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Evolutionary-developmental modeling of neurodiversity and psychopathology

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Abstract

Modeling the extremes of mental/emotional conditions requires explicit account of evolutionary-developmental sources of human neurodiversity, not merely psychopathology. The target article's approach could be improved by incorporation of a hierarchical scheme wherein mental/emotional infrastructure interacts across differentiated layers of function. The notion "symptom networks" thus calls for differentiation into hierarchically interacting components of mental/emotional evolution and development.

Main text

Borsboom et al. argue that content of thoughts interact to yield psychiatric disorders, proposing that psychiatric disorders are best seen as interactions among "symptom networks". The inherently abstract nature of these mental/linguistic interactions, and the "multiply realized" nature of mental states reveals that the mental states are, according to the authors, detached from biological foundations. Similarly it is claimed that symptoms themselves cannot be reduced to being direct consequences of a brain disorder. Thus thoughts and sentences associated with a disorder are seen to operate at a level of their own, one that cannot be reduced to description as, for example, the neural impulses of a brain state. My view is different. I think recognition of the abstract nature of mental/linguistic events does not diminish the importance of the neural impulses that, at another level, form the infrastructure *for* those events.

A more complete approach to modeling requires recognition that life is irreducibly structured in many abstract hierarchical levels, as explicated a half century ago by Michael Polanyi (1968). In this perspective, the levels of function must be seen as real, just as the authors' argue that schizophrenia and other conditions are indeed "real patterns" (Dennett, 1991). But the target article does not reflect the irreducible natural hierarchical levels needed to characterize the mind's underpinnings. I agree that the content of paranoid thoughts (symptom₁) cannot be reduced to the neural impulses associated with them. Neural impulses corresponding to paranoid thoughts may be driven at least in part by a state such as anxiety (symptom₂), which is also not reducible to the neural impulses upon which anxiety is grounded. Further, anxiety may be driven by hormonal imbalances (symptom₃). The three symptoms are related hierarchically, but the explication can go deeper. Hormone imbalance is surely founded on a system of gene expression driving imbalanced hormone production (symptom₄?). Gene expression similarly cannot be reduced to genetic makeup (symptom₅?),

but genetic makeup clearly limits gene expression. The implied hierarchy outlined here has five naturally-ordered levels, but many more are surely involved. And yet each level can be driven by levels above it. Paranoid thoughts can feed anxiety, which can feed hormone imbalance, etc. The target article's reasoning, represented in Figure 2, includes no account of hierarchical relations among levels of function, but instead treats brain states, symptoms, and environmental influences at a single level.

The approach has the advantage of emphasizing multiple realizability, the idea that there is no one-to-one mapping between brain states and symptoms, and no single "common cause" for symptoms. The authors persuasively point out that much effort has been wasted in pursuit of oversimplified expectations regarding roots of mental disorders. Still, the suggested alternative is itself oversimplified in my opinion. An important deficiency is that the terms symptom and brain state in the article are undifferentiated with regard to level of function. The terminology imposes an arbitrary boundary among levels, only lower levels of function being treated as biological. However, the more fundamental issue is that failure to directly acknowledge the hierarchy of levels squanders the opportunity to reflect the mind's hierarchical structure and the origin of the mind in evolution and development. A key feature of any model of mental disorders is its ability to predict, across development, the risk of mental disorder, and this implies a critical need to represent evolved and developmental foundations.

Natural selection and developmental processes are organized around functions of life, for example, the tendency to experience states such as fear. Such functions are abstract, but are nonetheless real because they constitute organizing principles, subject to selection pressures, corresponding to complex neural events. Furthermore all the levels of function considered here have deeply conserved features, just as it has been dramatically demonstrated that organization of life includes foundations in unicellular life that are reflected as conserved operations and characteristics in all multicellular forms (Carroll, 2005; Grant, 2015; Hills, 2006). Psychiatric conditions are grounded in the biology of humans and that of ancient life forms.

By suggesting the primary focus of psychopathology should be symptom-symptom networks, the target article radically de-emphasizes origins, presenting an undifferentiated list of symptoms and their interactions, with no explanation for how symptoms arise, why they persist, why they are heritable, or why, for example, psychotic symptoms arise predominantly in post-adolescence (Hare et al., 2010; Kessler et al., 2007), but reliably earlier in men than women (Skene, Roy, & Grant, 2017). In addition it tends to isolate individuals diagnosed with disorders from the neurodiversity of the human population of which they are a part, because it refers to behavior patterns as symptoms without indicating how one should treat normal behavior that resembles the symptoms. This isolation is undesirable given the tendency for psychiatric disorders to occur at higher than chance levels in proband families and for the same families to include higher than chance occurrence of other psychiatric conditions (Clementz et al., 2016). Furthermore unaffected members of these families typically show subclinical patterns. The target article's insistence on the term symptoms in its modeling creates an arbitrary boundary between behavior in affected and unaffected individuals.

In a dynamic systems evo-devo approach (Gottlieb, 2002; Müller & Newman, 2003; Newman & Müller, 2000), abstract levels of biological function are primary objects of modeling, with focus on interactions across the hierarchy. This approach facilitates prediction of risk for disorders, by focusing on evo-devo pathways. My colleagues and I have focused on origins of language and ways that a hierarchical scheme is required to characterize evo-devo foundations both of language itself (Oller, Griebel, & Warlaumont, 2016) and of divergences from typical development (Oller et al., 2010; Patten et al., 2014; Warlaumont, Richards, Gilkerson, & Oller, 2014).

In the attempt to combat oversimplified portrayals of psychiatric conditions, the authors argue for isolating thoughts as sources of disorder from the biological foundations that yield thoughts. I agree that thoughts are not merely neural impulses. Still, it is preferable for modeling to represent thoughts explicitly in relation to the developmental and evolutionary foundations that make them possible.

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