Getting to the bottom of things:

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Published in:
Behavioral and Brain Sciences

DOI:
10.1017/S0140525X18001097

Publication date:
2019

Citation for published version (APA):
O, J., & Brüne, M. (2019). Getting to the bottom of things: The value of evolutionary approaches in discerning the origin of psychopathology. *Behavioral and Brain Sciences*, *42*, [e18]. https://doi.org/10.1017/S0140525X18001097

Abstract

The network approach as a novel way of understanding psychopathology has helped address some of the issues associated with traditional biological interpretations. Nonetheless, it has failed similarly in explaining the fundamental etiology of mental conditions – a persistent conundrum that arguably could be addressed adequately only by evolutionary formulations, specifically evolutionary mismatch and life history theories.

Main Text

Borsboom and colleagues provide an informative account of the challenges inherent in the widespread adoption of traditional biology-oriented explanations of mental conditions. Nonetheless, although the network approach may offer a more integrative depiction of psychopathology, we contend that it is similarly based in part on an impoverished view of the term "biological," as is generally the case in medicine and psychology (Brüne 2016). Specifically, the prevailing undertaking of biological interpretations of mental conditions has focused largely on so-called proximate descriptions (e.g., biological mechanisms), while disregarding their "ultimate" or evolutionary dimensions (i.e., their phylogeny and adaptive value; Nesse 2013). When viewed through the lens of Tinbergen's framework encompassing both the proximate and ultimate levels of explanations of specific design features or traits, a couple key issues arise that render the network approach no more useful than the standard biological model in medicine and psychology (Nesse 2013; Brüne 2014).

The first claim is that the network approach offers a unique way of looking at psychopathology, whereby the synergism involving associated symptoms in a system of connections is purported to be key in the development of such conditions. Although it does make

sense that symptoms of many psychological conditions are likely to affect one another in a progressive and reciprocal manner, the network approach fails to clarify as to *why* this is the case. Is it because some symptoms are naturally influential on others? If so, why is this the case? The authors contend that the logical interpretation of intentional narratives behind certain symptoms could explain why. It begs the question, however, as to why such relationships are rational or comprehensible.

The second claim is that, in the context of a network approach, psychopathological features are believed to be engendered either as a result of individual developmental trajectories or as an outcome of environmental circumstances (i.e., akin to the proximate level). It is similarly ambiguous as to why these proposed antecedents effectively would bring about psychopathological symptoms. As an example, why exactly might exposure to thin models on media induce self-esteem issues and extreme eating habits in females? If it is because of social comparison, then why do humans compare so much? It is quite apparent that such explanations seem to pose more questions than answers.

We suggest that an evolutionary framework could plug these gaps by providing a comprehensive, ultimate account to complement such an otherwise detailed theoretical perspective relating to the proximate viewpoint. Specifically, two main evolutionary concepts, one relating to evolutionary mismatch (Durisko et al. 2016), and the other to life history theory (LHT) (Stearns 2000; Del Giudice 2014), respectively, are proposed to be highly valuable in shedding light on the actual origins of psychopathology.

For instance, the notion of evolutionary mismatch has been touted as a major underlying cause of a variety of mental conditions (Durisko et al. 2016). It refers to the manifestation of problems among people because relevant genes and cognitive/biological mechanisms (broadly

useful in the ancient context) could not evolve fast enough to match the dramatic metamorphoses in existential conditions that have materialized in the contemporary world (Grunspan et al. 2018; O 2018a).To illustrate, consider the proposition that depression is an outcome of residing in an evolutionarily novel setting whereby one's personal, occupational, and social experiences are drastically different from that of prehistoric individuals (Hidaka 2012). Such a contextual framework would have provided important elucidations for the existence of depressive symptoms (e.g., having a sense of hopelessness and anhedonia or entrapment in a seemingly hopeless situation), which the network approach might merely attribute to the occurrence of a precipitating event (e.g., failing an exam). Although it is conceivable that an exam failure indeed could play a role in the emergence of depression, such a proximate explanation precludes a sufficient understanding of why one is susceptible to feeling hopeless (and subsequently, to developing depression) following such an event. Apart from banking mainly on rationality and the general acknowledgement of extrinsic influences as is the case with the network model, Hidaka's (2012) theorization (e.g., the existence of evolutionarily novel inadequacy of social support in the current context) uniquely could explain why contemporary humans may be more vulnerable to develop the condition. Similar formulations relating to evolutionary mismatch likewise could describe the pathogenesis of a wide assortment of many other disorders, ranging from animal phobias (O 2018b) to schizophrenia (Abed & Abbas 2011).

Likewise, LHT is believed to be comparatively useful in deciphering the etiology of psychiatric conditions (Del Giudice 2014), because the concept is not only valuable in comparing species but also in explaining within-species variations (Stearns 2000). It is a concept derived from behavioral ecology which focused on the adaptive tradeoffs (e.g., a faster LH strategy involving spreading meagre resources across many offspring who will experience earlier sexual

maturity and a higher mortality rate vs. a slower LH strategy encompassing heavy investment in a few offspring with sexual maturity/death occurring at a later age) in relation to the nature of external circumstances (e.g., residing in a highly dangerous vs. predictably safer environment) as a means to realize favourable procreative outcomes (Stearns 2000).According to this approach, psychopathological problems are manifestations of either a slow (e.g., autism) or a fast LH strategy (e.g., attention deficit hyperactivity disorder [ADHD]) (Del Giudice 2014). Although a network model would argue that a child develops ADHD because of parental neglect, for instance, LHT can explain why the condition may emerge in the context of gene-environment correlation, which is fully compatible with the mismatch approach. For example, some psychological traits nowadays associated with ADHD were once adaptive in harsh and uncertain environments in relation to survival and reproduction over much of human evolutionary history. These traits are less profitable in the evolutionarily novel world and may become "symptoms," depending on the quality of parental input (Bakermans-Kranenburg et al. 2006).

Taken together, we contend that a combination of the LHT and the evolutionary mismatch approach, to name just two major evolutionary concepts, would provide a fundamental framework to complement the network model in understanding psychopathology.

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