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What is This?

Mental Disorders as Causal Systems: A Network Approach to Posttraumatic Stress Disorder

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Abstract

Debates about posttraumatic stress disorder (PTSD) often turn on whether it is a timeless, cross-culturally valid natural phenomenon or a socially constructed idiom of distress. Most clinicians seem to favor the first view, differing only in whether they conceptualize PTSD as a discrete category or the upper end of a dimension of stress responsiveness. Yet both categorical and dimensional construals presuppose that PTSD symptoms are fallible indicators reflective of an underlying, latent variable. This presupposition has governed psychopathology research for decades, but it rests on problematic psychometric premises. In this article, we review an alternative, network perspective for conceptualizing mental disorders as causal systems of interacting symptoms, and we illustrate this perspective via analyses of PTSD symptoms reported by survivors of the Wenchuan earthquake in China. Finally, we foreshadow emerging computational methods that may disclose the causal structure of mental disorders.

Ever since its diagnostic debut more than 30 years ago (American Psychiatric Association [APA], 1980), posttraumatic stress disorder (PTSD) has been among the most controversial syndromes in our nosology (Brewin, 2003; McNally, 2003; Rosen, 2004). One abiding controversy concerns its ontological status (McNally, 2012). Is PTSD a timeless, universal psychobiological entity emerging whenever people encounter overwhelming trauma (e.g., Osterman & de Jong, 2007; Yehuda & McFarlane, 1987)? Or is it a socially constructed idiom of distress arising in the wake of the Vietnam War and confined chiefly to contemporary Western culture (e.g., Summerfield, 2001; Young, 1995)?

Even some who never doubt the natural status of PTSD question whether it qualifies as a categorical entity, as implied by the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; APA, 2013). For example, after applying Meehl's (1995) taxometric methods to PTSD symptoms reported by Vietnam veterans, Ruscio, Ruscio, and Keane (2002) concluded that symptoms reflect the high end of a continuum of stress responsiveness rather than a discrete clinical entity. These differences notwithstanding, traumatologists on both sides of the categorical versus dimensional divide agree that PTSD symptoms should be psychometrically construed as reflective indicators of an underlying latent variable. According to this view, the construct of PTSD denotes a latent variable that functions as the common cause of each of the PTSD symptoms (Bollen & Lennox, 1991; Edwards & Bagozzi, 2000). Thus, PTSD symptoms cohere syndromically because they share a common determinant, just as headaches and dizziness may arise

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together if caused by a brain tumor (Borsboom & Cramer, 2014).

This conception of the relation between symptoms and disorder is not unique to PTSD. It is the primary lens through which our field views psychopathology, and it motivates the endeavor to identify the underlying disease entities that produce the symptoms of mental disorders (Borsboom & Cramer, 2014). This endeavor has not been without success in psychiatric research. For example, one of the most serious mental disorders prevalent in the 19th century was general paresis of the insane (GPI). The syndrome comprised a variety of symptoms including hallucinations, delusions, fatigue, apathy, and mood disturbance. It eventually progressed to dementia, paralysis, and usually death (Brown, 2000; Noguchi & Moore, 1913). After decades of research aimed at identifying the common cause of these symptoms, Noguchi and Moore (1913) identified *Treponema pallidum*, the spirochete bacterium that causes syphilis, in the brains of those who had died from GPI, thereby confirming Alfred Fournier's hypothesis about the causal connection between syphilis and GPI (cited in Brown, 2000).

Doctors suspected that an underlying common cause produced the diversity of symptoms in patients with GPI, and they eventually identified the physical referent of this conjectured latent entity: the spirochete bacterium. Unfortunately, similar successes have been rare during the century of research on psychopathology since Noguchi and Brown's discovery (Kendler, 2005). The field has certainly discovered many social, psychological, and neurobiological variables important in the etiology of mental disorders such as PTSD, but none seem to qualify as the central disease entity. The causes of mental disorders appear massively multifactorial (Kendler, 2012), thereby undermining the plausibility of a common cause explanation for the associations between symptoms.

In recognition of the limitations of the common cause view and the latent variable model that accompanies it, psychometricians have recently proposed a radically different approach to conceptualizing mental disorders. In this *network approach*, symptoms do not covary because of their common dependence on a latent disease entity; they covary because they are coupled through direct causal and homeostatic links (Cramer, Waldorp, van der Maas, & Borsboom, 2010; see also Kendler, Zachar, & Craver, 2011). The network approach aspires to disclose causal relations among symptoms in contrast to traditional categorical and dimensional approaches that conceptualize symptoms as expressions of an underlying, latent entity that causes symptom emergence (Borsboom, 2008; Borsboom, Cramer, Kievit, Scholten, & Franić, 2009; Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011; Borsboom, Epskamp, Kievit, Cramer, & Schmittmann, 2011; Cramer et al., 2010; Cramer, van der Sluis, et al.,

2012; Schmittmann et al., 2013). The central idea is that symptoms are *constitutive* of mental disorder, not *reflective* of it. Disorders, including PTSD (McNally, 2012), are networks of interacting, possibly self-reinforcing symptoms, not underlying disease entities that produce symptoms. That is, the network approach conceptualizes mental disorders as causal systems comprising their constitutive symptoms, and its analytic methods aspire to distinguish causal relations between symptoms from mere correlational relations between them.

The concept of *cause* figures in both latent variable and network models, but in different ways. For example, in the traditional latent variable approach, a stressor causes depression, and this underlying disease entity, in turn, causes the symptoms that reflect its presence. Thus, the latent variable can be seen as a mediator, transferring effects from stressor to symptoms. Accordingly, in the latent variable model, external variables, such as stressors, are usually modeled as conditionally independent of the symptoms (e.g., fatigue or concentration problems), given the latent variable (e.g., depression). Such conditional independence relations are a central building block of modern theories of causality (e.g., Pearl, 2009; Woodward, 2003), and accord with a causal conception of psychological measurement (Borsboom, Mellenbergh, & van Heerden, 2003, 2004).

