Project description

Meaning in multiplex: towards a network understanding of mental illness
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1. Introduction
The gap between fundamental neuroscience and psychiatric practice is widening rapidly. Neuroscientists are increasingly pressured to translate their results into meaningful clinical applications, while psychiatrists are struggling to translate the clinical problems they encounter into adequate neurobiological models.

For a long time, it was assumed that the relation between neuroscience and psychiatry could be understood in terms of reduction, and that psychiatric symptoms, signs and other features of mental disorders could be translated into neural processes and mechanisms. This assumption went hand in hand with an essentialist view of mental disorders as discrete neurobiological entities. However, there is a growing recognition that the reductionist notion of translation and the neurocentric view of mental illness are untenable. As Adam (2013, 417) puts it, “[d]espite decades of work, the genetic, metabolic and cellular signatures of almost all mental syndromes remain largely a mystery”.

In spite of this, brain reductionism still remains very influential in scientific and public debates on mental disorder. For example, the Research Domain Criteria (RDoC) initiative, launched by the National Institute of Mental Health, aims to transform the current DSM-ICD framework of psychiatric classification into a biological system that conceptualizes mental disorders as brain dysfunctions (Lilienfeld & Treadway 2016). The core idea behind this initiative is that mental disorders literally are brain disorders: “As new diagnostics will likely be redefining ‘mental disorders’ as ‘brain circuit disorders’, new therapeutics will likely focus on tuning these circuits” (Insel & Cuthbert 2015, 500). Indeed, psychiatrists are increasingly encouraged to discuss psychiatric problems with their patients in terms of brain deficits and to integrate “a modern neuroscientific perspective into every facet of clinical practice” (http://www.nncionline.org/clinic/). Because brain studies are often presented as having revolutionary implications for our understanding of mental health, there is a tendency among the media and the general public to single out the brain as the real cause of mental disorder and the privileged locus of potential interventions (De Hue et al. 2017).

2. The multiplex model of mental illness
The PhD project will contribute to the development of a novel non-reductionist account of mental illness that does justice to its sociolinguistic embeddedness and its network-like nature. The guiding hypothesis behind the project is that mental illness cannot be understood in terms of an essence - a latent (neurobiological) variable that underlies the symptoms associated with the disorder. Instead, mental illness should be conceptualized as a relatively stable set of interacting...
variables in a multilayer network or ‘multiplex’. The interactions between these variables might include interactions between the symptoms themselves. For example, in depression, insomnia predisposes to tiredness, and guilt predisposes to suicidal ideation. In schizophrenia, hallucinations can often produce delusions. Phobias lead to avoidance, which prevents habituation to the feared stimulus (Kendler et al. 2011). The individual symptoms of a mental illness often interact so as to sustain the other symptoms characteristic of the disorder. Because the symptoms are mutually re-enforcing, the disorder will appear as a more or less stable set of traits. But there is no underlying essence that is responsible for the clustering of the symptoms. Thus, as depicted in Figure 1, in depression suicidal ideation (S1) might be caused by both depression mood (S2) and feelings of guilt (S3). However, the set of interacting variables that are constitutive of the mental illness typically span multiple layers. Beck and Alford (2008), for example, explain how a depression mood (P1) can create automatic and reinforcing negative thoughts about the self (P2), the world (P3) and the future (P4) (what Beck calls the ‘cognitive triad’), which in turn can further exacerbate the depression mood (P1). Furthermore, there might also be relevant neurobiological variables that are (partly) constitutive of depression. For example, Disner et al. (2011) suggest that in depression, negative signals from incoming stimuli induce hyperactivity in the thalamus, from the thalamus to the amygdala and on to the subgenual cingulate cortex, which relays limbic activity to higher cortical regions such as the prefrontal cortex (N1). Concurrently, hypoactivity in the dorsolateral prefrontal cortex, which is associated with attenuated cognitive control, impairs the ability of the dorsal anterior cingulate cortex to adaptively regulate the lower regions (N2). The net result of this process (N3) is increased awareness and processing of negative stimuli in the environment. Furthermore, Disner et al. (2011) also propose that in depression, the dorsolateral prefrontal cortex and ventrolateral prefrontal cortex have less regulatory influence on subcortical regions that are involved in memory (N4), and this facilitates the undesired recall of negative events.
Thus, as depicted in Figure 2, the variables S1-S3 not only interact and mutually reinforce each other, but they also interact with the variables P1-4 and N1-4 at lower layers of the multiplex. Of course, this hypothetical multiplex account of depression is extremely simplified and only partly complete. Some of the variables that play a role in the multiplex might still be unknown (e.g., (Sx)), and some of the interactions between the variables (e.g., between (Sx) and (N1)) might still need further empirical investigation. However, an important advantage of multiplex accounts over traditional neurocentric models of mental disorder is that they do not privilege one type of variables (e.g., neurobiological) over another (e.g., psychological). Furthermore, multiplex accounts can in principle accommodate very different types of variables – not just symptoms, psychological/functional processes and neurobiological mechanisms, but also environmental triggers, social norms and institutions, patient histories and subjective experiences. This makes the multiplex a very promising model to address the gap between neuroscience and psychiatry.

