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Network Analysis of Psychopathology: Controversies and Challenges

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Abstract

Empirical publications inspired by the network approach to psychopathology have increased exponentially in the twenty-first century. The central idea that an episode of mental disorder arises from causal interactions among its symptomatic elements has especially resonated with those clinical scientists whose disenchantment with traditional categorical and dimensional approaches to mental illness has become all too apparent. As the field has matured, conceptual and statistical concerns about the limitations of network approaches to psychopathology have emerged, inspiring the development of novel methods to address these concerns. Rather than reviewing the vast empirical literature, I focus instead on the issues and controversies regarding this approach and sketch directions where the field might go next.

Contents

1. INTRODUCTION

In the first half of the twentieth century, clinicians under the spell of psychoanalysis interpreted symptoms as clues to the unconscious conflicts of their patients. Uncovering and resolving these conflicts was the central aim of psychotherapy [e.g., Freud 1973 (1924)]. Midway through the century, two groups challenged this view. Despite often disagreeing about etiology and treatment, they shared an opposition to psychoanalysis and a commitment to a scientific approach to psychopathology.

The first group comprised the pioneers of behavior therapy who viewed symptoms as maladaptive learned responses, not as symbols of problems requiring psychoanalytic excavation (e.g., Wolpe & Rachman 1960). Accordingly, they devised methods inspired by the science of learning and conditioning, such as systematic desensitization, to treat symptoms directly (Wolpe 1958). Their psychoanalytic critics warned that merely eliminating a symptom without resolving the underlying conflict would result in symptom substitution; a new fear, they predicted, would pop up to replace the desensitized one (Tryon 2008). When this therapeutic version of Whac-A-Mole failed to materialize, behavior therapy gained traction, largely because its explicit methods were suitable for rigorous empirical evaluation (e.g., Lang & Lazovik 1963).

The second group to challenge psychoanalysis comprised research psychiatrists aiming to rebut those who denied that their specialty was a legitimate branch of medicine (e.g., Guze 1992). Allergic to Freudian speculation, these Neo-Kraepelinians described the signs and symptoms of psychopathology as objectively as possible to bolster the reliability of psychiatric diagnosis. Led by Robert L. Spitzer, they transformed the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) into a largely atheoretical lingua franca shorn of its Freudian trappings. Their goal was to enable clinicians of diverse theoretical persuasions to agree about the presence of a disorder even if they disagreed about its cause or how to treat it. They hoped that a medically respectable

classification of discrete disorders, characterized by signs, symptoms, and course, would establish a foundation for research on the etiology, prevention, and treatment of mental illness.

Although improvements in reliability were not quite as impressive as hoped (e.g., Kirk & Kutchins 1992), the Neo-Kraepelinian revolution embodied in the DSM-III (APA 1980) was successful in many ways. Psychiatric epidemiology flourished as explicit criteria for diagnosing mental disorders permitted precise estimates of their prevalence. The DSM-III furnished a framework for basic research on the biological, behavioral, and cognitive aspects of psychopathology. Randomized controlled trials (RCTs) identified the most efficacious pharmacologic, behavioral, and cognitive behavioral therapies (CBTs) for specific disorders, thereby providing guidelines for evidence-based clinical practice.

Yet dissatisfaction with the DSM categorical system grew over the years (McNally 2011). The failure to identify biomarkers of categorical entities prompted one former head of the National Institute of Mental Health (NIMH) to say that DSM disorders were little more than reified labels (Hyman 2010). Criticizing the DSM-5 (APA 2013) revision process, the head of the previous DSM called it a "fiasco" (Frances 2014, p. 372), warning that the expanding girth of the manual signified the increasing medicalization of ordinary emotional distress (Frances & Nardo 2013). Dissatisfaction culminated in the announcement by the director of the NIMH that the DSM would no longer be the requisite framework for grant proposals submitted to the agency (Insel 2013). The NIMH, he said, would favor proposals that targeted transdiagnostic mechanisms as specified in the Research Domain Criteria (RDoC) initiative (Insel et al. 2010). Rampant comorbidity among supposedly discrete conditions motivated the development of dimensional systems to characterize the variegated tapestry of mental illness (Kotov et al. 2017, Lahey et al. 2017). Finally, striking an elegiac note, even leaders of the DSM-5 revision process concluded, "We are now coming to the end of the neo-Kraepelinian era" (Regier et al. 2013, p. 68).

2. THE NETWORK APPROACH TO PSYCHOPATHOLOGY

Despite their differences, clinicians favoring categorical versus dimensional approaches to psychopathology agree that signs and symptoms do not co-occur randomly; some are more likely to covary than others. However, they disagree about why this happens. The Neo-Kraepelinian categorical view holds that signs and symptoms cluster because they share a common cause—namely, the underlying, unobserved disease entity. The contrasting view holds that latent dimensions are the source of syndromic clustering.

The network perspective differs strikingly from both traditional accounts. According to Denny Borsboom and his psychometric colleagues, an episode of disorder is an emergent phenomenon arising from the causal interactions among its symptomatic elements (Borsboom 2017, Borsboom & Cramer 2013, Borsboom et al. 2019, Cramer et al. 2010a). That is, the relation of symptoms to disorder is mereological (i.e., parts to whole), not reflective (Borsboom 2008). A disorder is not a latent entity causing the emergence and covariance of the symptoms that supposedly reflect its underlying presence. Rather, symptoms are constitutive of disorder.

Borsboom's (2008) early critique of diagnostic practice foreshadowed the network approach. He noted that to justify an inference to a latent, common cause of syndromic coherence, one must satisfy the axiom of local independence. That is, the covariance among symptoms should vanish upon conditionalizing on the presence of the disorder.

Consider an everyday example of local independence. After placing six thermometers in a room, we will soon notice that their mercury readings all register 72°, reflecting the common cause of ambient temperature. If we place an ice cube on one thermometer, its mercury reading will plummet, but those of the other thermometers will remain unchanged.

Consider a medical example. A patient has a fever of 102°, fatigue, painful swallowing, and white patches in his throat, suggestive of the *Streptococcus* bacterium as the cluster's common cause. Strep remains a conjectured latent variable until a throat culture confirms its presence as the cause of the ontologically distinct symptomatic effects it produces. Hence, there is an important difference between a reflective latent variable whose existential referent has yet to be observationally confirmed and one that merely provides a useful summary of data (Borsboom et al. 2003). This difference echoes MacCorquodale & Meehl's (1948) distinction between hypothetical constructs and intervening variables.

