An Answer to "So What?" Implications of Network Theory for Research and Practice

Payton J. Jones, M.A., and Donald R. Robinaugh, Ph.D.

Research and practice in psychiatry and clinical psychology have been guided by differing schools of thought over the years. Recently, the network theory of psychopathology has arisen as a framework for thinking about mental health. Network theory challenges three common assumptions: psychological problems are caused by disease entities that exist independently of their signs and symptoms, classification and diagnosis of psychological problems should follow a medical model, and psychological problems are caused by diseases or aberrations in the brain. Conversely, network theory embraces other assumptions that are well accepted in clinical practice (e.g., the interaction of thoughts, behaviors, and emotions, as posited in cognitive-behavioral therapies) and integrates those assumptions into a coherent framework for research and practice. In this article, the authors review developments in network theory by focusing on anxiety-related conditions, discuss future areas for change, and outline implications of network theory for research and clinical practice.

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Some of the most radical shifts in psychiatry have been initiated by reconceptualization of what it means to have a mental illness. Such reconceptualization often entails a change in the causal attributions given to aberrant, distressing, or dysfunctional patterns of thoughts, emotion, and behavior (i.e., the experiences commonly referred to as the signs and symptoms of mental illness). For example, psychoanalytic approaches dominated the early 20th century with a focus on unconscious conflicts in the mind. Distressing thoughts, emotions, and behaviors could be attributed to these unconscious conflicts, and considerable effort was given to characterizing these conflicts and determining the best way to resolve them. Early behavioral theories explained mental problems exclusively via the environments of organisms. Mental problems were explained within the simple framework of stimulus-response. More recently, progress in neuroscience has led to a shift toward theories that focus on the structural and functional connectivity of specific brain circuits. These theories attribute symptoms to dysfunction in the brain, and considerable effort has been given to identifying the precise abnormalities that give rise to symptoms.

Despite clear differences, these approaches have a common theme: they attribute psychopathology to a single or relatively constrained set of causal factors that are distinct from the symptoms of mental disorders (e.g., unconscious conflicts or a dysfunctional brain circuit). This is perhaps not surprising, because psychiatry and related disciplines have long been influenced by the enormous success achieved by medical science in the 19th century with the discovery of bacterial causes for many common medical conditions (1). The success of the bacterial or medical model coincided with the rise of psychiatric research and shaped the way we think about and study mental disorders (2). This conceptual framework is so pervasive that it is rarely articulated, but it brings with it a set of interrelated implicit assumptions about the ontology and etiology of mental disorders.

First, this framework typically carries with it the assumption that psychological problems are caused by disease entities that exist independently of their signs and symptoms. Indeed, the very use of the term "symptom" to refer to patients' cognitions, emotions, behaviors, and somatic experiences is rooted in the notion that they arise from disease entities that exist independently of these experiences. Similarly, the common encouragement to not merely treat the symptom, but to treat the disease, presumes that there is a disease independent of the symptoms to treat. Second, this framework carries the assumption that classification and diagnosis of psychological problems should follow the medical model. The diagnostic system used in psychiatry is structured around the assumption of underlying disease entities that give rise to specific symptom patterns. These diseases are presumed to be independent not only of symptoms, but also of one another. A diagnostician's task is to use signs and symptoms to determine which disease entities may be present. Third, this framework often, although not always, carries with it the assumption that psychological problems are caused by diseases or aberrations in the brain. Elements of this assumption have been challenged many times (3, 4), but it remains influential. The search for lesions,

abnormalities in structure and function, chemical imbalances, aberrant communication between major networks, and other brain origin stories for mental disorders dominates much of modern psychiatric research.