In the network approach, causal relations also play an important role, but in a different fashion. In network models of depression, for instance, a stressor produces certain symptoms (e.g., sleep loss) that activate other symptoms (e.g., difficulty concentrating), possibly in circular, self-reinforcing ways. To be sure, the symptoms may be embodied psychobiologically, but there is no independent entity of *depression* apart from its constitutive symptoms. Thus, in this view, the symptoms are not mere passive psychometric indicators, but are active constituent ingredients of the disorder (see also Fried & Nesse, 2014; Fried, Nesse, Zivin, Guille, & Sen, 2014). Because symptoms often affect each other through feedback loops (e.g., insomnia → fatigue → feelings of worthlessness → insomnia), causal relations in psychopathology networks should not necessarily be viewed as directional; most are typically modeled as undirected networks (Constantini et al., 2014). The causal connotation that accompanies undirected network models implies that if one were to intervene clinically by addressing one symptom, this would affect symptoms directly connected to the target symptom, but only produce indirect effects elsewhere in the network. Such models are known as Markov random fields (Kindermann & Snell, 1980).

The purpose of our article is twofold. First, we review the psychometric and conceptual limitations of traditional latent variable models of mental disorder. These limitations motivate the search for an alternative

embodied in the causal systems perspective. Second, we illustrate the application of the causal systems approach by conducting network analyses of PTSD symptoms reported by survivors of the 2008 Wenchuan earthquake in China.

Limitations of Latent Variable Approaches to Mental Disorder

Attempts to understand the variegated tapestry of psychopathology begin with a fundamental question. Why do some symptoms tend to occur together? Answers often involve formulation of diagnostic constructs designed to impose order on the complexity of psychological suffering.

There are two common ways to formulate the relation between indicators and constructs (Schmittmann et al., 2013). The first is the *formative* model exemplified by the construct of socioeconomic status (SES). Most sociologists construe SES as a composite variable comprising three indicators: income, educational level, and occupation status. These indicators determine—indeed, define—one's SES. SES does not exist apart from the indicators that form it. Accordingly, one can experience an increase in SES after receiving a pay raise, but not vice versa.

The second important model is the *reflective* model presupposed in most theories of psychopathology (cf. Borsboom & Cramer, 2014) and personality (cf. Cramer, van der Sluis, et al., 2012). According to this model, symptoms reflect the presence of an unobserved, latent entity that causes their emergence and explains why they cohere as a syndrome (Borsboom et al., 2003). The reflective model justifies the endeavor to identify the underlying disease entity that produces the symptoms of PTSD, depression, and so forth. This latent variable, whether categorical or dimensional, is the presumptive common cause of the symptoms that reflect its presence. This approach comports well with philosophical realism (e.g., Putnam, 1984), whereby one can interpret the successive revisions of the *DSM* as increasingly more accurate attempts to describe the reality of mental disorders, as exemplified by the metaphor of carving nature at its joints.

The latent variable approach continues to flourish as clinical scientists aim to make sense of the widespread comorbidity among the supposedly discrete clinical entities in *DSM-5* (APA, 2013). Hence, discussing mood and anxiety disorders, Barlow, Sauer-Zavala, Carl, Bullis, and Ellard (2014) posit that current diagnostic categories reify trivial differences while missing the underlying latent reality producing such superficial symptomatic diversity. They argue that trait neuroticism drives the development of these syndromes, and explains their common co-occurrence. Barlow et al. (2014) offer this higher-order latent factor as a more suitable target for clinical intervention than its "symptom-level manifestations" (Barlow et al., 2014, p. 344). Caspi et al. (2014) broadened this endeavor even further to explain the *entire* domain of psychopathology by invoking a "General Psychopathology dimension" they call the "p factor" (p. 119), likening it to the *g* factor of general intelligence.

There are major limitations to these latent variable approaches to psychopathology. First, it seems implausible that a single factor could "explain" the diversity of phenomena falling under the rubric of mental disorder. Indeed, as Jerome Kagan wryly observed, "One could say that all physical diseases are due to one factor called 'Unwell'" (personal communication, March 5, 2014).

Second, constructs, such as neuroticism or the p factor, which describe differences *among* people, are not necessarily isomorphic with causal mechanisms that operate *within* people (Borsboom & Dolan, 2006; Borsboom, Kievit, Cervone, & Hood, 2009). To be sure, causal mechanisms within an individual somehow produce his or her scores on measures of personality, intelligence, and psychopathology, but constructs developed to describe individual differences need not have referential meaning within the individuals involved. That is, they often fail to satisfy the theorems of ergodicity (Molenaar & Campbell, 2009). Therefore, just as heritability in genetics emerges only at the level of the population, not the person (Lewontin, 1974), interindividual traits, such as neuroticism, may have no specific referent at the level of the person.

Third, the symptoms of mental disorders frequently violate the axiom of local independence integral to latent variable approaches (Borsboom, 2008; Borsboom et al., 2009). To say that an underlying, latent disorder is the common cause of its symptoms, covariance among the symptoms must not arise from any interactions among them; the symptoms must be locally independent. For example, assume that we bring three thermometers inside on a cold winter day (Schmittmann et al., 2013). Within a few minutes we will notice that the mercury within each of them has risen to 72 degrees. If we were to control statistically for ("conditionalize on") room temperature, the mercury readings would no longer correlate, confirming their local independence. To demonstrate their local independence of one another, we can place an ice cube against one of the thermometers. Its mercury reading will plummet while the values of the other two thermometers will remain unchanged.

The axiom of local independence, understood as the absence of direct relations between symptoms conditional on their common cause, is implausible in psychopathology (Borsboom & Cramer, 2014). Consider the symptoms of depression. Rather than being locally independent symptomatic indicators of an underlying disease entity, rumination, insomnia, fatigue, and concentration

impairment seem causally interconnected. People who ruminate are likely to experience insomnia as a result, thereby experiencing fatigue the following day, making it hard for them to concentrate at work. Thus, instead of being passive receptors of the causal influence of a common latent variable, symptoms are potent causal agents that almost certainly affect each other. The same holds for symptoms of PTSD. Encounters with reminders of trauma can trigger intense psychological distress and physiological reactions (e.g., Shin et al., 1999), and these, in turn, can motivate avoidance behavior (e.g., Keane, Zimering, & Caddell, 1985). Rather than being locally independent, symptoms exist in a network of dense causal associations, such that a latent variable account of them collapses under the weight of its own implausibility.