However, as Borsboom (2008) observed, with few exceptions (e.g., trisomy 21 in Down syndrome), most psychiatric disorders violate the axiom of local independence. Indeed, DSM criteria often specify direct functional relations between signs and symptoms (e.g., phobic fear motivating behavioral avoidance). Bulimia nervosa illustrates direct functional relations among signs and symptoms. For typical patients, anxiety about body shape and size motivates restricted food intake. Growing hunger prompts an eating binge, and surging shame about becoming fat triggers self-induced vomiting. It makes little sense to conceptualize these phenomena as functionally independent effects of an underlying common cause called bulimia nervosa.

The view of disorders as systems of functionally interrelated elements has gained network psychometricians many scientific allies among CBT clinicians who have long championed functional analyses of behavior (e.g., Hofmann et al. 2016, McNally 2016, Wolpe 1973). Indeed, recent conferences of the Association for Behavioral and Cognitive Therapies have featured many symposia on network studies of psychopathology.

Network studies on psychopathology have grown exponentially (Contreras et al. 2019, Robinaugh et al. 2020), especially since Borsboom & Cramer's (2013) seminal article appeared in the *Annual Review of Clinical Psychology*. Robinaugh et al. (2020) identified 363 articles, including 204 empirical ones. The range of coverage has been remarkable. Network studies have appeared on major depression (e.g., Cramer et al. 2012), autism (Ruzzano et al. 2015), complicated grief (CG) (e.g., Robinaugh et al. 2014), posttraumatic stress disorder (PTSD) (e.g., McNally et al. 2015), social anxiety disorder (e.g., Heeren & McNally 2016), substance abuse and dependence (e.g., Rhemtulla et al. 2016), obsessive–compulsive disorder (e.g., McNally et al. 2017b), psychosis (e.g., Isvoranu et al. 2017), childhood disorders (e.g., Boschloo et al. 2016a), eating disorders (e.g., Levinson et al. 2017), bipolar disorder (e.g., Koenders et al. 2015), psychopathy (Verschuere et al. 2018), body dysmorphic disorder (Summers et al. 2020), and sex offenders (van den Berg et al. 2020).

The vastness of the field now defies easy summary. Accordingly, my aims are modest. After providing a brief overview of key concepts, I focus on methodological and conceptual challenges and on controversies facing the network approach to psychopathology.

3. BASIC CONCEPTS IN NETWORK ANALYSIS

3.1. Nodes and Edges

Networks consist of nodes and edges. Nodes signify the objects of study—individuals in a social network, neurons in a neural network, and (usually) symptoms in a psychopathology network—and edges signify connections between pairs of nodes. Unlike social networks, where one can observe the nodes (persons) and the relations between them (e.g., friendships), networks of symptoms require statistical estimation.

Edges can be unweighted or weighted. An unweighted edge merely means that two nodes are connected, whereas the thickness of a weighted edge indicates the magnitude of association

between them (e.g., the size of a correlation coefficient) and hence the probability of their co-occurrence. The stronger the association (i.e., the edge weight), the thicker the edge will be. Typically, edges are colored green to indicate a positive association, whereas red is used to indicate a negative association.

Finally, networks can comprise undirected or directed edges. Undirected networks consist of edges that indicate an association between two nodes but are agnostic about whether the occurrence of node *X* predicts the occurrence of node *Y*, or vice versa, or whether the direction of prediction goes both ways. In contrast, directed networks feature edges with arrow tips at one end of each edge, pointing in the direction of prediction and perhaps causation.

Neo-Kraepelinians emphasize the importance of hallmark symptoms in differential diagnosis. Ideally, hallmark symptoms should bear a two-way pathognomonic relation to the disease whose presence they indicate. Their occurrence would confirm its presence, whereas their absence would rule it out. Unfortunately, psychiatric syndromes seldom have indicators that satisfy this austere standard. Rather, our hallmark symptoms are merely those more commonly appearing in some syndromes than in others.

The emphasis on hallmark symptoms has usually been accompanied by a de-emphasis on nonspecific symptoms (e.g., insomnia) occurring in many syndromes. Accordingly, to reduce diagnostic comorbidity, some experts suggest that diagnostic criteria sets should retain hallmark symptoms but eliminate nonspecific ones (Spitzer et al. 2007).

In contrast, drawing on the field of social network analysis (e.g., Borgatti 2005, Freeman 1978–1979), network researchers have adopted measures for evaluating how important a node is within a network. "Importance" indicates how interconnected it is with other nodes in the network. Because there are different ways to be interconnected, there are different metrics of node centrality. For example, a node's degree centrality is the number of edges connecting it to other nodes in the (usually unweighted) network. Betweenness centrality is the number of times a node appears on the shortest path between two other nodes. A node's closeness centrality is the average distance between it and all other nodes in the network. Although all three metrics have appeared in network studies on psychopathology, degree centrality is most useful in unweighted networks, whereas the conceptual relevance of betweenness and closeness centrality metrics seems less relevant to networks whose nodes are symptoms than to networks whose nodes are, for example, individuals in friendship or infectious disease networks (Bringmann et al. 2019). Hence, the most useful metrics for psychopathology are strength centrality and expected influence centrality.

Edge thickness signifies the magnitude of the association (e.g., Pearson correlation coefficient) between two nodes, and the strength centrality of a node is the sum of the absolute values of the edge weights connecting it to other nodes. In cross-sectional networks, the edge weight signifies the probability of coactivation between two nodes.

Accordingly, assessing a node's activating influence on other nodes by summing the absolute values of weights of the edges incident on the node works fine—as long as there are no negative edges connected to it. However, strength centrality will distort estimates of a node's activating influence to the extent that it has negative edges with other nodes in the network (Everett & Borgatti 2014).

Expected influence centrality addresses this problem by retaining the sign of edge weights prior to summing them (Robinaugh et al. 2016). Robinaugh and colleagues' (2016) simulations showed that expected influence performs identically to strength centrality when the network contains only positive edges but outperforms it as networks contain increasingly more negative edges. That is, expected influence was a better predictor of declines in the severity of symptoms of CG over time.

3.2. Types of Cross-Sectional Networks

Cross-sectional networks depict relations between pairs of nodes involving many subjects assessed at a single point in time (Borsboom & Cramer 2013). Most concern symptoms assessed via questionnaire or structured clinical interview. Epskamp and colleagues' (2012) R package, qgraph, has been indispensable for computing networks in psychopathology research.

