EDITORIAL

Networks and Nosology in Posttraumatic Stress Disorder

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Psychiatric symptoms do not co-occur randomly; some are more likely to covary than others. What accounts for such syndromic clustering?[1] One explanation holds that an underlying categorical disease entity is the common cause of the emergence and covariance of the symptoms that reflect its presence. Hence, major depression can be the common cause of anhedonia, insomnia, and fatigue just as a malignant lung tumor can cause bloody sputum, dyspnea, and chronic cough.[2]

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Another explanation holds that symptoms are reflective of underlying dimensions such as neuroticism. Despite their differences, proponents of both categorical and dimensional models of mental disorder agree that a latent entity causes symptom emergence and covariance.

Unfortunately, these models often prove problematic for conceptualizing psychopathology.[3] First, justifying a latent entity as the common cause of symptom covariance requires satisfaction of the axiom of local independence. That is, symptoms must be uncorrelated with one another once one conditionalizes on the presence of their underlying common cause. Yet a moment's reflection exposes the implausibility of this assumption. Causal connections between symptoms abound in psychopathology, as every clinician knows. Insomnia produces fatigue that impairs concentration in people with depression, obsessions prompt compulsions, binge eating triggers purging, and so forth.

Second, psychopathologists have rarely discovered the existential referent of latent variables postulated as the common cause of symptom emergence and covariance. Rare exceptions include the spirochete bacterium for general paresis of the insane and a third (or partial) copy of chromosome 21 in the case of Down syndrome. In view of the likely multifactorial sources of mental illness, this state of affairs is unlikely to change.[4]

Third, in the oncology example mentioned here, a biopsy confirms the malignant tumor as the existential referent of the hitherto latent common cause of signs and symptoms.[2] The cause is conceptually and empirically distinct from its effects. Indeed, a person may yet to experience any symptoms despite having the disease, as "silent" tumors illustrate. In contrast, it makes little sense to say that an entirely asymptomatic person has depression when there is no obvious discoverable referent for the conjectured entity distinct from the symptoms it supposedly causes.

Fourth, many latent variables are measured between patients (eg, neuroticism and externalizing), and it is unclear whether they capture processes operative within patients.[5] Dimensional latent variables in psychopathology are likely akin to psychometric *g*, another construct that does not signify a causally efficacious mechanism operative within an individual person.[6]

The Network Alternative

Borsboom and colleagues[2,7,8] have developed a network model of psychopathology that differs radically from the traditional latent categorical and dimensional models.

According to this perspective, symptoms are constitutive of mental disorders, not reflective of them. Syndromic coherence arises as a consequence of dynamic causal interactions among symptoms themselves, not as result of an underlying disease entity, whether construed categorically or dimensionally. (Note that although the term *symptom* implies a disease model, I use it throughout this editorial for expositional ease.)

Visual representations of networks comprise 2 elements: nodes and edges. Each node represents a symptom, and each edge represents an association connecting 2 symptoms. A triggering event external (eg, bereavement) or internal (eg, inflammation) to the person may activate 1 or more symptoms (eg, insomnia and worry) that in turn activate other ones (eg, irritability and anhedonia). Whether one symptom will activate another depends on the presence and strength of the edge connecting them. Probability of activation is represented by edge thickness.

An episode of disorder occurs when activation spreads throughout the network, turning on many symptoms that persist. Networks consisting of densely connected symptoms may be especially prone to exhibit hysteresis,[9] a phenomenon characterized by a self-reinforcing pattern activation whereby symptoms persist despite the disappearance of the original stressor, as exemplified by complicated grief.[10] Successful treatment does not entail the psychiatric equivalent of chemotherapy whereby intervention cures the underlying disease. Rather, treatment is directed at symptoms, especially those scoring high on out-strength centrality, a metric indicating the number and magnitude of edges issuing from a node and hence vital to maintaining activation in the network. Recovery involves deactivating nodes, weakening edges, and altering circumstances to reduce stressors that may prolong activation.[9]

The Network Approach to Posttraumatic Stress Disorder

The ontology of posttraumatic stress disorder (PTSD) has long been a topic of lively debate. One debate concerns whether the disorder is a trauma-induced psychobiological natural kind occurring throughout history and across cultures, or whether it is a socially constructed idiom of distress. Another concerns whether symptoms reflect an underlying categorical entity or

a dimension of stress responsiveness. The network model constitutes a third option for construing the ontology of PTSD.[11]

In their innovative and important study, Bryant and colleagues[12] estimated the network structure of PTSD symptoms among 1084 individuals who had sustained a traumatic injury sufficient to require hospitalization. Assessors used structured interviews to evaluate PTSD symptoms within about 2 weeks after injury and then again 12 months later (n = 838).

Bryant et al[12] estimated graphical Lasso based on extended Bayesian information criterion (referred to as GLASSO) and relative importance networks for both the acute and chronic data sets. The GLASSO network depicts (regularized) partial correlations between pairs of symptoms, controlling for the influence of all other symptoms in the network. Hence, although one cannot tell whether symptom X causes symptom Y (or vice versa) or whether influence goes both ways, we can be sure the association between symptoms X and Y is nonspurious. The GLASSO algorithm computes a sparse network whereby only partial correlations exceeding a certain threshold appear, and smaller, possibly false-positive, ones are driven to zero and vanish from the graph. The GLASSO network itself is undirected as no arrow tips appear on edges, signifying the direction of potentially causal influence. In contrast, the relative importance network does compute the direction of prediction. For example, an edge issuing from symptom X to symptom Y that is thicker than one going from symptom Y to symptom X means that activation of symptom X is a stronger predictor of activation of symptom Y than vice versa. Such bidirectional edges suggest a self-reinforcing loop between the symptoms.

Consistent with a previous network study on PTSD,[13] Bryant et al[12] confirmed established findings while discovering new ones disclosable only via the computational methods of network analysis. For the acute data set, the GLASSO revealed strong associations among avoidance of thoughts about the trauma and reexperiencing symptoms such as flashbacks and intrusions. The relative importance network confirmed bidirectionality of prediction between avoidance of thoughts about the trauma and intrusions, suggesting that the more one tries to suppress thoughts about the trauma, the more intrusive they tend to be. Bidirectionality also emerged between numbing and social detachment. Finally, intrusions and physiological reactivity to reminders of the trauma scored high on centrality metrics, indicating that activation of these 2 symptoms are especially likely to activate other symptoms in the network. Conversely, successful early intervention targeting these symptoms would likely prevent the full syndrome of PTSD from emerging.

At the 12-month assessment, overall network density was greater than during the acute phase, indicating more numerous and larger associations among symptoms. Moreover, 2 especially interconnected clusters became apparent. One fear circuitry cluster consisted of reexperiencing symptoms, hypervigilance, and startle. Another dysphoric cluster comprised concentration impairment, irritability, and sleep disturbance coalesced, and this cluster was connected to numbing, loss of interest in activities, social detachment, and foreshortened future.

Finally, concentration impairment is a nonspecific symptom of PTSD, unlike hallmark symptoms such as traumatic nightmares and flashbacks. Yet the centrality metrics confirmed its importance to chronic PTSD, exemplifying how network analysis can identify nonobvious important targets for intervention.

In conclusion, Bryant and colleagues[12] have made a valuable contribution to the rapidly growing field of network analysis of psychopathology.[14] Their study will surely inspire further work on PTSD and other syndromes.

ARTICLE INFORMATION

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