Note that matters often differ for many nonpsychiatric medical conditions (Borsboom & Cramer, 2014). Consider a person who chronically spits up blood, coughs, and experiences chest pain. A biopsy reveals a malignant lung tumor that is the common cause of these symptoms, explaining their covariance. Not only does the axiom of local independence hold, but the underlying common cause has a physical referent that is independent of its symptomatic indicators. Indeed, a person can have a malignant tumor and not yet experience any symptoms. But this cannot happen with most mental disorders. For example, it makes no sense to say that a person who sincerely denies having any symptoms of PTSD suffers from PTSD, whereas we can diagnose someone with a "silent" malignant tumor even if that person is asymptomatic.

PTSD as a Causal System

Mental disorders are best construed as causal systems embodied in networks of functionally interconnected symptoms. Accordingly, the relation between symptoms and diagnosis is mereological—part to whole—rather than causal (Borsboom & Cramer, 2014). Likewise, the relation between the 50 states and the United States is mereological: parts to whole. The states are not caused by or reflective of an underlying entity called the United States; they are constitutive of it.

Moreover, the network perspective does not imply a formative model exemplified by constructs such as SES. Networks are empirically discovered, not formed by theorists who construct them to suit certain purposes. Indeed, the causal system perspective is ontologically realist as it presupposes mind-independent phenomena discoverable via network analyses. This realist commitment, however, does not require the postulation of an underlying, latent essence that mysteriously causes the emergence of symptoms. Accordingly, networks provide an alternative to the essentialism and social constructivism that have dominated the debate regarding PTSD's

ontological status. That is, the essentialist view of PTSD holds that an underlying categorical or dimensional latent entity is the common cause of the covariance among PTSD symptoms. The social constructionist view explains symptom covariance by appeal to cultural and historical forces that shape the phenomenology of the syndrome. The network approach is just as realist as the latent variable approach, but it locates causality among the symptoms themselves, whereas the latent variable approach holds that the underlying essence is the common cause of symptom emergence and coherence.

Networks depicting psychiatric disorders consist of *nodes*, representing symptoms, and *edges*, representing the relations between pairs of symptoms. An episode of disorder unfolds over time as nodes turn on, transmitting activation to connected nodes, and settling into a pathological equilibrium. Apart from offering a plausible account of the relation between symptoms and disorders in psychopathology, network approaches also suggest the application of novel, data-analytic techniques that have been developed in the area of complex network analysis (Borsboom & Cramer, 2014). These techniques allow the researcher to determine the network structure of psychopathology symptoms from empirical data.

To illustrate how the network perspective can guide both data analysis and substantive theorizing, we present network analyses of PTSD symptoms reported by adult survivors of the massive earthquake the occurred on May 12, 2008, in Wenchuan county, Sichuan province in southwest China. Registering 8.0 on the Richter scale, the earthquake killed 69,227 people, injured 374,643, and rendered about 4.8 million homeless; another 17,923 people remain missing (Wang, Zhang, Wang, et al., 2009).

Method

Participants

The participants were 362 Chinese adults (women = 266, men = 96) who survived the Wenchuan earthquake, and who had lost at least one child in the disaster. A large minority had been injured (38.1%), had been temporarily buried under rubble (33.4%), and helped rescue other victims (41.4%). At the time of data collection, most were married (84.5%), and their mean age was 44.8 years old ($SD = 10.9$).

The participants were recruited from a large cohort of earthquake survivors enrolled in a clinical research program conducted by Li Wang and his colleagues at the Institute of Psychology, Chinese Academy of Sciences in Beijing (e.g., Cao et al., 2013; Wang, Cao, Wang, Zhang, & Li, 2012; Wang, Zhang, Shi, & Wang, 2009; Zhang, Shi, Wang, & Liu, 2012). Participants were contacted by phone and asked whether they would be interested in

completing a battery of questionnaires related to mental health. Those expressing an interest underwent a brief phone screen to determine their eligibility. Participants had to be between 18 and 75 years of age, to have personally experienced the earthquake, to have lost a child during the disaster, and to understand the purpose and meaning of the questionnaires. Eligible participants completed the questionnaires during large group testing sessions at the regional mental health center in Wenchuan County, funded by the Institute of Psychology.

Due to the limited local job opportunities after the earthquake, many men left the region to seek work elsewhere to enable them to support their families. Accordingly, more women than men were available to enroll in our study. Although we did not track the number of people who were contacted, the vast majority of those contacted and assessed as eligible participated.

After completing the questionnaires, they were invited to ask further questions and discuss any concerns with the research team. They received an honorarium for participating, worth approximately $10. Data collection occurred between November 15 and November 30, 2013, approximately 5 years and 6 months after the earthquake. Both Harvard University's Committee on the Use of Human Subjects and the corresponding ethics committee of the Institute of Psychology, Chinese Academy of Sciences approved the materials, protocol, and consent procedures.

In this article, we report network analyses on PTSD symptoms reported by participants on the Mandarin Chinese version of the Posttraumatic Checklist–Civilian version (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1993). The translation, back-translation, and psychometric work (reliability, validity) were done by the team at the Institute of Psychology (Li et al., 2010). This widely used questionnaire (Terhakopian, Sinaii, Engel, Schnurr, & Hoge, 2008) comprises 17 items, each assessing one of the symptoms of PTSD, according to *DSM-IV* (APA, 1994). Participants rate each item on a scale ranging from 1 (*not at all*) to 5 (*extremely*) to indicate how seriously they have been bothered by the symptom during the past month. Hence, total scores can range from 17 to 85. In our study, participants rated symptoms in reference to their experience of the earthquake.