3.3. Association Networks

Association networks comprise undirected edges between pairs of nodes that signify the probability of their co-occurrence (e.g., Verschuere et al. 2018). The aim of network analysis is to discern causal relations among symptoms. Although correlation does not confirm causation, it is one step in the direction of establishing it.

However, association networks have two limitations. First, because they are cross-sectional and undirected, one cannot tell whether symptom *X* predicts the activation of symptom *Y*, or vice versa, or both. Moreover, one cannot be certain whether the relation between these symptoms is spurious as a result of the influence of other nodes in the network.

3.4. Concentration (Partial Correlation) Networks

To ascertain whether the association between nodes is direct or spurious, one can compute a concentration network whereby edges represent partial correlations between node pairs after one adjusts for the influence of all other nodes in the network. One can see whether a connection evident in an association network survives adjustment and remains in a concentration network. For example, in a study of PTSD symptoms among earthquake survivors, a strong association between hypervigilance and exaggerated startle that was evident in the association network remained robust in the concentration network (McNally et al. 2015).

3.5. Relative Importance Networks

In a relative importance network, each edge signifies the relative importance of a node as a predictor of another node (Johnson & LeBreton 2004). The edges are directed as well as weighted, and arrow tips signify the direction of prediction. A node's relative importance indicates both its direct effect on another node and its effect after one has adjusted for all other nodes in the network. Direction of prediction, however, does not confirm causality. The lmg metric quantifies this relation on a scale of 0 to 1.00 (Grömping 2006, Robinaugh et al. 2014).

3.6. Regularized Partial Correlation Networks

As the number of nodes and edges increases, we wind up estimating a very large number of parameters and boosting the chances of false alarms. Accordingly, network researchers in psychopathology have favored regularized partial correlation networks (Epskamp & Fried 2018). This procedure entails estimating a network via a graphical Gaussian model whereby edges depict conditional independence relations among nodes (i.e., partial correlations between node pairs, adjusting for the influence of the remaining nodes in the network). In contrast to a concentration network, one regularizes the model by running the graphical LASSO (least absolute shrinkage and selection operator) (Friedman et al. 2008) algorithm—which implements an L1 penalty—and estimating a sparse inverse covariance matrix that shrinks small partial correlations to zero such that these edges never appear in the final graph. Regularization returns a sparse network that parsimoniously

accounts for the covariance among nodes by including the smallest possible number of edges. The edges that survive regularization are the largest, and presumably genuine, ones.

Epskamp and colleagues' (2012) qgraph package provides an extended Bayesian information criterion (EBIC) model comparison procedure, which identifies the tuning parameter that optimizes model fit most parsimoniously (Chen & Chen 2008) given the value of the hyperparameter gamma (γ). The procedure estimates 100 models that vary in their sparsity from 0 to 1.00. It identifies the model with the lowest EBIC value as the one most likely to maximize the number of genuine edges while minimizing the likely nongenuine edges.

However, Williams and his collaborators have shown that regularizing partial correlation networks via the graphical LASSO returns sparse graphs but does so at the expense of omitting genuine edges (Williams & Rast 2020, Williams et al. 2019). Hence, they have recommended against regularizing when computing partial correlation networks by, for example, setting the hyperparameter to zero. They observed that the graphical LASSO was designed for high-dimensional data sets in which the number of nodes vastly exceeds the number of cases (e.g., genes versus subjects in genomics). By contrast, psychopathology networks have low dimensionality (i.e., more subjects than symptoms).

3.7. Bayesian Networks (Directed Acyclic Graphs)

Bayesian networks represent an ambitious method to characterize cross-sectional data as a causal system (Pearl & Mackenzie 2018). However, two strict assumptions must be met to estimate a causal network. First, all relevant causal variables must appear in the data set.

Second, all edges connecting nodes in a directed acyclic graph (DAG) are directed. Hence, the arrowhead at the tip of an edge issuing from one node and incident on another signifies the direction of probabilistic dependency. For example, an edge issuing from node X and incident on node Y (i.e., $X \rightarrow Y$) means that the presence of node Y more strongly implies the presence of node X rather than vice versa. The "parent" node X could be present without its "offspring" node Y, but the presence of the child implies the presence of a parent. Consider a detective who discovers gunpowder residue on the hand of a murder suspect. There is a very high likelihood that the suspect fired a pistol within the previous 4–6 hours. However, because a careful gunman would wear discardable gloves before firing the gun, the homicide does not as strongly imply the presence of gunpowder on the hand of the killer.

However, in cross-sectional DAGs, one must not confuse direction of dependence with temporal antecedence. DAGs depict conditional independence relations—the joint probability distribution of the variables (Moffa et al. 2017). Accordingly, a DAG is decomposable into the conditional distribution of each node given its parent nodes, thereby revealing whether the downstream presence of node Y probabilistically implies the upstream presence of node X more than vice versa. Probabilistic dependence cannot confirm temporal precedence in cross-sectional data.

Clinical scientists have computed two types of DAGs (e.g., Jones et al. 2018, McNally et al. 2017b). In one type, edge thickness signifies the importance of the edge to model fit. The thicker the edge, the more vital it is to the model's fit to the data.

In the other type, edge thickness signifies the level of confidence (probability) that the direction of prediction is as depicted in the graph in at least 51% of the bootstrapped samples. For example, sadness predicts suicidal ideation and attempts rather than vice versa (McNally et al. 2017b). A relatively thin edge in a direction-of-prediction DAG means that the arrowhead was often pointing in the opposite direction in many of the bootstrapped samples. Thin edges suggest bidirectionality.

However, a bidirectional relation between nodes (i.e., $X \rightarrow Y$, and $Y \rightarrow X$) is only one type of cycle (loop) prohibited by a DAG. That is, the aspirational causal modeling rests on the

assumption that there are no cycles in the network, including ones such as $X \rightarrow Y \rightarrow Z \rightarrow X$. One concern with this prohibition is that clinical observation strongly suggests such loops in psychopathology. For example, Clark's (1986) model of panic rests on such a feedback loop (e.g., bodily sensations → catastrophic misinterpretation → increased fear → increased bodily sensations). However, as García-Velázquez et al. (2020, p. 247) observed, "Cyclic relations are inherently difficult to analyze, and thus it seems wise to start with robust findings on directional dependence and only then build toward more complex models."

4. CHALLENGES AND CONTROVERSIES

4.1. Comorbidity

Cramer et al. (2010a) showed how the network perspective can solve the comorbidity problem that has long vexed the Neo-Kraepelinians. Syndromes that are frequently comorbid often share nonspecific symptoms, such as insomnia and concentration impairment in generalized anxiety disorder (GAD) and major depressive disorder (MDD). Accordingly, activation of such bridge symptoms may spread to symptoms of both syndromic clusters, producing diagnostic comorbidity. Hence, comorbidity is a natural consequence of partially overlapping syndromic clusters.