THE NETWORK THEORY OF PSYCHOPATHOLOGY

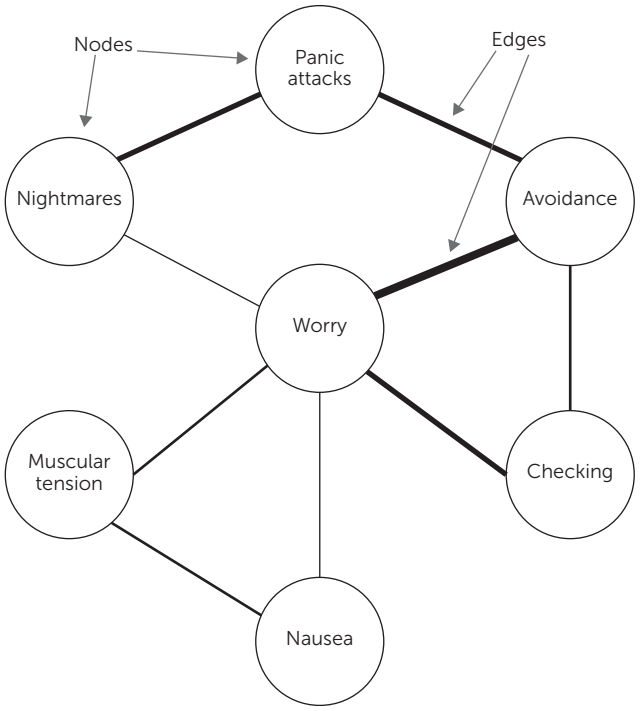
The network theory of mental disorders offers a different conceptualization of mental disorders that challenges the assumptions enumerated in the preceding paragraph. The central tenet of network theory (5) is simple: the signs and symptoms of mental disorders commonly co-occur, at least in part, because of a network of causal relations among the symptoms themselves. For example, difficulty sleeping is plausibly causally related to fatigue and to problems with concentration; physiological reactivity to reminders of trauma may elicit trauma-related avoidance; and obsessions (e.g., regarding contamination) frequently give rise to compulsions (e.g., hand washing). Symptoms may be initially activated through outside forces (e.g., a traumatic event), but the symptoms cohere as a syndrome that persists over time because of mutually reinforcing relationships among the symptoms (6).

Motivated by this theory, researchers have begun to develop methods (7–12) for investigating networks of symptoms and other psychological characteristics. In these network psychometric methods, the signs, symptoms, and other psychological problems are called "nodes," and the relationships between these nodes are called "edges" (Figure 1). Most commonly, edges represent statistical associations between two symptoms. The merits of various network psychometric methods, however, are distinct from the merits of network theory itself. No single statistical method is sufficiently informative to address the complexities of a psychological theory, which must be tested through multiple lenses. That said, network psychometric methods allow for a visualization of relationships that helps illustrate how mental disorders are conceptualized by network theory in its most basic form. With the aid of this visualization, we can review the implications of network theory for each of the three common assumptions that we have argued are prominent in psychiatric research.

Assumption 1: Mental Illnesses Exist Independently of Their Signs and Symptoms

Network theory posits that the set of psychological problems traditionally referred to as "mental illnesses" do not exist independently of their signs and symptoms. Instead, the relationship is mereological: whole to part. To illustrate, consider a school of fish. A school of fish does not exist independently from the fish. Whereas an independent disease entity (e.g., a cancerous tumor) can exist without the presence of any symptoms, a school of fish cannot exist without fish. In a similar way, network theory claims that mental disorders arise from a set of components (e.g., thoughts, emotions, behaviors, somatic experiences, and other relevant biological, psychological, or social factors) and the causal relationships among these components. The components of the network include the experiences we commonly refer to as symptoms. In other words, a pattern of pervasive worry is not caused by generalized anxiety disorder, the pattern of pervasive worry (and accompanying features) is generalized anxiety disorder.

FIGURE 1. Example network model of worry and closely related nodes



This idea is illustrated in Figure 1. In this figure, there is no independent panic disorder node that causes panic attacks, avoidance, and worry. Instead, panic disorder is a state that the network can be in: a state characterized by the presence of these specific experiences. The stable, concurrent presence of panic attacks, avoidance, and worry is panic disorder. In other words, network theory provides an explanation for the observation that some symptoms tend to cohere as syndromes that obviates the need for an independent underlying cause. In doing so, it challenges the assumption that mental illnesses must necessarily exist independently of their signs and symptoms.

Notably, when taken literally, the term "symptom" itself is incompatible with network theory. The word "symptom" denotes a surface-level indicator of an underlying cause. Network theory asserts that most things that have traditionally been called symptoms (e.g., worry) are not indicators of underlying diseases, but are nodes in a causal web. This idea—that many "symptoms" are in fact causal players—is a key concept in network theory. Moreover, some things that have not traditionally been called symptoms (e.g., the belief that bodily sensations associated with arousal are dangerous) fit nicely as nodes within these networks (e.g.,

panic disorder) (13–14). Therefore, we prefer the terms "nodes" or "elements" rather than "symptoms" when speaking of the constituent parts of networks, because these terms clarify that these parts can be understood not as passive indicators, but rather as active causal components that constitute the mental disorder.