Although our interest was in investigating the network structure of PTSD among earthquake survivors, we did use standard stringent criteria for diagnosing probable PTSD via the PCL-C. That is, we identified participants as probable PTSD cases who scored 3 (*moderately*) or higher on at least one B (reexperiencing) symptom, at least three C (avoidance and numbing) symptoms, and at least two D (arousal) symptoms, and whose total score was 44 or higher. Li et al. (2010) found that 44 was the optimal score for identifying earthquake survivors as qualifying for PTSD as determined by structured clinical interview. These investigators found that this cutoff score was associated with excellent sensitivity (.83), specificity (.97), positive predictive power (.92), and negative predictive power (.94).

Data analysis

Using the R package qgraph (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012), we computed several networks. Each node in the network represented one of the 17 PTSD symptoms measured by the PCL-C. Each edge represented the strength of association between two of the symptoms.

Association networks. We first computed an *association network*, in which each edge represented the zero-order correlation between two symptoms. Thicker edges between symptoms denote larger correlations. This network is weighted, but not directed. That is, it represents the magnitude of the correlation between symptoms, but not its direction. The qgraph package implements Fruchterman and Reingold's (1991) algorithm, which positions nodes with stronger correlations near the center of the network and those with weaker correlations near the periphery of the network. Constructing a network of zero-order correlations is a first step toward characterizing a causal system. As densely interconnected networks can appear complex, we computed a second association network whereby only edges depicting inter-node correlations of at least $r = .30$ appeared in the graph. Correlations between symptoms having magnitudes less than .30 were suppressed.

Concentration and relative importance networks. Although association networks provide an initial approximation of the causal structure of a network, a correlation between symptoms can occur in multiple ways (Borsboom & Cramer, 2014). The correlation may arise because activation of one symptom causes the second symptom. In depression, for example, insomnia can cause fatigue. A second possibility is that the correlation between two symptoms may arise due to their shared association with a third symptom. A third possibility is that a third variable causes both symptoms, thereby explaining their correlation.

Elucidating causal structure in a network requires that one identify relations between symptoms that reflect causal associations, not mere correlations spuriously arising via shared association with another symptom. To that end, we calculated two additional types of networks. In the first, known as a *concentration network*, an edge depicts the correlation between two symptoms after one has controlled statistically for all other symptoms in the

network. In other words, each edge represents a partial correlation.

In the second type of network, referred to as a *relative importance network*, each edge represents the relative importance of a symptom as a predictor of another symptom (Johnson & LeBreton, 2004). Relative importance reflects both the direct effect of symptom X on Y and the effect of symptom X on symptom Y after one has adjusted for all other symptoms in the network. Relative importance networks are both weighted and directed. That is, the graph illustrates the magnitude of the relation and the direction of prediction, with arrows originating in the predictor symptom and terminating in the predicted symptom. To calculate relative importance, we used the *lmg* metric in the R package relaimpo (Grömping, 2006).

Measures of node centrality. To quantify the importance of each of the 17 symptoms to the PTSD network, we calculated three indices of *centrality*: strength, closeness, and betweenness. The *degree* of a node is the number of edges connected to it. In a weighted network, one can also report *strength* of a node by summing the weights (i.e., correlation magnitudes) of each edge linked to the node.

The *closeness* of a node reflects the average distance from that node to all other nodes in the network. Closeness is the inverse of *farness* (i.e., the mean shortest weighted path length between a given node and all other nodes in the network). Accordingly, a high closeness score indicates a short average distance between a given node and the remaining nodes in the network.

Finally, we computed the *betweenness* index for each node. To ascertain the betweenness of a node, one first calculates the shortest path length between each pair of nodes in the network. The betweenness of a node equals the number of times that node lies on the shortest path between two other nodes. If the shortest path between node X and node Y has the connecting edge passing through node Z, then node Z has (at least) a betweenness of one. If node Z lies on the shortest path between nodes E and F, then node Z has a betweenness of two, and so forth.

For all measures of centrality, higher values reflect a node's greater centrality to the network. We ran the R package qgraph (Epskamp et al., 2012) to calculate each of the three measures of centrality. We created centrality plots that depict these values.

Results

The mean PCL-C total score for the 362 participants was 45.3 (*SD* = 14.5). As for probable PTSD, 38% (*n* = 139) met stringent criteria, whereas 4% (*n* = 13) met symptomatic criteria for the disorder, but their total PCL-C score

fell short of 44. The remaining 210 participants did not qualify for probable PTSD. (A 363rd participant chose not to answer more than half of the PCL-C items, compelling us to exclude this person's data from all analyses.)

In the figures depicting the networks and centrality plots, we use the following abbreviations to designate the 17 *DSM-IV* PTSD symptoms from the PCL-C. Cluster B includes (a) *intrusion* = intrusive memories, thoughts, or images of the trauma, (b) *dreams* = traumatic dreams, (c) *flash* = flashbacks, (d) *upset* = feeling upset in response to reminders of trauma, (e) *physior* = physiological reactivity to reminders of the trauma; Cluster C includes (f) *avoidth* = avoidance of thoughts or feelings about the trauma, (g) *avoidact* = avoidance of activities or situations reminiscent of the trauma, (h) *amnesia* = having trouble remembering parts of the traumatic experience, (i) *lossint* = loss of interest in previously enjoyed activities, (j) *distant* = feeling distant or cut off from people, (k) *numb* = feeling emotionally numb, (l) *future* = feeling that your future will be cut short; Cluster D includes (m) *sleep* = difficulty falling or staying asleep, (n) *anger* = feeling irritable or having angry outbursts, (o) *concen* = difficulty concentrating, (p) *hyper* = hypervigilant or watchful or super alert, and (q) *startle* = feeling easily startled or jumpy.