Payton J. Jones broadened the concept of bridge symptoms to cover comorbidity arising in the absence of shared symptoms. To identify bridges, he devised metrics such as bridge strength centrality and bridge expected influence centrality [see the bridge function in the R package networktools (Jones 2017)]. A symptom of one syndrome having strong connections with symptoms of another syndrome (and vice versa) will score high on these metrics. Jones et al. (2019) validated them in four studies. The first showed that the bridge function accurately identified known bridge nodes in two artificially generated networks (sensitivity = 92.7%, and specificity = 84.9%), and the second simulation showed that the function robustly identified bridges, especially for samples exceeding 300 "subjects."

The third study revealed that experimentally deactivating ("treating") nodes high (versus low) on bridge centrality was more effective in preventing contagion ("comorbidity") between clusters, consistent with network theory (Borsboom 2017). The fourth study concerned 18 published empirical networks whose authors had endeavored to identify bridges between syndromic clusters. The algorithm matched the accuracy of the authors' bridge identification for simple networks and exceeded their performance for complex ones.

These methods for identifying bridges have been appearing in empirical studies (e.g., Heeren et al. 2018), suggesting solutions to persistent puzzles in psychopathology (e.g., Bellet et al. 2018), such as the relation between CG and posttraumatic growth (PTG)—subjectively appraised beneficial changes noted by people who have undergone trauma (Tedeschi & Calhoun 1996). Is the relation positive, negative, quadratic, or none of the above?

Bellet et al. (2018) addressed this issue by computing bridge expected influence between clusters of nodes representing elements of PTG and those representing CG. For example, one CG symptom—the degree to which the death of the loved one changed the mourner's view of the world—was strongly and positively related to the PTG cluster, especially to the PTG element of having different priorities in life. The CG symptom of an inability to care about others had a strong, negative bridge expected influence value vis-à-vis the PTG cluster, whereas the PTG element of enhanced ability to cope with difficulties had a negative bridge expected influence on the CG cluster. These findings indicate how construing phenomena such as CG and PTG as networks can illuminate interconnections that are otherwise obscured by treating them as sum scores (Fried et al. 2014).

4.2. Replicability

Computing networks from two epidemiologic data sets, Forbes et al. (2017, p. 969) concluded that "popular network analysis methods produce unreliable results." In their reply, Borsboom et al. (2017, p. 997) observed that Forbes et al. had confounded "evidence for replication problems that concern a *particular estimated model* with evidence for problems of *the model in general.* This is a non sequitur" (italics in original). As Borsboom et al. (2017, p. 997) observed, such an inference is akin to concluding "that 'regression analysis has limited replicability'" after one discovers that regression coefficients differ in two samples after one has fitted a certain regression model to each sample. That is, unless one knows that the "true" relationship between variables is identical across the samples, one cannot determine whether discrepant results arose from differences between the samples or from an inherently faulty method. After detecting errors in Forbes and colleagues' analyses, Borsboom et al. (2017) reanalyzed the data and found that all four network models replicated well.

In fact, network researchers had already been addressing the challenge of reproducibility (Fried & Cramer 2017, Fried et al. 2017). For example, Epskamp and colleagues' (2018a) tutorial describes procedures in the R package bootnet that enable researchers to gauge the accuracy of edge weight estimates and the stability of centrality metrics. The former is accomplished by calculating bootstrapped confidence intervals (CIs). For each edge weight, the true value of the corresponding parameter will fall within the CI in 95% of cases. Bootstrapping entails repeatedly estimating a model with sample (or simulated) data to obtain a sampling distribution for the edge weight of interest (e.g., one connecting exaggerated startle and hypervigilance in a PTSD network). Nonparametric bootstrapping involves generating plausible data sets by resampling the data with replacement. This method is appropriate for most psychopathology studies that typically involve ordinal measures (e.g., a five-point rating scale that represents symptom severity), and it requires no theoretical assumptions and is appropriate for regularized correlational data.

The bootstrapped difference test evaluates whether one edge weight differs significantly from another (e.g., "is the association between startle and hypervigilance greater than the one between flashbacks and nightmares?"). One first subtracts the difference between one edge weight and another and then computes a bootstrapped CI around the difference scores.

To estimate the stability of centrality measures, Epskamp et al. (2018a) recommended the case-dropping subset bootstrap. This entails randomly dropping an increasingly larger percentage of subjects from the data set and then recomputing the centrality measures. The question is whether the order of magnitude of the centrality indices remains stable. To quantify the stability of a network's centrality values, Epskamp et al. (2018a) proposed the correlation stability (CS) coefficient. CS signifies the maximum proportion of cases one can drop, such that with 95% probability the correlation between the original centrality values and those obtained from the networks comprising progressively smaller subsets of the data is at least $r = 0.7$. Simulations have shown that the CS coefficient should not be lower than 0.25, and ideally it should exceed 0.70 to enable confident interpretation of the order of centrality values. Strength and expected influence centralities meet this standard more often than do betweenness and closeness centralities in psychopathology networks.

Forbes et al. (2019) reanalyzed several network studies concerning depression, anxiety, and PTSD. Using conventional network indices, they found that characteristics of these networks were, indeed, robust and stable. However, when they reanalyzed the data with their novel "direct metrics of replication," they concluded that networks have "limited reliability" (Forbes et al. 2019, p. 1).

In their rejoinder, Jones et al. (2020) emphasized three points. First, one cannot conclude that a method (e.g., multiple regression, network analysis) is faulty merely because results differ between two samples of patients. Empirical data alone can neither confirm nor impugn the validity of a method; sampling variability and imprecise measurement of symptoms may also produce different results across samples.

Second, contrary to the claim of Forbes et al. (2019), statistical removal of shared variance between variables, via partial correlational analysis, does not invariably attenuate reliability. Indeed, statistical adjustment bolsters reliability, reducing it only when used inappropriately with regard to the causal model generating the data.

Third, their direct metrics rest on the dubious assumption that any variation in a parameter estimate constitutes nonreplication. However, one cannot expect equivalence of parameter estimates; sampling routinely produces departures from invariance. Accordingly, one must model the extent of variation to assess the value of their direct metrics to ascertain whether differences in parameter estimates across samples are genuine. To accomplish this, Jones et al. (2020) devised a Bayesian analytic method that quantifies such uncertainty. Reanalyzing Forbes and colleagues' (2019) data, they found considerable evidence in support of the replicability of edges, and scant evidence favoring nonreplication. As Jones et al. (2020, p. 6) concluded, "although psychological network analysis faces many challenges, we find no evidence that limited replicability is among them."