Assumption 2: Classification of Psychological Problems Should Follow the Medical Model

Many medical conditions can be summarized by a classification system that simultaneously describes both cause and consequence. When a certain medical symptom pattern arises (dizziness, shakiness, sweating), physicians are tasked with providing a differential diagnosis to determine which disease entity has led to the symptoms (influenza or hypoglycemia). By identifying the presence of the disease entity (which, together with the symptoms, constitutes a cause-consequence pair), the physician can typically rule out the presence of other disease entities. Such classification systems can be used to great effect when specific causes (i.e., diseases) reliably lead to the same general consequences (i.e., symptoms), when some consequences are unique to a given cause (e.g., pathognomonic symptoms, such as Koplik spots), when the different causes are mostly independent of one another, and when the consequences do not feed back into the causes (e.g., dizziness from hypoglycemia does not cause the flu). We will here refer to such cause-consequence classification systems as a medical model of classification, although we note that this phrase is fraught, defined in different ways by different sources, and an oversimplification of the actual practice of modern medicine.

The network perspective challenges the assumption that classification should follow this simple medical model. Most fundamentally, as we argued in the previous section, it does so by demonstrating that there need not be an independent underlying cause that gives rise to symptoms and, thus, there need not be an underlying disorder to which the symptoms can be attributed. However, there are other characteristics of psychopathology that further argue against a simple cause-consequence classification system. First, the boundaries between our diagnostic categories are fuzzy, with many symptoms appearing across multiple disorders. Second, equifinality (a given end state can be reached by many different starting points) and equipotentiality (many given end states can be reached from the same starting point) tend to be the rule, rather than the exception, in psychiatry. A given component in psychiatry (e.g., impaired sleep) can have diverse consequences (e.g., poor concentration, mania), and a given consequence may have many potential causes (e.g., poor concentration may arise not only from impaired sleep, but also from anhedonia, preoccupying worry, or intrusive memories of trauma). Third, symptoms in psychiatry are often causal agents with their own set of effects. In an idealized medical model, observable symptoms are the endpoint of the causal system: dizziness arising from hypoglycemia does not cause the flu, allowing diagnosticians to confidently rule out the flu if they attribute dizziness to hypoglycemia. In practice, things are perhaps not so simple (e.g., dizziness may not cause the flu, but it may lead to nausea, falls, or other health consequences), but this general framework holds well enough to make informative diagnostic decisions.

In psychiatry, the complex web of causality is even more dense. For example, anxiety about social situations can (and frequently does) result in problematic patterns of alcohol use, which may give rise to feelings of worthlessness, loneliness, and depression. Similarly, intrusive memories following a trauma may evoke physiological reactivity and, in turn, fatigue, social disconnection, anhedonia, and irritability. In other words, symptoms are not the end of the causal story; they frequently result in consequences that do not respect diagnostic boundaries. Together, these commonly observed features of psychopathology present significant obstacles for a medical model of classification.

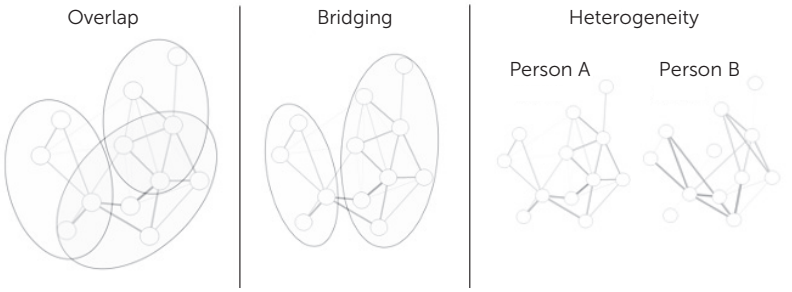
Viewing psychopathology from a network lens provides a new perspective on these obstacles. As depicted in Figure 2, the fuzziness among diagnostic categories arises because mental disorders are overlapping communities of causally interacting components, not discrete disease entities. For example, posttraumatic stress disorder symptoms overlap considerably with the symptoms of depression, a substantial challenge for diagnosticians working from a medical model, but an expected state of affairs when viewed from a network perspective, where individual symptoms can have a range of causal effects. Even in the absence of syndromic overlap, syndromes may bridge to each other if the symptoms of one disorder cause symptoms of another (e.g., persistent social anxiety may lead to problematic alcohol use). In addition, network theory allows that similar components may have divergent effects, thereby producing considerable heterogeneity across individuals in symptom presentation.