As evident from the association network (Fig. S1 in the Supplemental Material available online) and the centrality plots (Fig. 1), PTSD symptoms in earthquake survivors are densely interconnected. The strength of each symptom is at least six, and the betweenness value is zero for each of them, meaning that the shortest path between each pair of symptoms is the direct association between those symptoms. Symptoms having high centrality include hypervigilance, concentration impairment, physiological reactivity to reminders of the trauma, sleep disturbance, and flashbacks.

The strongest associations are especially apparent when we exclude correlations falling below *r* = .30 (Fig. 2). Several features are immediately apparent. Especially thick edges occur between hypervigilance and startle, between avoidance of thoughts about the trauma and avoiding activities associated with trauma, and between loss of interest in formerly enjoyed activities and feeling distant from other people. Also, there are strong associations among dreams about the trauma, flashbacks, and intrusive memories of the trauma. These associations conform to clinical observations as embodied in the *DSM-IV* clusters of hyperarousal, avoidance/numbing, and reexperiencing clusters, respectively. In addition, several other strong associations less obvious to clinicians include those between startle reactions and concentration problems, and between concentration problems and anger. These are suggestive of possible causal links in the system.
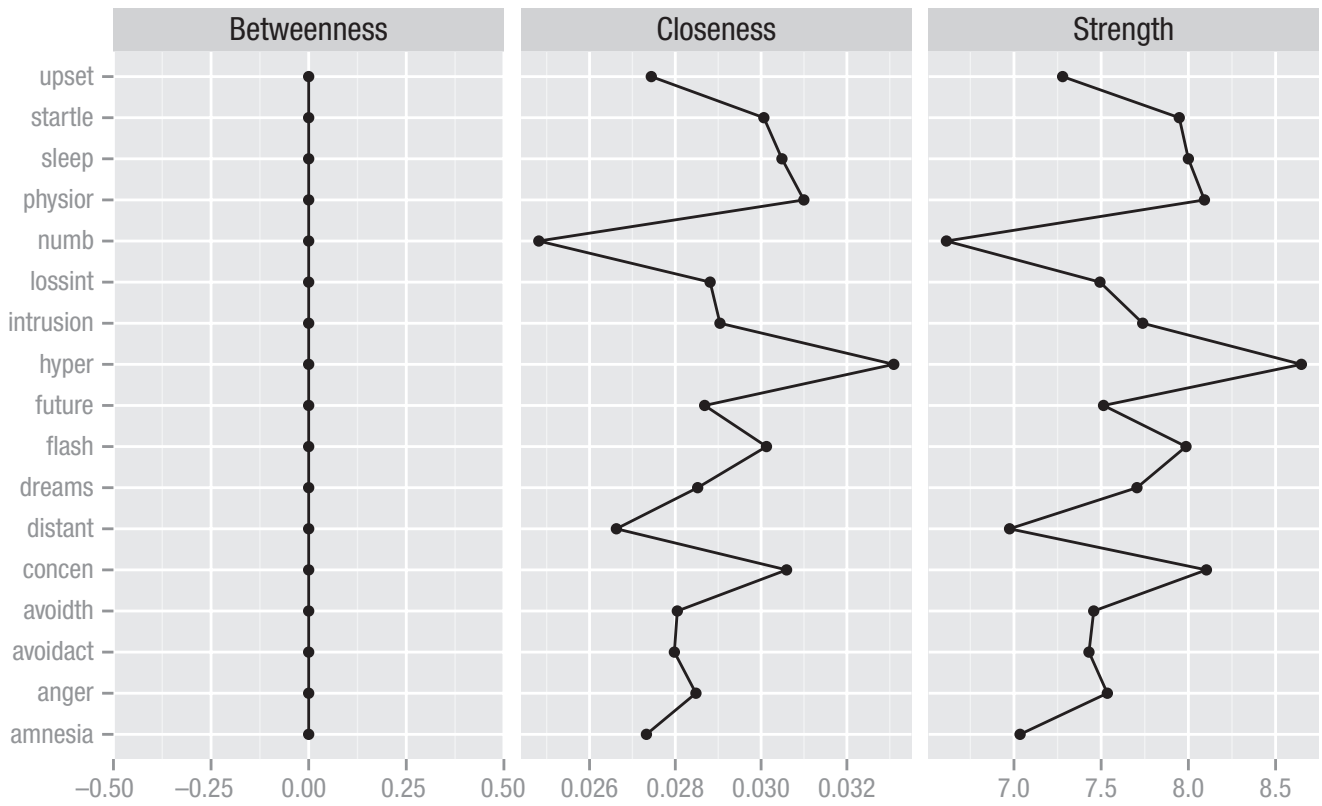
Fig. 1. Centrality plot for the association network depicting the betweenness, closeness, and strength of each node.
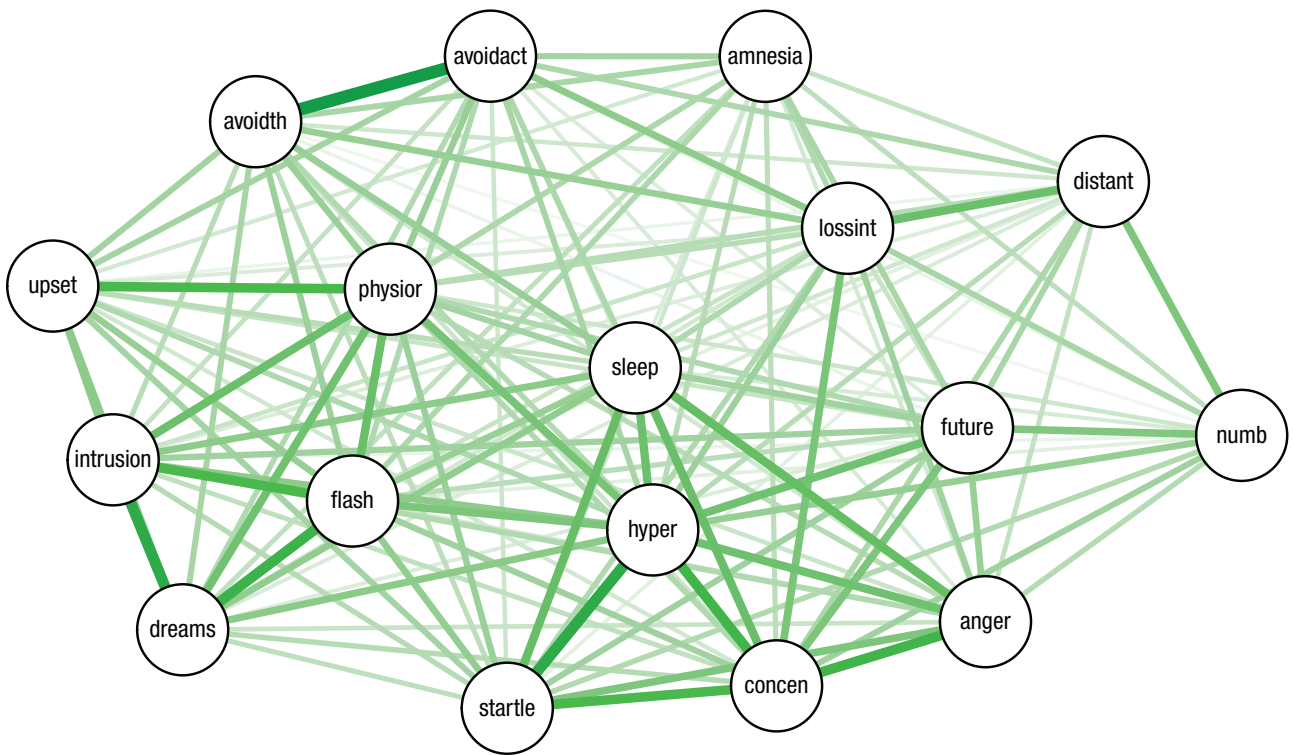

