What's in a model?

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Commentary to Borsboom et al. "Brain disorders? Not really... Why network structures block reductionism in psychopathology research".

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"Brain disorders? Not really ... Why network structures block reductionism in psychopathology research." Authors: Denny Borsboom, Angélique Cramer, and Annemarie Kalis

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Abstract

Network models only block reductionism about psychiatric disorders if models are interpreted in a realist manner, i.e., taken to represent "what psychiatric disorders really are". A flexible and more instrumentalist view of models is needed to improve our understanding of the heterogeneity and multifactorial character of psychiatric disorders.

Commentary

What's in a model?

This commentary targets the claim that "if mental disorders are indeed networks of causally related symptoms, reductionist accounts cannot achieve [success]" (Borsboom et al. 2018, p.3). Borsboom, Cramer, and Kalis pose that psychiatric disorders are in essence networks of symptoms rather than brain states, and that this rules out reductionism. This claim rests on a particular realist interpretation of disease models, namely that models represent what diseases in essence are. We take issue with this interpretation. Models should not be understood as representing the true nature of psychiatric disorders, but as tools to improve our understanding of different aspects of these disorders. A realist interpretation of models might be detrimental for progress in psychiatry, as it may invite exclusivity in the use of models.

Before we engage in argument, we want to align with Borsboom et al. on their anti-reductionist and multifactorial conception of mental disorders. We share many of their views: an exclusive focus on brain processes will hamper progress in psychiatry, because most psychiatric disorders result from interacting biological, psychological, and environmental factors (1). Research focusing on all of these levels will thus benefit psychiatric science more than an exclusive focus on underlying brain processes. In addition, many psychiatric disorders describe a heterogeneous group of patients in terms of symptoms, etiology, and course of illness (2). To deal with the heterogeneous and multifactorial nature of psychiatric disorders, psychiatry needs flexibility in its research methods. That is exactly why we are critical of Borsboom et al.'s line of reasoning. The implicit realism in the arguments of Borsboom et al. might invite another kind of exclusivity, namely in the use of network models. We believe this might have similar adverse effects on progress in psychiatric science.

Borsboom et al. use a realist view about models

In their first argument against reductionism, Borsboom et al. move from two observations (psychiatric symptoms are highly correlated, and no single biological cause has been found to explain these relations) and a common sense notion of causality (it seems natural that, e.g., insomnia and fatigue are directly causally related) to a preference of network models over latent variable models. They then argue that if psychiatric disorders are indeed symptom networks, reductionism will fail, because in symptom networks there is no common cause. However, this argument only works if we adopt realism about the models.

First, concerning latent variable models in psychiatry, the authors are right to point to a strong association between such models and reductionism. They invite a physicalist and causal interpretation: the latent variable gets the role of cause – hence the terminology of "common cause models" – and then this cause is imagined to have some physical realizer, e.g., a neurobiological process. However, the association between latent variable models and reductionism is by no means a conceptual necessity (3). It is perfectly possible to use latent variable models without interpreting the

latent as pointing to an entity in the world, let alone as cause. Moreover, even if we interpret the latent as causal, we can still avoid physicalism if we drop the specific physical realization of the latent cause. Thus, the so-called common cause models are only inviting reductionism if we give them a particular realist interpretation.

Second, consider the claim that network models make physicalist reductionism unfeasible. As the authors briefly indicate themselves, the use of network models is in principle compatible with a reductionist viewpoint but, taking symptom networks as realist representations, this looks rather contrived (p.19-20). However, we could employ network models in a more pragmatic manner, namely as instruments of prediction and control, without committing to the idea that they provide a picture of the world. Then it does not say much that network models lack a representation of biological causes: a complete picture is not expected. Anti-reductionism only follows from network models if those models are interpreted as providing a realist account.

Summing up, latent variable models are only reductionist, and network models are only anti-reductionist, if we give those models a realist reading. Rather than maintaining realism in the service of an argument against reductionism, we would do better to reconsider this realism itself.

Alternative view on status and function of models

So how should we think of the status of models? Following a well-developed line of thinking in the philosophy of science, we think that models function as instruments of investigation instead of being exact representations of reality (4). Statistical models, but also other models - the Bohr model of the atom, the double helix model of DNA, general equilibrium models of markets – typically involve aspects of both theory and data. A model is an autonomous tool, because it is partially independent of both of them. Precisely because models are partly independent of both, they can be used as instruments of exploration in both domains. As such they play a mediating role, helping us to connect empirical facts about disorders to theoretical accounts of them, thereby performing a variety of tasks.

In the context of psychiatry, this means that psychiatric disorders must not be thought of as being symptom networks, nor as being brain disorders, but that different aspects of psychiatric disorders can be investigated using different types of models. This ties in with our general idea that our attitude towards models must be guided by what will bring psychiatric science further (5). For example, network models are well-suited to investigate complex dynamic interactions among multiple variables in the development of psychopathology (6). Latent variable techniques instead may be used to achieve data reduction, and to explore underlying latent factors (3). Thinking in terms of a common cause led to the recent discovery of anti-NMDA receptor encephalitis – a rare auto-immune disorder that leads to psychotic symptoms and agitation (7). An instrumentalist view of models offers more flexibility for switches between, and divergence among research strategies, and this is needed to improve our understanding of the heterogeneity and multifactorial character of psychiatric disorders.

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