To be clear, our argument is not that classifications are useless in the context of network theory or that these classifications should be avoided altogether. Even rough, messy, and imprecise classification systems can be useful for quickly summarizing and communicating information in a *lingua franca* (15). For example, knowing that a patient has a diagnosis of panic disorder provides us information about potential causal relationships among panic attacks, worry, and avoidance that may produce this pattern of symptoms. However, it is important that the diagnoses in our classification systems are treated as rough shorthand and not as established independent disease entities.

In situations where classification is complex, it is often beneficial to have multiple systems of classification depending on one's goals (e.g., one classification system for treatment and another for etiology) (16). Indeed, multiple classification systems have been used productively in biology, and such a system could readily apply to clinical psychology (16, 17). As an example, consider that treatments for depression show different efficacy for different people, and this does not seem closely linked to the depression diagnosis as defined by its *DSM* signs and symptoms. Indeed, the treatments with the

best evidence for depression (antidepressantmedications and cognitive-behavioral therapy) are not even specific to depression at all (18–20). In developing a treatment-focused classification system, other variables might bemore important than the same symptom patterns we have relied on for years. For instance, one study (21) has found that marital status (being married or cohabiting) predicts an advantage of cognitive-behavioral therapy over antidepressant medication among individuals with depression. In a treatment-focused classification system, marital status might be an important variable to consider (perhaps more important than the specifics of the symptom patterns). Classification systems could also rely on the within-person temporal dynamics of variables and relationships between variables, rather than just on the presence or absence of the variables (22). In sum, there may be advantages in departing from psychiatry's traditional view of classification.

FIGURE 2. Three challenges of diagnostic classification as viewed from a network perspective: overlap, bridging, and heterogeneity[a]



[a] Overlap occurs when nodes are shared between multiple classifications (e.g., worry is common in almost all anxiety disorders). Bridging: even when categories are constrained to be mutually exclusive, they are often bridged by important causal connections and are thus rarely truly disentangled in practice. Heterogeneity: relationships between symptoms are unlikely to be perfectly consistent across different individuals.

Assumption 3: Psychological Problems Are Caused by Diseases or Aberrations in the Brain

Perhaps most controversially, the network theory challenges the idea that psychological problems are caused by illnesses in the brain. Borsboom, Cramer, and Kalis (23) have presented three arguments for why a network view is incompatible with a reductive explanation of mental illnesses as brain disorders. Their first argument juxtaposes the "many causes" network model with a "common cause" brain disorder. If all symptoms are simultaneously dependent on a common latent variable, then one can accurately describe the system in terms of a common cause (in the brain). However, if a network model is correct, and there are many interacting causes, this type of reduction is blocked. Second, they argue that it is implausible that neurobiological causes could be a simultaneous explanation for all symptoms, because many symptoms are rational in the context of the specific content of other symptoms (e.g., hand washing is rational when one believes one has been contaminated). The specific content of thought is therefore necessary to explain the full symptom pattern. The fact that many symptoms are "intentional" (i.e., about something) means that there are an infinite variety of possible symptom manifestations. A small difference in thought content could lead to radical differences in behaviors or other symptoms. Third, they argue that specific connections in symptom networks rely on cultural and historical contexts, again blocking the idea that mental illnesses can be reduced to a single common cause in the brain invariant to context.

Critically, this does not mean that psychological problems cannot result from insults to or impairments in the brain—history is replete with such examples, from syphilitic insanity to postconcussive syndrome. Rather, it provides a plausible alternative to this model that may apply to some specific types of problems. Equipped with this alternative account, we can begin to ask which type of theoretical framework offers the best explanation for a particular syndrome. Given the lack of progress in identifying reliable brain markers of most mental problems over the past century, we believe there is good reason for us to devote greater energy to alternative accounts, such as network theory.

We would again emphasize that our call to further investigate alternative accounts need not be an inflammatory position—network theory does not assert that structural or functional abnormalities in the brain are irrelevant. Within the framework of network theory, brain processes can be viewed as a separate level of analysis that can help explain the connections among nodes (6). Alternatively, these processes could be directly incorporated as components that play an important role within the network, providing a biopsychosocial network that accounts for the complex interplay among components across separate levels of analysis (13). Ultimately, we suspect that an integration of network theory with research on the genetic and neurobiological factors that contribute to psychopathology will be the most fruitful path toward meaningful progress in psychiatric research and practice.