3. PhD project
The PhD project examines how a multiplex account of mental illness can contribute to closing the translational gap. The project is divided into three phases.

a) In the first phase, the PhD candidate will investigate what kind of explanation the multiplex model offers by situating it in contemporary debates on mechanistic explanation – the currently dominant framework for thinking about the philosophical assumptions underlying many areas of science, especially biology, neuroscience, and psychology. Proponents of mechanistic explanation typically define a mechanism as a structure performing a function in virtue of its component parts,
component operations, and their organization (Bechtel 2008). A neuroscientific explanation, then, is thought to describe how the orchestrated functioning of a neurobiological structure gives rise to, or “constitutes”, the phenomenon of interest. The mechanistic explanatory strategy has had considerable success in various neuroscientific subdisciplines, including molecular (Craver 2007; Machamer et al. 2000), cognitive (Kaplan and Craver 2011), and computational neuroscience (Kaplan 2011). Systems neuroscience, however, with its focus on the study of networks at various levels of brain organization, has proven to be a challenge. It is commonly accepted that mechanistic explanation involves structural and functional decomposition - breaking down a system into concrete parts and activities in order to identify the causal relationships that realize the phenomenon. But systems neuroscientists seem to abstract away from concrete parts and activities and instead focus on general properties of neural networks, such as robustness and functional redundancy. Indeed, it has been suggested that systems neuroscientists provide topological explanations, which aim to explain how a system can resist or react to various perturbations. Some have argued that these explanations can still count as mechanistic, as long as we are willing to consider a less restrictive notion of mechanistic explanation that focuses primarily on the identification of causal relationships (Zednik 2014). By contrast, those who emphasize the importance of structural decomposition in neuroscientific research have claimed that topological explanations are not explanations at all. The PhD candidate will investigate whether multiplexes can shed new light on the tension between mechanistic and topological explanation, given that they seem to qualify as topological explanations but at the same time can handle structural information about concrete parts and activities (such as information about anatomical connectivity obtained with diffusion-weighted magnetic resonance imaging).

b) The second phase of the project focuses on the question of how multiplex accounts can do justice to the fundamental situatedness and sociolinguistic embeddedness of mental illness. Here the starting point is the network approach, which has been proposed by Borsboom et al. (2018; see also Borsboom 2008, Borsboom and Cramer 2013). The PhD candidate will address two critical issues with this approach, and investigate how they can be dealt with by a multiplex account of mental illness. First, an important feature of mental disorders is that they involve intentional information (Kalis 2011). That is, descriptions of mental states such as beliefs, desires, and emotions indicate ‘what they are about’. We have beliefs about being persecuted, or about the floor being dirty. We have fears about spiders or germs. The symptomatology in psychiatric classification systems such as DSM-V frequently relies on such intentional information. For example, the symptom of ‘craving’ in alcohol use disorder is defined as “a strong urge or desire to use alcohol” (APA 2013). Borsboom et al. (2018) claim that we ‘make sense’ of the relations between the symptoms of a disorder by explicating the background of rationality in the person’s behavior, fears, desires and beliefs. But it is precisely this background of rationality that is often distorted or even completely lacking in patients that suffer from severe psychopathology. For example, even though patients with severe anxiety disorder know that their thoughts are irrational and thus have some insight into their illness, their capacity
for rational agency is severely limited in the sense that they fail to exercise any form of reasonable control over these thoughts (Glas 2003). The question for the multiplex account is how to account for the (lack of) rationality in mental illness.

Second, Borsboom et al. (2018) claim that some causal relations between symptoms are literally realized outside the person and depend on mechanisms in the environment. They discuss the example of the relation between excessive gambling and the desperate financial situations it leads to, both of which play an important role in the DSM-V diagnostic criteria for gambling disorder (APA, 2013). This relation, according to Borsboom et al. (2018) is forged entirely outside of the person, namely by the operational specifications of gambling setups (e.g., fruit machines, Roulette tables, etc.). Importantly, even if the desire to gamble is taken to be a mental state that is realized in the person’s brain, the operating characteristics of the fruit machine are not; and these operating characteristics realize the causal connection between gambling and the debts it leads to. However, the worry here is that this stretches the limits of the notion of mental disorder too far (called ‘cognitive bloat’). The challenge for a multiplex account of mental illness is to draw a principled line between those relations with the (socio-cultural) environment that should be included in the multiplex and those that should not.

c) In the third phase of the project, the PhD candidate will apply the multiplex account to an empirical case study. The case study will be chosen based on two main criteria: (a) the PhD candidate’s interests and research background, and (b) the expertise of the collaboration partners. Possible case studies include anxiety, addiction or autism spectrum disorder.

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