4.3. Ontology

Many psychometricians have observed that latent variable and network models are statistically fungible (e.g., van Bork et al. 2019). However, mathematical equivalence is not the same as ontological equivalence (Borsboom 2017). Indeed, despite the predictive equivalence of the heliocentric and geocentric mathematical models of the solar system [Galilei 2012 (1615)], it makes a great deal of difference whether the earth orbits the sun or vice versa (McNally 2019). And the difference between statistical and substantive models is especially crucial in medicine. Using aspirin to suppress one's headache is not the appropriate treatment if the pain results from an undetected brain tumor (Borsboom & Cramer 2013).

In a thoughtful defense of latent variable modeling, Bringmann & Eronen (2018) observed that if two symptoms have a common cause, then it must explain at least some of their covariance but not necessarily all of it. That is, a common cause does not require local independence among the effects of the cause unless we presuppose that there is no direct connection between symptom pairs. If we establish that two symptoms are correlated and that there is no causal relation between them, we can assume that they have a common cause. Moreover, as Bringmann & Eronen (2018) observed, some paradigmatic instances of common causes cited by network theorists, such as lung cancer, turn out to be more complicated than originally conceptualized (Molassiotis et al. 2011). A malignant lung tumor can certainly produce chronic cough, bloody sputum, and chest pain (Borsboom & Cramer 2013), but chronic cough can itself worsen chest pain directly. Hence, the tumor does not entirely account for the covariance among symptoms. Others have also suggested hybrid models whereby, for example, a trauma serves as the common cause that simultaneously incites several PTSD symptoms, which subsequently cause others, until a self-maintaining network crystalizes (Fried & Cramer 2017, Fried et al. 2018).

However, the real issue is not whether there a common cause; what matters is its ontological status (Borsboom et al. 2003). As Borsboom (2008) observed, the cause must be distinguishable from its effects, and a traumatic event clearly qualifies. There is nothing "latent" about it. Likewise, a physician assessing a patient who complains of bloody sputum, chest pain, and a chronic cough

may conjecture that a malignant lung tumor is the cause of these symptoms. The tumor is a latent variable only insofar as it remains unobserved; it is not in principle unobservable. The tumor has an existential referent, discoverable by X-ray and biopsy, which operates causally within a person (McNally 2016). Its ontology differs from that of latent variables emerging from between-subjects factor analyses (Borsboom et al. 2003). For example, dimensional theorists have suggested that the p factor (Caspi et al. 2014)—a general psychopathology factor akin to psychometric g, the general factor of intelligence—"may have a physical reality" (Lahey et al. 2011, p. 187), perhaps genetic. However, as van Bork et al. (2017) have demonstrated, any data set comprising highly intercorrelated measures (i.e., "positive manifold") is mathematically certain to yield a general factor, even if that factor has no existential referent apart from the data from which it emerges.

Finally, psychologists have expressed concerns about measurement error in network studies (e.g., Krueger et al. 2010), especially when a single five-point ordinal scale is the sole source of gauging symptom severity. However, as Cramer et al. (2010b) noted, one can often form a composite variable for sleep-onset insomnia, for example, by using self-report, actimeter, and electroencephalographic measures. As Cramer et al. emphasized, such a composite variable has a natural referent operative within the person: latency to fall asleep.

4.4. Centrality

There are multiple ways to define the importance of a symptom. For diagnosis, one might bestow importance on hallmark symptoms occurring in one syndrome, but rarely in others (e.g., flashbacks in PTSD). For treatment, we might regard important symptoms as the most severe, the most impairing, or the most dangerous (e.g., suicidal ideation) ones. Network researchers identify the most important symptoms as those that score highest on measures of strength centrality and expected influence centrality. That is, the most important ones are those most strongly connected to other symptoms in the cluster.

These centrality metrics have captured the attention of clinicians because they promise to identify important targets for therapeutic intervention (e.g., Hofmann et al. 2016, McNally 2016). In principle, if one can therapeutically deactivate a high-centrality symptom that maintains a person in a pathological state, then this process should initiate a beneficial cascade that resolves the episode of disorder (Borsboom 2017, Cramer et al. 2016), assuming that certain conditions are met.

Most studies report cross-sectional data, visualized as regularized partial correlation networks. However, these networks do not enable one to determine whether a high-centrality symptom is the source of activation for other symptoms, the recipient of activation from other symptoms, or both. Attempts to treat a symptom whose high centrality results from its status as the recipient of activation are unlikely to succeed. Such attempts are akin to expelling smoke from one's home with a fan rather than using a fire extinguisher to squelch the incipient conflagration.

Noting that centrality is a relative measure of symptom importance, Haslbeck & Fried (2017) developed a measure of node predictability. Consider two symptom networks whereby insomnia has the strongest strength centrality value. In the first network, 80% of the variance in insomnia is predicted by only two other symptoms—worry and concentration impairment—whereas in the second network, only 11% of the variance in insomnia is predicted by all other symptoms in the network. Hence, the predictability value for insomnia in the first network means that interventions to treat worry would likely reduce insomnia, whereas this would not be the case for the second network. Indeed, despite insomnia's high strength centrality relative to other symptoms in the network, one would best look outside the network to identify variables to reduce insomnia. Hence, an absolute measure of predictability may prove a better marker for identifying symptoms suitable

for intervention rather than only the relative metric of strength centrality. A highly predicted symptom may constitute low-hanging fruit suitable for intervention.

Network researchers have been developing solutions for problems that can bedevil the correct identification of high-centrality symptoms. The first is topological overlap (Fried & Cramer 2017), which occurs when a network includes nodes that are functionally indistinguishable from one another. For example, PTSD assessments often have different items tapping avoidance of thoughts and feelings about the trauma and avoidance of activities reminiscent of the trauma. Although these symptoms are not precisely synonymous, they may functionally be so. Uncorrected topological overlap will artificially inflate the centrality metrics of the relevant symptoms.