IMPLICATIONS OF NETWORK THEORY FOR RESEARCH

The network view of psychopathology provides credible challenges to assumptions underlying the medical model of mental illness. Moreover, it provides a new lens for conceptualizing problems of anxiety and stress. Examining anxiety and traumatic- and stress-related disorders through this lens suggests changes may be warranted in the ways that we as scientists and practitioners approach research, diagnosis, and treatment. We consider several of these implications here.

Avoid Reification

Classifications of disorders are unlikely to be precise, and diagnostic labels should not be reified and treated as established

disease entities (24). Because the network theory challenges the view that disorders are discrete entities, future network research should work toward elucidating more flexible systems of classifying mental problems. The presence and absence of specific symptoms are useful for creating classifications of mental problems, but other factors (including network properties, such as the relationships between symptoms) may also be useful.

Shift the Level of Analysis

Analyses that focus on disorders (e.g., the joint occurrence of major depressive disorder and social anxiety disorder) rather than on individual components of disorders (e.g., the joint occurrence of depressed mood and social avoidance behavior) are useful descriptively, but they are unlikely to produce substantial progress in our understanding of psychopathology on their own, because they entirely overlook the important component-level associations posited to give rise to these disorders and their joint occurrence. Analyses that focus on individual psychological problems (e.g., social avoidance) and the relationships among them (e.g., the effect of social avoidance on social fear) are likely to provide critical new information, helping us to understand not only how these problems interact as a causal system, but also how other genetic, neurobiological, or social components may affect this system (25).

Move Beyond Symptoms Alone

A focus on symptoms typically implies a focus on the specific symptoms encoded in the *DSM*. Yet *DSM* symptoms do not appear to have a privileged role in networks (26, 27). Symptoms are typically identified as such because they are highly distressing to the patient, highly visible, or because they seem especially tied to a specific syndrome (i.e., hallmark symptoms). But there is no reason to assume that the causal feedback loops generating mental disorders will exclusively contain experiences with these qualities. Accordingly, although symptoms may be a tractable starting point for considering which components may be present in the network, it will likely be important for networks to expand to encompass more than just the symptoms enumerated in diagnostic manuals (28, 29). Other well-researched factors in psychopathology (e.g., anxiety sensitivity) (30) can also be incorporated into network models. In addition, more complex models, such as hybrid networks, should be used to incorporate variables that are causally relevant but not necessarily part of the feedback loops that maintain a disorder over time (e.g., genetic factors or trauma) (28).

Specify Formal Theories

Ultimately, empirical network research aims to advance our understanding of precisely how a given disorder may operate as a causal system. That is, it aims to facilitate well-developed theories that can represent the system that gives rise to a disorder (e.g., a vicious cycle between arousal-related bodily sensations and perceived threat that gives rise to panic attacks). Recently, some (31) have argued that the causal structure of even the relatively simple systems thought to give rise to many anxiety and stress-related disorders will be difficult to estimate directly from empirical data. Instead, these researchers (32) have proposed that it may be necessary to formalize initial theories about how a disorder may operate as a causal system by expressing those theories as mathematical or computational models. Those models can be used to critically examine what the theory can explain and what it cannot, including the patterns of association identified in empirical network studies. In doing so, these initial formal theories can identify a theory's shortcomings, and thus opportunities to improve the explanatory breadth or precision of the theory, and move us toward greater understanding of the system that gives rise to the disorder.

Choose Statistical Models That Fit the Research Aims

Some common statistical models (i.e., [reflective] factor analysis) assume that a single underlying latent construct (e.g., a mental disorder) causes the emergence of diverse psychological problems. This assumption is broadly at odds with network theory (28, 33), which emphasizes the interactions among components. To best inform network theories, it is likely to be more fruitful to focus on statistical models that aim to examine the interrelationships among components. Although network psychometric models may serve this purpose in some cases, we would more fundamentally argue that researchers should carefully select the statistical models that will best inform their theories. In our experience, it is often the practice of researchers in psychiatry to focus on statistical models that are popular, well-accepted, or familiar to the researcher. However, this practice can result in choosing statistical models ill-suited to addressing the research questions of interest. Statistical tests and their interpretations should be justified by the theoretical aims of the researchers. Researchers should specify their aims and then choose statistical models accordingly; they should not choose statistical models and then specify their aims accordingly.

Take Causality Seriously and Conduct Experiments

Most empirical research in the network literature has focused on cross-sectional network analyses that use regularized, partial correlations between pairs of nodes (7). Within-person analyses have also been conducted, typically by calculating lagged partial correlations, lagged regression parameters, or similar statistical parameters (7). Both types of analyses can provide valuable information about interrelationships among components. However, both are also extremely limited in their ability to inform our understanding of causal interactions among components (10).