Fig. 2. Association network depicting PTSD symptoms. Each node represents a symptom, and each edge represents the zero-order correlations between two symptoms where $r \geq .30$. The thickness of an edge reflects the magnitude of the association.
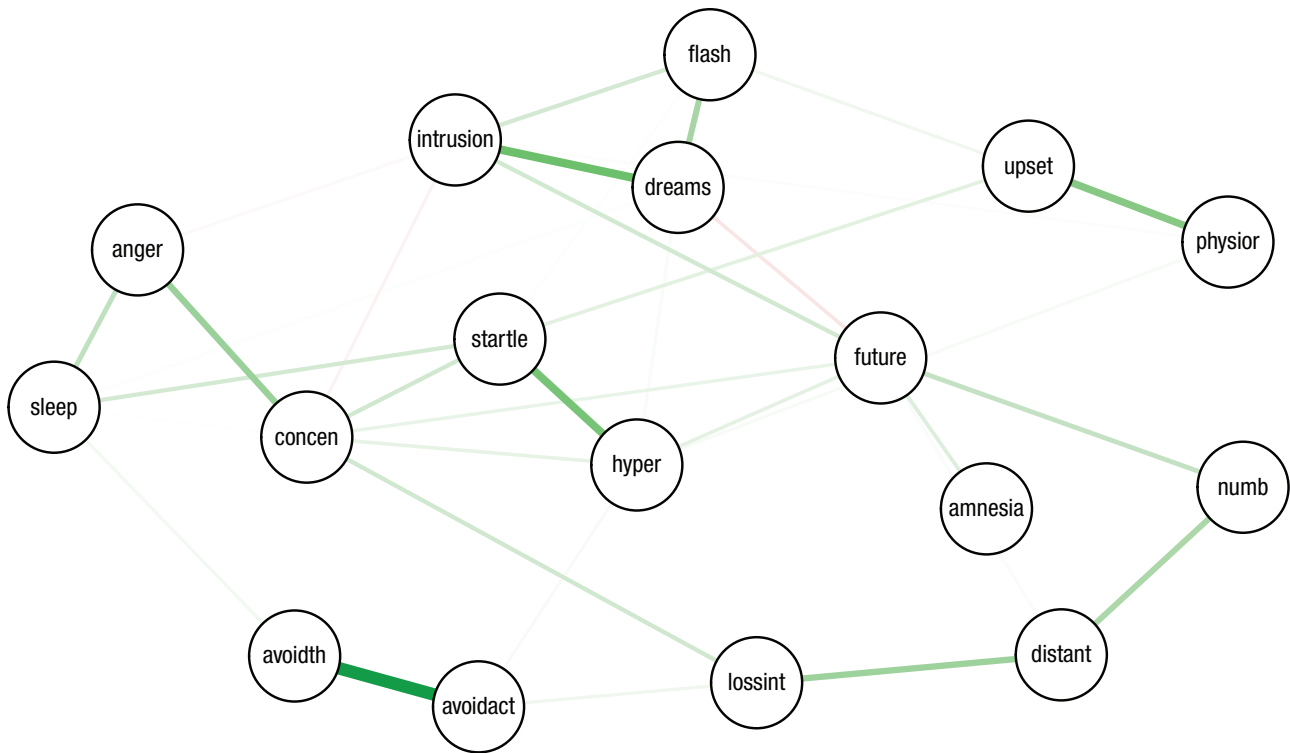
Fig. 3. Concentration network depicting PTSD symptoms. Each node represents a symptom, and each edge represents the partial correlation between two symptoms. The thickness of an edge reflects the magnitude of the association.

Supporting this interpretation are findings from the concentration network in which these relations are especially pronounced (Fig. 3). This network depicts the associations remaining between symptoms after we control for all other symptoms in the network (i.e., partial correlations). Pronounced edges remain between avoidance of thoughts about the trauma and avoidance of activities reminiscent of it. Clusters of associations remain among anger, sleep problems, and concentration impairment, and among intrusive thoughts, dreams, and flashbacks. However, the other two reexperiencing symptoms, physiological reactivity in response to reminders of the trauma and feeling upset upon encountering these reminders, have essentially no connection to the other reexperiencing symptoms although the latter two are strongly interconnected. Feeling distant from other people is strongly linked to emotional numbing and to loss of interest in previously enjoyed activities. Finally, hypervigilance and startle are strongly linked.