Payton J. Jones devised a formal method of identifying topological overlap between nodes, embodied in the goldbricker function included in his R package, networktools (Jones 2017). For example, Jones and his colleagues conducted a network study involving eating disorders that included the nodes overeating and binge eating (Levinson et al. 2018). If these two symptoms exhibit topological overlap, then not only should they be highly correlated but also their respective patterns of correlations with all other symptoms in the network should be strikingly similar. In this example, goldbricker returned 52 correlations corresponding to the correlation between overeating and the other symptoms (except binge eating), computed across all participants in the study, and returned another 52 correlations between binge eating and the remaining symptoms (except overeating). If these two symptoms are functionally indistinguishable, then there should be relatively few significant differences between the pairs of correlations (e.g., the correlation between overeating and the desire to be thin versus the correlation between binge eating and the desire to be thin). Investigators need to specify what proportion of the 52 comparisons must reach a target level of statistical significance (e.g., $p < 0.05$, $p < 0.01$) to count as unacceptable topological overlap, being mindful of sample size. If, say, 75% are significant, one can either create a composite node for the overlapping symptoms or omit one of them from the network analyses (see Jones 2017).

Severe topological overlap can result in a nonpositive definite matrix that plays havoc with the computation of networks. For example, initial network analyses of the Liebowitz Social Anxiety Scale (Liebowitz 1987), a self-report measure gauging the severity of fear and of avoidance of a range of activities and situations, revealed consistently high correlations between ratings of fear and avoidance across items for patients with social anxiety disorder, but much less so for healthy comparison subjects whose levels of shyness fell far short of the diagnostic threshold for social anxiety disorder (Heeren & McNally 2018a). Patients tended to avoid the things they feared, whereas comparison subjects reported much less avoidance. To eliminate the problem of a nonpositive definite matrix, we created a composite fear-avoidance variable for each item (e.g., going to a party) and reran the analyses, publishing a correction to our article (Heeren & McNally 2018b).

Unfortunately, we may have solved our statistical problem by replacing it with a clinical problem. As behavior therapists have long known (e.g., Lang 1968), verbal, behavioral, and physiological measures of fear (and anxiety) are often discordant, especially for people who are neither seriously phobic nor utterly fearless. That is, courageous individuals approach situations they deem threatening despite experiencing intense fear. By combining ratings of fear and avoidance, we obscured the phenomenon under study, especially in the healthy group.

The aforementioned study (Heeren & McNally 2018b) points to yet another challenge for network researchers. How shall we define our study group? What should our inclusion criteria be? Sometimes the criteria are event based, such as the death of one's spouse (Robinaugh et al. 2014) or exposure to an earthquake (McNally et al. 2015). Yet not uncommonly, clinical researchers compute networks on data from subjects who qualify for a certain diagnosis. Unfortunately, having inclusion criteria strongly related to nodes in the network can affect the results in unpredictable

ways, evincing "Berkson's bias" (a.k.a. "conditioning on a collider"; de Ron et al. 2021). Edges may shrink, appear, disappear, or reverse sign depending on inclusion criteria.

This bias is not confined to network analysis. Consider an example from the National Basketball Association (NBA). The NBA hosts a slam dunk contest every winter during the weekend of the All Star Game. If we were to condition our analysis on whether a participant plays in the NBA, we might find that height is a weak predictor of success at slam dunking (indeed, Nate Robinson has won the contest a league-leading three times, and he is five feet nine). On the other hand, if we opened the contest to the general population, height would be a very strong predictor of success in slam dunking.

There is no obvious solution to this problem (de Ron et al. 2021). However, it seems that the greatest threat to valid inference would entail generalizing findings from a subset to the general population, such as drawing conclusions about the network structure of depression symptoms in the general population from studies on patients who qualify for a diagnosis of MDD.

Another issue concerns the relation between centrality and node variance. Terluin et al. (2016) found that differential variance across symptoms can distort centrality metrics. That is, a symptom whose variance is minimal (restricted range) is likely to have low values of centrality metrics such as strength and expected influence. Accordingly, investigators have begun to compute correlations between symptoms' centrality values and standard deviations (McNally et al. 2017a) or variances (Elliott et al. 2020a,b). Nonsignificant correlations between a node's centrality and its index of variability can bolster confidence in the validity of the centrality metric.

Changes in symptom variance over time may explain certain otherwise puzzling findings. Network theory predicts that activation of symptoms is more likely to produce an episode of disorder in strongly interconnected networks than in weakly interconnected ones (Borsboom 2017). However, Beard et al. (2016) found that patients whose symptoms had diminished after a 2-week, intensive partial hospitalization program exhibited an increase in network density (i.e., sum of the edge weights in a network).

The seemingly paradoxical finding of density and symptom severity moving in opposite directions may have arisen as a function of increased symptom variance. Patients in the program described by Beard et al. (2016) tend to score uniformly high on symptom measures upon admission to the unit, producing a ceiling effect that constrains variance, thereby attenuating edge weights and thus network density. A diversity of responses to treatment, plus regression to the mean, would increase variance and increase the likelihood of network density increasing at posttreatment.

To say that we should therapeutically target high-centrality symptoms presupposes that we can in fact reduce the severity or frequency of these symptoms, and that we can do so with surgical precision. Moreover, selectively deactivating a symptom is more easily said than done. Biologists who conduct gene knockout studies do so with a "slim finger" that enables them to deactivate a single gene and then observe the downstream consequences of the knockout. Alas, clinical psychologists and psychiatrists are cursed with fat fingers. It is difficult to target a single symptom without simultaneously affecting other ones. For example, a clinician might prescribe a benzodiazepine to reduce insomnia for a patient with GAD, but the clinician's fat finger will simultaneously reduce anxiety as well.

Timing is important. To confirm that we have selectively isolated and deactivated a single target symptom, we need to know whether others were simultaneously affected. On the one hand, parallel deactivation of multiple nodes renders interpretation ambiguous. On the other hand, serial reduction is precisely what we want to see if our theory of action is correct. That is, once we deactivate the target source symptom, we should observe a subsequent decline in severity of downstream, connected symptoms.

Another issue is the timing of this therapeutic cascade or, for that matter, the development of an episode of disorder. For example, a person whose developing depression includes loss of appetite may not exhibit weight loss until days later, whereas someone developing panic disorder may misinterpret premature ventricular contractions as signifying a heart attack, and panic minutes later.

4.5. Temporality

At least two measurement points are required to test the therapeutic promise of centrality metrics. Although no cross-sectional study has yet to target specific high-centrality symptoms, several representative studies have provided clues. Examining CG symptoms from an observational study of spousal bereavement in the community, Robinaugh et al. (2016) found that reduction in the severity of symptoms with high expected influence centrality predicted psychological recovery following the loss better than did reduction in the severity of low-centrality symptoms.