Causal interactions among components are at the heart of network theory. Thus, it is essential that network researchers develop and integrate randomized experiments into their investigations, because such experiments provide

insights into causality that cannot be matched by cross-sectional or within-person observational studies. Such experiments are not the only tool for causal inference, but we believe they have been especially underutilized in the network approach literature.

IMPLICATIONS FOR PRACTICE

In the clinical world, anxiety and stress-related disorders have long been understood to result from interactions among symptoms (e.g., anxiety and avoidance), environmental stressors, biological systems (e.g., the hypothalamic-pituitary-adrenal axis), and other key variables. This assumption is exemplified in functional analysis (34, 35), where therapists are encouraged to deeply consider the functions of patients' behaviors, thoughts, and emotions, and the relationships among them. Network theory thus provides a theoretical framework for thinking about mental problems that is consistent with how clinicians have been thinking about these problems for years. The assumptions made in this theoretical framework suggest several key implications for clinical practice.

Avoid Reification

Here again, we would stress that diagnosis does not reveal an underlying disease that exists within a patient. Classifying patient conditions into subgroups (e.g., as social anxiety or obsessive-compulsive disorder [OCD]) may be useful for descriptive and communicative purposes, but these classifications should be interpreted as rough descriptions of common patterns, not as reified disease entities. The value of these descriptions through the lens of network theory is that they may point us toward the plausible systems driving maintenance of these syndromes. For example, knowing that an individual has received a diagnosis of OCD suggests a causal feedback loop between obsessions and compulsions that leads these symptoms to persist together over time.

Move Toward Process-Based Therapy

Therapists should continue to use functional analysis and case conceptualization (i.e., process-based therapy) (34–36) throughout the course of treatment, attempting to understand the relationships between patients' thoughts, behaviors, and emotions as a fundamental approach to psychotherapy.

Focus on Targeted Symptoms

One productive approach may be to focus on a set of targeted symptoms jointly defined by the clinician and patient. This clinical approach fits with the idea of self-reinforcing feedback loops. By thwarting the activation of specific nodes in feedback loops, problematic patterns can be efficiently disrupted. Choosing the ideal symptoms to target is not a trivial task and differs for individual patients. In choosing symptoms to target, clinicians should carefully consider which symptoms are present, directly malleable, causally linked to other downstream symptoms (considering both generalized scientific evidence and personalized case conceptualization), and relevant to the patient's goals. To measure the success of therapy, clinicians and patients should jointly track progress on specific goals.

Expect Comorbid Conditions and Avoid Ruling Them Out

A physician who establishes hypoglycemia as the cause of dizziness is probably justified in ruling out the presence of other potential causes, such as influenza. This is because these conditions follow a traditional disease-symptom pattern with minimal causal influence from the symptom (i.e., dizziness) to other conditions (e.g., the flu). Network theory predicts the opposite. From the perspective of network theory, one should not rule out one diagnosis on the basis of the presence of another (e.g., ruling out OCD because generalized anxiety is present and more prominent). Rather, network theory predicts that the presence of one problematic pattern of nodes increases the likelihood of the presence of another problematic pattern. That is, the presence of one disorder should lead us to expect that other disorders may also be present.

CONCLUSIONS

The network approach to psychopathology is a quickly growing viewpoint in the field of psychiatry and clinical psychology. This approach argues that mental disorders do not arise from latent disease entities. Instead, they arise from complex feedback loops among behaviors, thoughts, emotions, and other relevant variables. The implications of this framework are widespread, and in this article, we have aimed to provide a roadmap for research and practice into psychological problems. In the future, researchers serious about developing network theories should expand the scope of their investigations to include nonsymptoms and to further investigate key causal links by using a variety of methods, especially randomly assigned experiments. Clarifying causal connections may help theorists to build formal computational network theories of psychiatric syndromes (32). By challenging assumptions that have guided clinical research for many years, the network approach to psychopathology has provided a burst of research that, if channeled productively in further research, may lead to significant enhancements in understanding, treating, and preventing psychological problems.

AUTHOR AND ARTICLE INFORMATION

Department of Psychology Harvard University, Cambridge, MA (Jones); Center for Anxiety and Traumatic Stress, Massachusetts General Hospital, Boston, MA (Jones, Robinaugh). Send correspondence to Mr. Payton J. Jones (payton_jones@g.harvard.edu).

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