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The eccentric manners of explanatory models: towards an account of perspectival mosaic unity in psychiatry

In this thesis, I advocate a pragmatic, relativistic, post-dichotomous and enactive version of mechanistic explanation in psychiatry. I also advocate augmenting mechanistic explanations with the methods from network analysis. Use of network analysis combined with a mechanistic approach can aid us in detection of the parts and help uncover the interactions that together comprise psychiatric phenomena. The account, which is outlined rather roughly, follows in response to the central question of how we can account for the integration of different perspectives on a target phenomenon in psychiatry.

Below, I outline the central questions which I set out to address and the contents of the chapters that follow. As I discuss in the Chapter 9, my findings during the project lead me to reformulate the second of the central questions. The central questions with which I began, and to which I refer in the chapters to come, are:

I.) What is the utility of the biopsychosocial model at the level of a) philosophy, b) science and c) clinical practice?
II.) How can we understand the use of the biological, psychological and social levels of explanation in science and account for the coherence and possibilities for integration of these different levels of explanation?
III.) What are the implications of the answers to the above questions for the scientific explanation and clinical understanding of the phenomenon of apathy?

Chapter 2: in order to stay as close to psychiatric practice as possible, I survey a literature sample. Because a large proportion of the patients I encounter meet the diagnostic criteria for schizophrenia and because the negative symptoms are a huge source of misery, the literature sample zooms in on apathy in schizophrenia. I offer short introductions of schizophrenia, the negative symptom cluster and apathy in particular before outlining my search strategy. Ten articles that meet my search criteria are then briefly summarized with a view to detecting possible philosophical assumptions at play and evidence of biomedical or biopsychosocial thinking. I show how evidence of specific medical explanatory models is indirect and meagre at best.

In Chapter 3, the biomedical model is introduced. It is a model with origins in the 19th century and with even earlier roots that date back to Cartesian thinking. The
biomedical model’s main limitation is its adherence to reductionist methods: the model seeks to explain diseases solely in terms of pathophysiological mechanisms and thus neglects other facets such as interpersonal and psychological aspects. The problems of reductionism are the theme of the remainder of the chapter. Different contexts in which the term features are examined and various types of reductionism are distinguished. I advocate continuing the search for a model that avoids reductionism and thus does not seek to explain by reducing all phenomena to occurrences at a particular level of organisation such as the biological level.

Chapter 4 introduces the biopsychosocial model. The model was developed by Engel and first presented in his 1977 article. Engel expands the model in many of his later publications and this literature is examined. I discuss how the biopsychosocial model aims to avoid the problems of biomedical thinking. The model is inspired by the doctrine of dynamical systems thinking and Engel himself refers to the writings of von Bertalanffy. I introduce this doctrine and some different variants of systems theory. The degree to which systems theory is incorporated in the biopsychosocial model to help integrate the different domains turns out to be somewhat limited. The chapter concludes with a short comparison of the biomedical and biopsychosocial models.

The fifth chapter delves into the shortcomings of the biopsychosocial model, which, as I note is the model that is commonly taught to medical students. The first line of critique is that Engel does not differentiate between clinical and scientific practice – in fact, he deems this a false dichotomy. I instead embrace the notion of different roles and perspectives and show that differentiating between them is a heuristic tool that can help avoid confusion. On Engel’s account, it is unclear whether the model is intended as a clinical or scientific model. A second objection is that Engel alludes to, but does not further develop, the ideas of systems theory in the biopsychosocial model. He offers little guidance on how to bring together the biological, psychological and social domains. Third, the model fails to help provide a definition of what disease is in the first place and risks inviting an over-inclusive interpretation of “disease”. Finally, I address the question of whether the biopsychosocial model should not include other dimensions, such as an existential dimension. In the concluding section, I address the first central question of the thesis, proposing that the biopsychosocial model is best regarded as a guide for clinical practice rather than a scientific model.