A prospective longitudinal study in The Netherlands revealed that subjects who reported subthreshold levels of high-strength centrality depression symptoms at baseline were more likely to experience an episode of MDD during the 6-year follow-up period than were subjects whose subthreshold levels were of low strength centrality (Boschloo et al. 2016b). Notably, high-centrality harbingers of trouble included two hallmark symptoms of depression (depressed mood and anhedonia) and two nonspecific ones.

Computing networks on 142 patients who had undergone treatment for anorexia nervosa, Elliott et al. (2020a) tested whether symptoms high on expected influence centrality at baseline predicted outcomes at 6-, 12-, and 24-month follow-ups. Consistent with network theory, they found that the more central symptoms on expected influence (e.g., *feeling fat* and *fear of weight gain*) strongly predicted failure to recover and the degree of clinical impairment.

In another study of 910 patients with social anxiety disorder who had participated in RCTs of CBT, Rodebaugh et al. (2018) identified high-centrality symptoms at pretreatment baseline. They found that centrality metrics predicted how strongly changes in symptoms correlated with the remaining symptoms of the network. Oddly, these findings pertained solely to the measure of social anxiety in the network analyses and did not generalize to three other measures of social anxiety severity. Moreover, the most commonly endorsed symptom was an even more influential predictor of the course of response to treatment relative to strength centrality.

Analysis of a treatment study of women with PTSD (or subclinical PTSD) and substance use indicated that a symptom's strength centrality and predictability in the pretreatment network strongly correlated with the association between change in the symptom's severity and overall change across the network following treatment ($r = 0.79$ and 0.75, respectively) (Papini et al. 2020). However, the mean severity of a symptom at pretreatment was not significantly predictive ($r = 0.27$).

Metrics other than centrality have been assessed as harbingers of outcome over time. After developing a method for comparing network structures, van Borkulo et al. (2015) identified two groups of individuals who had participated in a naturalistic longitudinal cohort study of depression. One group had recovered by the 2-year follow-up, whereas the other group remained depressed. They compared the network density (sum of the edge weights) between the two groups and discovered that the group with persistent symptoms had greater network density at baseline than did the group whose members had recovered.

However, a subsequent study of depressed adolescents tempered enthusiasm for network density as a predictor of response to treatment (Schweren et al. 2018). The baseline network density of those who failed to recover following treatment was not statistically greater than the network

density of those who recovered. That is, network density predicted failure to recover in a naturalistic follow-up study of adults, some of whom did receive treatment, but it was unrelated to response to treatment in this adolescent patient sample.

Finally, a related study of important clinical significance concerned symptomatic harbingers of first-episode MDD in 768 Dutch subjects (Blanken et al. 2020). Applying the novel technique of network outcome analysis, Blanken et al. (2020) discovered that sleep-onset insomnia, but not sleep duration, directly predicted the onset of depression over the course of the following 6 years.

5. TEMPORAL TIME-SERIES NETWORKS

Most network studies concern cross-sectional analyses of psychiatric symptoms on large numbers of subjects. Most of these studies also involve data sets collected for other purposes.

Cross-sectional studies have been a rich source of hypotheses about functional relations among symptoms construed as potentially causal systems. However, they have two chief limitations. First, because the networks depict relations among symptoms at the aggregate, group level, it is unclear how often the same patterns obtain at the level of the individual person (i.e., the ergodicity question). Second, static, cross-sectional networks cannot disclose how interactions among systems unfold over time (i.e., the temporal question). I next address how investigators are grappling with these questions.

Commenting on van Borkulo and colleagues' (2015) study, Bos & Wanders (2016) cautioned against drawing inferences from cross-sectional networks to individuals within the group. Relations among variables at the group level, they argued, need not apply at the level of the individual (e.g., Bos et al. 2017). While acknowledging this possibility, van Borkulo et al. (2016) replied that it seems unlikely that associations between symptoms at the individual level would depart dramatically from those at the group level. For example, it is implausible that the more a person feels worthless, the less likely the person is to experience depressed mood. Individual patterns, they suggested, are more likely to differ from group patterns in degree, not in kind.

However, inferences across levels can sometimes be wildly incorrect, exemplifying the ecological fallacy (Robinson 1950, Selvin 1958). Consider the relation between typing speed (i.e., words per minute) and error rate (i.e., percentage of typos) (Hamaker 2012). At the level of the group, these variables correlate negatively; experienced typists are faster and less error-prone than novices are. Yet at the level of the individual, the variables are positively correlated; the faster anyone types, the more likely it is that errors will occur, regardless of level of experience.

A clinical example of correlations between variables switching sign across levels is what Dückers et al. (2016, p. 300) called "the vulnerability paradox" in the cross-national prevalence of PTSD. Although socioeconomically disadvantaged individuals exposed to trauma are at heightened risk for PTSD, countries characterized by socioeconomic disadvantage have lower PTSD prevalence rates than do advantaged countries even when rates of trauma exposure are indistinguishable. However, the paradox vanishes if one sidesteps the ecological fallacy by suspending cross-level inferences while identifying the factors accounting for this otherwise surprising finding (McNally 2018).

Nevertheless, the analysis of van Borkulo et al. (2016) agrees with that of Bos & Wanders (2016) regarding the value of network studies based on intensive collection of time-series data. Their agreement rests partly on the hitherto esoteric concern about ergodicity (or rather the lack thereof) in the psychological, social, and medical sciences (e.g., Fisher et al. 2018, Hoffart & Johnson 2020, Molenaar 2004, Molenaar & Campbell 2009). The concern is that findings emerging at the group (i.e., interindividual) level will fail to generalize to the level of the person (i.e., intraindividual) in the group unless the relevant process is ergodic. A process is ergodic

when the mean and variance are the same for the group as for each individual member of the group (i.e., homogeneity criterion) and neither the mean nor the variance changes over time (i.e., stationarity criterion). Strict ergodicity is very rare, and some have argued that it is not a binary matter—ergodic versus nonergodic—but, rather, a matter of degree (Adolf & Fried 2019, Medaglia et al. 2019). That is, as departures from strict ergodicity become more pronounced, it becomes more hazardous to generalize from the group to the individual. Fisher et al. (2018) have provided methods of assessing such departures. However, to do so requires the collection of intensive intraindividual assessments. Finally, it remains unclear how clinicians should cope with the stationarity criterion. Most would expect reasonably stable means and variances during the pretreatment baseline period as well as following successful treatment. But the whole point of treatment is to reduce the variances and means for symptom measures over the course of therapy, and it is unclear whether detrending the data is entirely appropriate clinically.

