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Taking an engineer's view: Implications of network analysis for computational psychiatry

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Abstract

An engineer's viewpoint on psychiatry asks: *What are the failure modes that underlie psychiatric dysfunction?* And: *How can we modify the system?* Psychiatry has made great strides in understanding and treating disorders using biology; however, failure modes and modification access points can also exist extrinsically in environmental interactions. The network analysis suggested by Borsboom et al. in the target article provides a new viewpoint that should be incorporated into current theoretical constructs, not placed in opposition to them.

Borsboom et al. challenge the notion that the brain should occupy a privileged position in mental health nosology and science, contending instead that symptom networks reflect the best units of analysis. However, psychiatry has made great strides in understanding and treating disorders using biology, and it is naïve to assume that because the model does not completely account for the full complexity, it is therefore useless.

Because all behavior arises from brain function, neurobiology is obviously critical for understanding psychiatric phenomena and not simply an example of “local reduction” of symptom networks. However, we think that the authors do have an important point that has not been incorporated well into current psychiatric reasoning: namely, that the trajectory of interactions with the external environment contains consequence chains that provide additional access points for treatment.

It is important to remember that psychiatric problems are not simply social constructs, but lead to real devastating consequences. For example, a patient with obsessive-compulsive disorder (OCD) unable to stop compulsive hand-washing is damaging their skin, leading to an increased risk of infection (Swedo et al. 1989) – certainly not something one would want anywhere, especially in a plague situation.

To help patients overcome their difficulties, we take an engineer's point of view, which asks two questions: (1) What are the failure modes that underlie psychiatric dysfunction? and (2) How can we modify the system?

The concept of a failure mode comes from reliability engineering – it recognizes that the structure of a process has specific ways in which it can fail. If we understood how environmental and neurobiological effects lead to behavior, then we could identify how this interaction can fail (Redish 2013; Redish et al. 2008; MacDonald et al. 2016). We agree that it is unrealistic to think that only the brain impacts disease – these failure modes may also be occurring outside of the individual’s brain, that is, in the extended world.

Neurobiological effects on behavior have to be understood computationally, as information processing (Redish 2013). While these definitely have environmental interactions, the way to understand this is through quantitative theory. We believe that the network analysis that the authors propose here can provide an important contribution to quantifying the brain-environment interaction in computational models. Once you go to these theoretical models, the problem of reductionism falls away. The question is not whether an emergent phenomenon exists at a lower level, but rather how lower-level effects combine to produce the emergent phenomenon. This more-nuanced scientific discussion can be seen in the emergent phenomenon of traffic: Traffic on city streets is something that emerges only from structure. However, we can derive very accurate models of traffic patterns from understanding the underlying phenomena (physical structures of cars, timing of traffic lights, reaction times of typical drivers, etc.) (Seibold 2015). The fact that traffic is an emergent network phenomenon does not mean that we can’t reduce it to a complex interaction of other parts. It just means that we need to understand both the parts and their interaction.

The target article provides an important insight that has not been included in current theoretical conceptualizations: that some of these failure modes arise not from neurobiological failures, but from environmental failures that neurobiological systems are unable to cope with. Thus, for example, sleeplessness causes anxiety (Ohayon & Roth 2003), but anxiety can cause sleeplessness (Marcks et al. 2010), which could lead to a spiraling dysfunction. Intrusive memories in post-traumatic stress disorder (PTSD) are episodic, not semantic (Shay 1994; Talarico & Rubin 2003), and may arise from insufficient neural memory consolidation during sleep (McClelland et al. 1995; Payne & Nadel 2004; Rasch & Born 2013; Redish 2013). Even if normal consolidation processing were intact, a patient with PTSD unable to sleep might be unable to consolidate memory due to the lack of sleep and would remain subject to episodic flashbacks. If this scenario were true, it would mean that concentrating research looking for dysfunction in the neural system’s underlying consolidation may be less fruitful (practically) than finding ways to facilitate an intact consolidation process.

Similarly, one could imagine scenarios in which treatment can use intact neural systems to bypass dysfunctional failure modes through modification of environmental components. For example, we have proposed that contingency management (a behavioral treatment in which addicts are rewarded monetarily for not taking drugs [Petry 2011]) works, in part, by shifting the decision process from a dysfunctional habit-based system into a non-dysfunctional deliberative system (Regier & Redish 2015). If this hypothesis is correct, then this treatment is bypassing, not curing, the brain dysfunction. Thinking about the interaction of brain and world provides alternative controls for treatment. Importantly, however, this proposal requires understanding the neuroscience underlying the information processing because it

implies that we need to test for the intactness of the deliberative neural system before assigning someone to contingency management.

While extrinsic causal chains are discussed in the psychiatric literature, as in the widely taught “biopsychosocial model” (Frances 2014), they remain non-computational word-theories, which make them difficult to implement practically. Network analysis provides a mathematical toolbox to study these brain-behavior interactions, whether the causal chains arise from brain or environmental dysfunction. Importantly, network analysis is not atheoretical, and the network analysis requires hypothesized constructs. Network analysis applied to a taxonomy of DSM-based symptoms will produce one answer (Borsboom et al. 2011; American Psychiatric Association 2013), while a network analysis run over the Research Domain Criteria (RDoC; Insel 2014; Flagel et al. 2016; National Institute of Mental Health, 2018) or decision-making constructs (Rangel et al. 2008; Redish 2013; Redish et al. 2008) will produce another. It will require collaborations with experimental and clinical psychiatrists to determine which of these taxonomies provide better explanation of the data and whether these tools can aid in practical psychiatric treatment.

Psychiatry has made tremendous progress over the last century, and computational psychiatry has opened up new ways of understanding the interactions of brain and behavior over the last decade (Huys et al. 2016; Redish & Gordon 2016). We think that network analysis has a part to play, but it needs to be incorporated into the current theoretical constructs, not placed in opposition to them.

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