Having discussed the two most common medical models of explanation, Chapter 6 turns to more abstract, philosophical models of explanation. Key facets of explanation, such as description, prediction and intervention are discussed. I borrow heavily from the philosophers Wesley Salmon and Carl Craver by adopting their five constraints
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on a good explanation. Put simply, the five constraints are aimed at detecting causal relevance. Armed with these constraints, I discuss three philosophical models of scientific explanation: the Covering Law model, the Representation model and the Unification model. I offer examples from the field of psychiatry and show how these models fail to meet the five constraints. There are of course many more models but I focus on these three because a more exhaustive treatment is beyond the scope of my project. In the following two chapters, I discuss two further models in greater detail as they hold more promise for the field of psychiatry: mechanistic explanation and a statistical method called network analysis.

Chapter 7 offers an in depth exploration of mechanistic explanation. The chapter zooms in on the notion of levels, their appearance in the psychiatric literature and also on Craver’s idea of “levels of mechanism”. Furthermore, I investigate what a “mechanism” is, showing that it consists of “parts” or “entities” and their interactions. The accounts of different mechanistic thinkers (e.g. Glennan, Machamer, Darden, Bechtel, Abrahamsen, and Richardson) are compared and the consequences of the differences are discussed for the field of psychiatry specifically. I revisit the apathy literature sample, searching for evidence of mechanistic thinking, and provide examples of proto mechanisms. I take issue with some inconsistencies within Craver’s account and also with the aspects of the accounts of mechanistic explanation that hinder the model’s applicability in the field of psychiatry. The most important problem for mechanistic explanation is the lack of robustly detectable, physiologically plausible parts in mechanisms for psychiatric phenomena. I suggest that there is an inherent fuzziness in psychiatry.

Network analysis, the topic of Chapter 8, is a statistical approach that entails using mathematical analyses to identify relations between phenomena. However, before any data can be represented in terms of a network, it must be collected. Psychometrics is the field that studies how mental phenomena may be measured. I argue for the importance of sound psychometrics, highlighting failings such as statisticism, the measurement problem and the idea of the latent variable. Next, three types of network that are most commonly used to model psychiatric phenomena are distinguished. In order to determine whether network analysis can be regarded as a model of scientific explanation, I investigate if the three types of network can meet the constraints for a sound explanation introduced in Chapter 6. I conclude that none of them can fully meet the requirements imposed by Craver and adopted by me. I include a discussion on Mechanistic Property Clusters, a hybrid between mechanistic thinking and statistical approaches. I then proceed to further develop the idea of a hybrid model for explanation that draws upon both Mechanistic Explanation and Network Analysis to explain a psychiatric phenomenon.
Chapter 9, titled “Perspectival Mosaic Unity” serves to bring together the findings from the previous chapters and attempts to answer the remaining questions fuelling this project. I begin by offering an enactivism-inspired set of basic assumptions upon which I base my own attempt to offer a hybrid account of explanation that is suitable for psychiatry. Many dichotomies that are pervasive in traditional philosophy and also in scientific practice, are afforded the status of heuristic tools rather than absolute categories (for instance the ontic-epistemic divide and the subject-object divide). I argue that the perspectival approach, based on the inescapable reciprocity between self and world at which I thus arrive, runs deeper than the dichotomy-dependent notion embraced by Craver. I finally return to the central questions of the project. Given my findings, I adapt the phrasing of the second central question: How can we account for the integration of different perspectives on a target phenomenon? In answer to the question, I explore the idea of perspectival integration as a means to develop a more unified scientific landscape.

I then move on to the third central question: what are the implications of the answers to the above questions for the scientific explanation and clinical understanding of the phenomenon of apathy? I draw on an example from the scientific literature to illustrate the implications of my refusal of biomedical and biopsychosocial thinking. I instead offer a post-dichotomous account of mechanistic explanation and supplement it with network analysis which is more suitable for the thorny issues presented by the field of psychiatry. In this account, I suggest that the use of statistical methods can help determine which parts are relevant to a mechanism in the first place by using statistical methods to identify possible causal relations.

Finally, I conclude the last chapter and my thesis with reflections on how my findings have affected my clinical practice and suggest that an adapted account of mechanistic thinking can be of help in formulating a descriptive diagnosis and making individualised treatment plans with patients.