Furthermore, cross-sectional network analyses are incapable of capturing the dynamic features of psychopathology. Accordingly, researchers have increasingly collected time-series data whereby subjects rate their symptoms (or moods) in response to being pinged on their smartphones multiple times per day for (usually) several weeks (e.g., Bringmann et al. 2013, Wichers 2014). For example, van de Leemput et al. (2014) studied the dynamic features of networks and identified markers of impending tipping points whereby people suddenly shift from a healthy stable state to a depressed one (i.e., hysteresis) (Borsboom 2017, Cramer et al. 2016). They had healthy and depressed participants digitally rate four moods (content, cheerful, sad, and anxious) several times daily for 5–6 days. They found that increased variance in the ratings and increased temporal autocorrelation of ratings of negative moods were harbingers of flipping into depressed states. These metrics signify critical slowing whereby dynamic networks take increasingly longer to rebound from disturbances, eventually culminating in a tipping point. However, Bos & De Jonge (2014), cautioning that the authors relied on between-subject variability, suggested that investigators should disaggregate intraindividual variability from interindividual variability to make predictions on the level of the person.

In another study, Pe et al. (2015) assessed the emotional states of subjects with MDD and healthy comparison subjects multiple times daily for 1 week. Relative to healthy subjects, those with MDD had a notably dense network, especially for negative emotions. Bringmann et al. (2015) used multilevel vector autoregressive (MVAR) modeling to analyze weekly symptom reports of depressed patients over 14 weeks and noted the importance of anhedonia in the temporal dynamics of the disorder.

MVAR studies enable the computation of three networks (e.g., Aalbers et al. 2019, Bringmann et al. 2013, Epskamp 2020, Fisher et al. 2017). The temporal network depicts how variables at one time point (time t) predict variables in the next time window (time $t + 1$), including how a variable assessed at the first time point predicts its value at the next one. These are directed networks, and thus they permit the computation of instrength centrality and outstrength centrality. A node scoring high on the former is strongly predicted by the values of other nodes in the previous assessment window, whereas a node scoring high on the latter strongly predicts the values of other nodes in the next time window.

The contemporaneous network depicts the (undirected) edges connecting nodes within the same measurement window. Typically, this network features edges that represent the partial correlations between nodes in this measurement window after adjusting for all other variables in the window as well as all other variables in the previous measurement window. Contemporaneous networks have a better chance of detecting functional relations between nodes that unfold faster than a temporal network featuring assessment windows that occur, say, 3 hours apart (Epskamp et al. 2018b). For example, consider a patient with panic disorder who experiences skipped

heartbeats and then fearfully interprets them as an impending heart attack. The edge between these two nodes would be detectable within a measurement period but would be missed across measurement periods in the temporal network. That is, contemporaneous networks can detect faster-moving processes than can temporal networks. Finally, "traditional" networks featuring between-subjects associations are also computable. Importantly, time-series data can yield both temporal and contemporaneous networks on a single subject (Epskamp et al. 2018b). Fisher's group has conducted intensive, time-series studies involving patients with GAD, MDD, and PTSD (Fisher et al. 2017, Reeves & Fisher 2020). Despite similar diagnoses, patients often differ in the temporal dynamics and in the symptoms that score high on centrality metrics.

Relevant to this issue is a novel method for vector autoregressive modeling called impulse-response function (IRF) analysis, automated in an R package (Blaauw et al. 2017). The automated IRF function estimates the impact of a variable (e.g., an increase in stress, a bout of exercise) on the network as a whole. Bos et al. (2018) used such methods to model the impact of variables (e.g., a boost in positive affect) on the entire network of mood states in subclinical subjects with and without anhedonia. The method allows one to estimate the impact of these variables on individuals as well as on aggregated data from groups of individuals. In one sense, it provides a solution to the fat-finger problem hobbling centrality metrics.

Finally, Robinaugh et al. (2019) have taken the dynamic approach to network analysis to another level. Using simulations involving difference equations and differential equations, they specified computational, testable network models of panic disorder with agoraphobia. It is too soon to tell where all this will lead, especially as statisticians and psychometricians are devising new methods faster than most of us clinical psychologists can master them. Ultimately, the crucible of evaluation will turn on whether network analysis can enhance treatment efficacy, perhaps as yet another pathway for personalized assessment and intervention.

SUMMARY POINTS

1. The network approach to characterizing psychopathology departs from traditional latent categorical and dimensional approaches. Disorders are emergent phenomena arising from the causal interactions of their symptomatic elements. Hence, symptoms are constitutive of disorder, not reflective of it.
2. The approach has inspired an exponential growth in the number of empirical studies as well as novel approaches to modeling the structure and dynamics of psychopathology.
3. Network researchers have developed a solution to the "problem" of rampant comorbidity among presumptively discrete syndromes such as major depressive disorder and generalized anxiety disorder. Indeed, comorbidity emerges as a natural consequence of conceptualizing syndromes as networks of partially overlapping symptom clusters.
4. Researchers have devised and adapted metrics for gauging the "importance" of symptoms, conceived as their interconnectedness with other symptoms. Strength centrality and expected influence centrality have proved especially robust. However, it remains uncertain whether they will prove useful as therapeutic targets whose resolution hastens recovery from an episode of disorder.
5. Concerns about the replicability of findings in the network field have been reassuringly settled.

FUTURE ISSUES

1. Most network studies feature cross-sectional snapshots of data aggregated across many subjects. These studies furnish hypotheses about possible causal relations, but they have limitations. First, it is often unclear whether patterns among symptoms emergent at the level of the group hold at the level of the individual person. Second, cross-sectional networks lack the temporal dimension exemplified by time-series studies.
2. Accordingly, investigators have developed methods for computing temporal networks by analyzing time-series data that comprise multiple daily assessments of moods and symptoms over the course of weeks. These studies will surely proliferate in the near future as they characterize predictive relations among symptoms unfolding over time. Moreover, time-series data enable computation of networks on single patients.
3. Relatively unexplored are how variables external to the network itself (e.g., biological variables, social variables) affect network structure and dynamics.
4. A key goal for the future is to devise methods for minimizing the burden on subjects while still collecting valid mood and symptom data in real time before, during, and after treatment. Measures that do not rely on self-report may prove valuable.
5. A very important goal is to evaluate how well network theory, metrics, and modeling can guide therapeutic interventions. Another is to devise user-friendly procedures to permit their use among therapists untrained in the advanced statistical methods of network psychometrics.
6. Dynamic computational network modeling enabling the testing of specific theories of disorder is on the horizon.

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Contents

Errata

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