The centrality plots for the concentration network appear in Figure 4. They affirm the importance of hypervigilance, concentration impairments, and dreams about the trauma. However, future foreshortening now emerges as a highly central symptom.

The directed, relative importance network appears in Figure S2 (in the Supplemental Material available online).

Many associations apparent in the association network remain in the relative importance network. Surprising links, such as between concentration problems and anger, remain, as do the ones between hypervigilance and startle, and between emotional numbing and feeling distant from others.

Discussion

Collectively, our network analyses provide cues to the causal system constitutive of PTSD, at least among people exposed to a catastrophic natural disaster. Some findings comport well with clinical observations of PTSD patients, as embodied in the *DSM-IV* symptom clusters, whereas other findings point to unexpected potentially causal interconnections. We summarize key findings and their theoretical implications.

First, hypervigilance emerged as a highly central symptom. Many theorists distinguish PTSD from other anxiety disorders by emphasizing that PTSD concerns memory for past threats, whereas other anxiety disorders concern future threats (e.g., Brewin, 2003; McNally, 2003; Rubin, Berntsen, & Bohni, 2008). Yet the central prominence of hypervigilance suggests an emphasis on PTSD as a syndrome marked by a sense of continual, impending threat, as Ehlers and Clark (2000) have stressed.
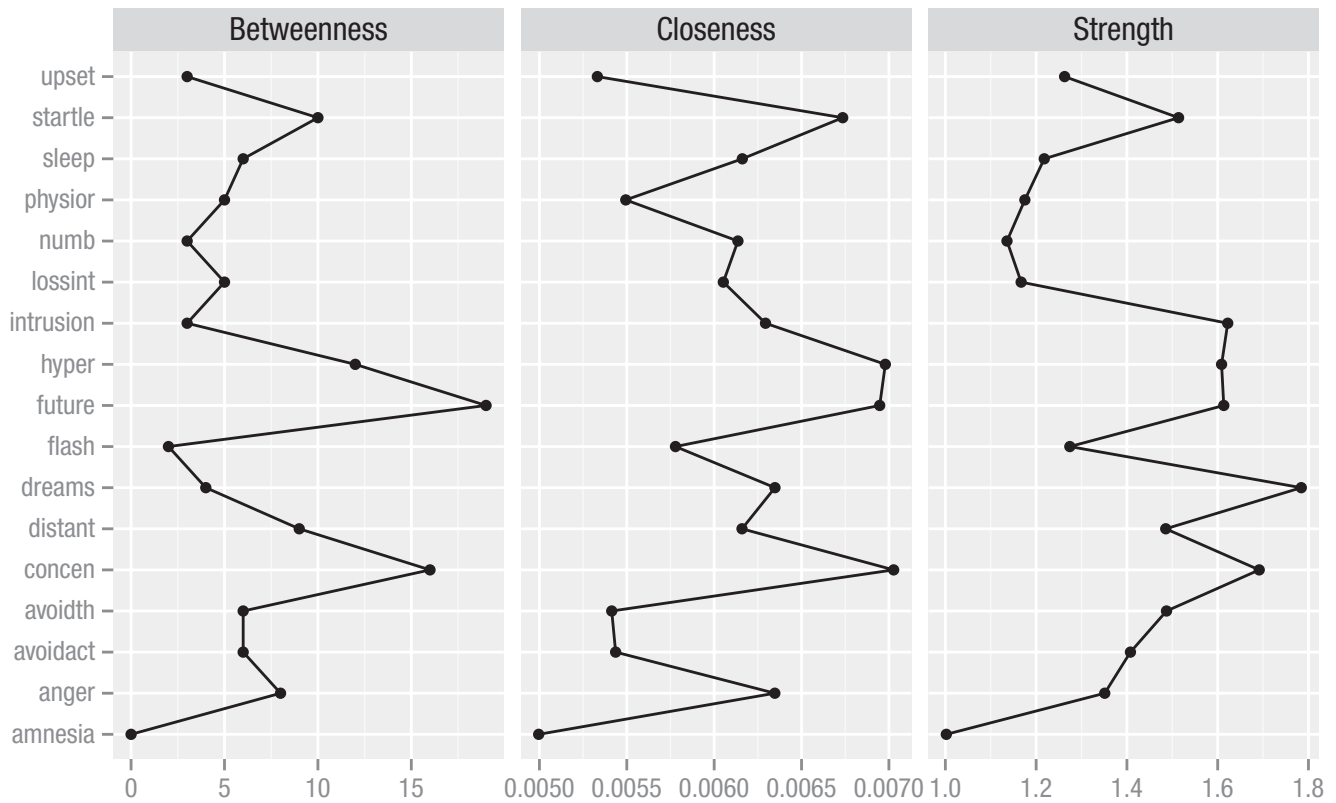
Fig. 4. Centrality plot for the concentration (partial correlation) network depicting the betweenness, closeness, and strength of each node.

Second, future foreshortening also emerged as a symptom of high importance to the network. In contrast to hypervigilance, which exhibited the highest strength of any symptom in the association network, future foreshortening did not exhibit especially high strength (see Fig. 2). However, in the concentration network, it emerged as a highly central symptom, with the highest betweenness of any symptom (see Fig. 5). These results suggest that the importance of the future foreshortening symptom is derived largely from its position in the network rather than the strength of its associations. As depicted in Figure 4, future foreshortening appeared to connect the hypervigilance and intrusive memory symptoms with emotional numbness, and in turn, with feelings of social disconnection and anhedonia. Accordingly, future foreshortening may figure prominently in the structure of PTSD, bridging symptoms related to fear and intrusive memories with those related to emotional numbness and anhedonia.

Third, although the relative importance network concerns the direction of prediction and not necessarily causal relations, it suggests the possibility of bidirectional relations between symptoms directly connected by edges in the network. For example, this pattern suggests that

hypervigilant people may be prone to startle, and that startling may foster continued hypervigilance, for example. Any causal bidirectionality would likely maintain the syndrome, leading to chronicity.

