pathic personality disorders, respectively. The obsessive-compulsive personality disorder is defined largely by maladaptive conscientiousness (e.g., perfectionism, compulsivity, workaholism, and ruminative deliberation), but most measures of FFM conscientiousness do not assess for these maladaptive variants. Measures to assess maladaptive FFM traits, though, have been developed, including the Five Factor Model Personality Disorder scales[5], the Personality Inventory for DSM-5[6], and the Personality Inventory for ICD-11[7].

There are a number of advantages in conceptualizing the ICD and DSM personality disorders from the perspective of the FFM. Many of the ICD and DSM personality disorder syndromes have limited research interest and inadequate empirical support. The FFM brings to the personality disorders a substantial body of construct validation, including a resolution of such notable controversies as gender bias, excessive diagnostic overlap, and temporal instability. An understanding of the etiology, pathology and treatment of the personality disorders has been hindered substantially by the heterogeneity within and the overlap across the diagnostic categories. The American Psychiatric Association has been publishing treatment guidelines for every disorder within the DSM, but guidelines have been provided for only one of the ten personality disorders (i.e., borderline). The complex heterogeneity of the categorical syndromes complicates considerably the ability to develop an explicit, uniform treatment protocol. The domains of the FFM are considerably more homogeneous and distinct, lending themselves well for more distinct models of etiology, pathology and treatment[8]. Empirically validated treatment protocols have already been developed for FFM neuroticism[9].

A common concern regarding the FFM and any other dimensional trait model is that clinicians will be unfamiliar with this approach and will find it difficult to apply. However, the FFM organization is consistent with the manner in which persons naturally think of personality trait description. Persons who apply the FFM typically find it quite easy to use. There have in fact been a number of studies concerning the clinical utility of the FFM in comparison to the DSM syndromes. A few of these studies have favored the DSM syndromes but, when the methodological limitations of these particular studies were addressed in subsequent studies, the results consistently favored the FFM[8]. Experienced clinicians prefer the FFM and dimensional trait models for the conceptualization of personality disorders[8].

In sum, the FFM is the predominant model of general personality structure and offers the opportunity for a truly integrative understanding of personality structure across the fields of clinical psychiatry and basic personality science. The ICD and DSM models for the classification and diagnosis of personality disorder are shifting toward the FFM because of its empirical validation and clinical utility.

Thomas A. Widiger, Cristina Crego
Department of Psychology, University of Kentucky, Lexington, KY, USA

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The network approach to psychopathology: promise versus reality

The network approach to psychopathology has recently generated enthusiasm in the research community. This is likely due in large part to network methods being promoted with the promise of improving clinical prevention and intervention strategies by explicating the dynamic causal architecture of mental illness[1]. As a result, studies using network methods have proliferated with the aim of understanding causal interactions between psychiatric symptoms through empirical data.

As one example, there has been a substantial number of studies on the network structure of post-traumatic stress disorder (PTSD) wherein each network typically includes estimation of centrality indices for 16-20 symptoms, as well as the presence and weight of 120-190 edges. Few guidelines inform how to parse the multitudes of exploratory results in each symptom network. Confirmation bias is consequently hard to avoid, and the validity of a network is easily rationalized by the identification of intuitive findings[2]. By contrast, a variety of *post-hoc* explanations are available to dismiss unintuitive findings.

Estimated edges may represent a direct association between two symptoms (e.g., A→B or A←B), a reciprocal effect (A←→B), the common effect of an unmodelled variable (A←X→B), shared item content or method variance, or simply error (noise) in the data. Absent edges may represent conditional independence of two symptoms, or be the result of the specificity in the regularization method used. Central symptoms may cause other symptoms in the network and represent important targets for clinical intervention, or may be the consequence of those other symptoms and thus not useful targets for clinical intervention. Alternatively, as for estimated edges, high symptom centrality may summarize reciprocal relationships among symptoms, relationships with unmodelled variables, shared item content, method variance, or error. There are no methods for disentangling these different explanations of the focal parameters in cross-sectional symptom networks, which severely limits their utility. In other words, the results are equivocal.

