioners and scientists with the aim of addressing the issue of human health and illness from different professional perspectives. The group is multidisciplinary, with experts and practitioners from a variety of areas: philosophy of medicine, epidemiology, qualitative health research, person-centred medicine, public health science, disease ontology, medical humanities, MUS research (CFS, LBP, physiotherapy, neuro-biology, behavioural science in medicine, pharmacology, nursing, cancer research, mental health, probability theory and risk, autism, burnout, medical sociology, philosophy of psychiatry, medical research ethics, experimental psychology, phenomenology, paediatrics and philosophy of causation.

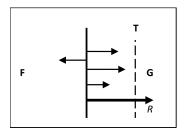
Report from the first CauseHealth meeting

At the first meeting of CauseHealth, held on the 12th and 13th of October, 2015 at the Norwegian University of Life Sciences, the aim was to describe the situation from our different perspectives, identify some problems and agree upon a way forward.

A new model of causation

Stephen Mumford and Rani Lill Anjum presented the project and their theory of causation, emphasising its features of complexity, singularism, context-sensitivity and holism as particularly useful for a more person-centered approach to healthcare. The vector model (Figure 1) was also introduced as a better way to represent causal situations, allowing representation of a threshold effect, additive and subtractive interference, multifactorial causation and nonlinear interaction. Each cause is represented as a vector tending towards a possible outcome, with multiple causes combining and represented as a resultant vector, *R*.

Figure 1 The Mumford-Anjum vector model of causation



The vector model was developed as an alternative to the neuro-diagram, which represents Hume's [9] standard mono-causal, two-event model of one cause, one effect and a causal relation between them (Figure 2).

Figure 2 The standard neuron model of causation



Here we get the impression that any intervention a, if it is a cause of b, should produce b in every case. This is also what the Humean theory of causation effectively claims. In contrast, the vector model shows a range of causal factors that tend either towards or away from the threshold effect.

The vector model makes it clear that no single cause is responsible for producing the effect, since it is only in interaction with other causes that the effect is brought about. Furthermore, it shows the possibility of causal interference, either by subtraction of a causal factor that tends towards the threshold or by adding a factor that tends away from it. A third important feature of the vector model is that it explains medical uniqueness and why the same intervention in two patients can give different results. If the same vector with the same intensity is plotted into two different contexts, the resultant vector will be different too. This illustrates the importance of tailoring the treatment to the patient's unique situation.

The rest of the meeting was divided into four discussion sessions, introduced by members of the Network.

Session 1 The problem

In the first session the problem of complexity and heterogeneity was addressed. Focus was directed towards the medically unexplained, complex diseases, multimorbidity and medical uniqueness.

Vegard Bruun Wyller presented the case of chronic fatigue as a typical case of comorbidity, often combined with chronic pain, insomnia, sensory hypersensitivity and cognitive impairment. He also addressed the controversies that this research has stirred up among the patient group,

which strongly favours a biomedical explanation to a biopsychosocial one. His own research was presented, where CFS is seen as a stress response, linked to a complexity of causal factors including genetic factors, critical life events, immune disturbances, personality traits and long-lasting infections.

After this, Elisa Arnaudo presented the comorbidity of fibromyalgia and depression, arguing that these illnesses are resistant to a model that studies either organic or psychological factors. A truly integrated approach should acknowledge the individual expression of suffering as emerging from the interaction of genetic and biographical history together with environmental issues.

The final presentation in this session was by Karin Mohn Engebretsen on professional burnout. She has found that burnout is a process in four phases: achievement, pressure, psycho-somatic collapse and personal reorientation, with shame as a central mechanism in this process.

Session 2 The method

The second session was about the limitations of scientific methods and models, including issues of reproducibility, external validity and idealisations *versus* reality.

Roger Kerry opened the session by noting that evidential priority in EBM is given to methods that explore large populations, which is supported by a Humean account of causation, while health decisions at the individual level seems to require something more than this. A different model of causation is needed to bridge the inferential gap between population level evidence and clinical decisions.

Samantha Copeland argued that her analysis of serendipitous discovery demonstrates the importance of narrative, context and social-epistemological values to scientific progress. Innovation often occurs by analogies from single cases to generalizable conclusions, not by deductive reasoning. Therefore, case reports that emphasise context and narrative should play a vital evidentiary role in the health sciences.

Elena Rocca ended the session with an overview of the current difficulties in preclinical medical research with genetically identical animal models. While on one hand reduction of complexity and isolation favours the repeatability of experiments in the lab, it often reduces the translatability of the results to the real world.

Session 3 The model

In the third session we moved over to the biomedical model and its underlying ontological assumptions. Issues that were addressed included reductionism, dualism and the biopsychosocial model.

Linn Getz drew attention to the fact that while medical specialisms and clinical guidelines are generally tailored for single diseases and universal treatment, multimorbidity seems to be the rule rather than the exception. She argued that a new model must include an ethically informed epistemology, integrate biology and

phenomenology and acknowledge the importance of lived experience and meaning for health and illness.

The second presentation was by Michael Loughlin, who argued that a genuinely holist approach to health might have to involve a re-examination of a number of dichotomies, not only mind-body dualism: objective and scientific *versus* judgment and opinion, fact *versus* value, generalisable and repeatable *versus* context-specific and unique.

Session 4 The practice

The final session was focused on practice and the clinical situation. Two presentations were given.

Matthew Low showed how using the vector model in clinical practice as a tool for person-centered education and patient communication has allowed him to include a wide range of causal factors from the patient's story and, in collaboration with the patient, to evaluate which of these it was possible to change.

The last presentation was given by Stephen Tyreman, who linked the notion of health to function and agency and illness to lack of such. He presented what he called an anthropo-ecological model for productive agency, showing how health is affected by a wide range of personal capacities, responsibilities and contextual resources and challenges.

Conclusions of first meeting

After the sessions, a discussion followed where the group summed up some central lessons from the meeting:

- We lack adequate tools for handling the complexity of individuals, illness and evidence.
- We should avoid reduction to a single method, or at least we need more flexible methods.
- Specialists from different disciplines need to cooperate in order to best meet the complex needs of the patient.
- A correct understanding of biology includes the psychosocial. The biomedical model overlooks that biology is saturated with meaning.
- Phronesis, judgement and clinical experience must be given high epistemic value, since it is only in clinical situations that different types of evidence can be evaluated as a whole.
- Personal experience should be at the centre of a medical model.
- Theory is important in medicine. It is not sufficient to show how often an intervention works. We also need to understand how and why it works.

 The question of whether an intervention works occurs within a method, which might bring its own criteria of success. A challenge is to avoid relativism or "anything goes".

To follow up on the conclusions of this discussion, the next CauseHealth event will be 'N=1 - Causal reasoning and evidence in clinical decisions', to be held at NMBU in Norway on 12 & 13 January 2016.

Conflicts of interest

The authors declare no conflicts of interest.

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