The fundamental reason for this undermining ambiguity

is that, with few exceptions, the data used to investigate the network approach to psychopathology are ill-suited to do so. Network theory is alluring because it describes dynamic causal processes that play out within individuals. However, no statistical procedure can extract this information from the type of cross-sectional between-subject data that dominate the literature[3]. Indeed, networks estimated on these data are not expected to accurately reflect individuals' experiences or underlying causal processes, by network theorists' own arguments[4]. As such, the current state-of-the-art networks lack the capacity to provide the very insights they have been promoted to offer.

The unreliability of edges further complicates the interpretation of symptom networks, which change based on the specific set of symptoms in the network, the measures used to assess the symptoms, the use of a clinical or community sample, the sample size, and the type of network analysis adopted[5]. Remarkably, even when these characteristics are all held equal, key details of the model often do not replicate within or between samples[5,6]. This unreliability is predictable, given the intercorrelated nature of psychopathology symptoms, the limited reliability of single self- or clinician-report items, and a focus on the fully partialled relationship between each pair of symptoms (e.g., edge A–B represents what symptoms A and B share with each other, but not with any other symptoms in the network). Together, these common features of current network methods result in edges that are prone to substantial measurement error, leading to spurious associations and high sensitivity to minor variations in study methods and samples. It is therefore difficult to identify generalizable insights in the symptom network literature that advance our understanding of psychopathology.

In contrast, proponents of the network approach recently stated[7] that network structures replicate and generalize well, citing examples including "nearly identical" major depression and generalized anxiety disorder symptom networks, and a comparison of four PTSD networks. A closer look at these examples reveals, however, that almost a quarter (23%) of the total estimated edges were unreplicated between the two depression and anxiety networks[5], and that well over half (64%) of the edges were inconsistently estimated – as present or absent, or positive or negative – among the four PTSD networks[6].

The broader PTSD symptom network literature enables comparisons between additional studies that further highlight substantial inconsistencies[8]. Among eight of the studies in this literature that have used "state-of-the-art" network methods[9] in samples of people who have experienced trauma, the large majority (88%) of symptoms have been reported to have particularly high centrality – many in only a single paper, and none in a majority of the papers. Further, among these studies, all but three (98%) of the 120 possible edges among the PTSD symptoms in DSM-IV and DSM-5 have been estimated, and vary between studies in their presence, strength, sign, and hypothesized importance in the network.

There are not yet any methods that can indicate *a priori* whether or not a specific edge is likely to replicate. While it may be naive to expect exact replication, observed levels of inconsistency between networks seem particularly problematic in the context of a theory that emphasizes interpretation of the presence, absence, strength and sign of each individual edge and the corresponding centrality of individual symptoms. Importantly, optimistic perspectives on the reliability and replicability of symptom networks are often based on methods (e.g., bootnet, the omnibus NetworkComparisonTest, and correlations between lists of edges) that shift the focus away from these detailed features, and towards global network patterns that do not correspond with the basis of network theory or the insights that symptom networks have been promoted to provide[6]. The result is that these popular methods create an impression of reliability and replicability that fails to translate to the level at which networks are interpreted.

Our concerns surrounding the equivocal, stationary and ungeneralizable nature of current symptom network results contrast with the rhetoric in much of the network literature promising meaningful clinical insights from these methods. Alternative modeling methods and research designs – for example, collecting experimental data with reliable measurement of symptoms over time – are needed to make causal inferences about relationships between symptoms, and thus to achieve the aims of the network approach to psychopathology. Ultimately, it remains unclear what can be meaningfully concluded from the extant network literature with respect to the onset, maintenance or treatment of psychopathology.

Miriam K. Forbes[1], Aidan G.C. Wright[2], Kristian E. Markon[3], Robert F. Krueger[4]

[1]Centre for Emotional Health, Department of Psychology, Macquarie University, Sydney, NSW, Australia; [2]Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA; [3]Department of Psychological and Brain Sciences, University of Iowa, Iowa City, IA, USA; [4]Department of Psychology, University of Minnesota, Minneapolis, MN, USA

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