Fourth, the connections between anger/irritability and sleep, and anger/irritability and concentration problems suggest the possibility that sleep problems may render it difficult to control one's irritability in everyday life. Anger problems and concentration difficulties may arise from sleep-related limitations in executive resources, thereby impairing the regulation of both emotions and attention. These findings illustrate how network analyses may disclose relations among symptoms that are not immediately obvious.

Fifth, consistent with the *DSM-IV* symptom clusters, edges among intrusive thoughts, flashbacks, and dreams about the trauma remained in the concentration network. Yet the other two reexperiencing symptoms of being upset at reminders of the trauma and reacting to them physiologically did not. Likewise, feeling distant from others remained linked to loss of interest in previously enjoyed activities and emotional numbing, whereas avoidance of thoughts and activities reminiscent of the trauma did not.

Advantages of the causal system perspective

Network analyses now span fields from physics (e.g., Reichardt & Bornholdt, 2006) to sociology (e.g., Cacioppo, Fowler, & Christakis, 2009), but only recently have empirical studies on psychopathology appeared, featuring the syndromes of major depression (Cramer, Borsboom, Aggen, & Kendler, 2012; van de Leemput et al., 2014; see also Fried et al., 2014) and persistent complex bereavement disorder (Robinaugh, LeBlanc, Vuletich, & McNally, 2014). Adding to this body of work, our article illustrates how the approach can illuminate PTSD as a causal system (McNally, 2012).

What advantages does the causal systems perspective provide? First, it directs empirical efforts toward elucidating the causal relations among symptoms as they unfold over time, and directs them away from chimerical latent entities. Indeed, it remains mysterious *how* a latent entity could produce symptoms when its very existential status is uncertain. That is, it is unclear how abstract latent entities, defined via between-subject analyses, become embodied within at the level of the person as causal drivers of symptoms. The causal systems approach remains firmly in the camp of philosophical realism, but without the conceptual and psychometric burdens of latent variable modeling.

Second, it identifies symptoms having high centrality in a causal system, thereby pointing to urgent targets for clinical intervention. By turning off an activated symptom that has many outgoing edges, one can foster a therapeutic cascade of downstream benefits, deactivating other symptoms as its effects propagate throughout the network. For example, we found that sleep difficulty had high centrality in earthquake survivors even though it has never been deemed a core, defining feature of PTSD. Accordingly, clinicians may aim to stabilize sleep in patients with PTSD before initiating exposure therapy to deactivate nodes implicated in intrusive experiencing symptoms. Likewise, cognitive-behavior therapists may endeavor to help patients distinguish the present context from the ones associated with the trauma. Success in doing so may deactivate hypervigilance, thereby producing a therapeutic cascade as linked symptoms deactivate in turn.

Third, it may identify harbingers of relapse in recovered patients, thereby enabling rapid early intervention to prevent a full-blown episode from erupting (van de Leemput et al., 2014). For example, recovery is often incomplete following treatment, and clinical concern about residual symptoms may vary as a function of symptom centrality. Isolated symptoms having low centrality may be less likely to require restarting treatment, whereas

those having high centrality may call for treatment, thereby preventing a full-blown relapse.

Fourth, network analyses can identify bridge symptoms shared by disorders, thereby solving the comorbidity problem (Cramer et al., 2010) that has vexed our field. Psychopathologists have often sought to purify discrete diagnostic constructs by identifying symptoms specific to certain disorders, viewing these as more diagnostically important than nonspecific ones shared by multiple disorders (e.g., for PTSD, see Spitzer, First, & Wakefield, 2007). They have regarded shared symptoms as an obfuscatory nuisance. By contrast, the causal systems perspective recognizes them as bridges connecting nominally discrete syndromes, and thus especially important. Activation spreading from a bridge symptom can propagate to related networks, leading to the emergence of several syndromes in the same person (Cramer et al., 2010). For example, in their network analyses, Robinaugh et al. (2014) found that loneliness exhibited strong associations with both the network of persistent complex bereavement disorder symptoms and the network of depression symptoms, suggesting that it may be one mechanism contributing to the high prevalence of depression in those with persistent complex bereavement disorder.

Fifth, most paradigms in science are adrift in a sea of anomalies, yet researchers seldom abandon a paradigm unless a persuasive alternative is available, as Kuhn (1970, p. 77) observed many years ago. We now may have arrived at this point in the field of psychopathology. In fact, a recent director of the National Institute of Mental Health wondered whether *DSM* diagnoses are mere reifications, not labels for genuine medical conditions (Hyman, 2010). And until recently (Cramer et al., 2010), the comorbidity problem has resisted a solution, and every clinician, upon learning about the axiom of local independence, will realize that it cannot be true for mental disorder. Symptoms are deeply intertwined causally. Suffice it to say, the causal systems approach may furnish a new paradigm for conceptualizing mental disorders.

Many psychopathologists favor a transdiagnostic perspective as a new paradigm for conceptualizing mental disorders (Insel et al., 2010). This approach can mesh well with the network approach (e.g., Cramer et al., 2010), depending on how one conceptualizes the variables that cut across diagnostic categories. For example, genomic studies that identify allelic variants common to schizophrenia and bipolar disorder (for a review, see Gratten, Wray, Keller, & Visscher, 2014) are wholly compatible with network construals of mental disorder, whereas transdiagnostic variables, such as the p factor, are not. In the genomic case, allelic variants have clear, direct physical referents within subjects, whereas this is not true for latent variables, such as the p factor.

Despite these manifest advantages, skeptics may wonder whether network approaches provide anything really new. Clinicians may roll their eyes when network analyses reveal that hypervigilance is tightly linked with exaggerated startle response in PTSD. Surely we knew this already, they may remark. Yet confirming well-established knowledge inspires confidence in the findings of network analyses. Moreover, network analysis does more than confirm the obvious; it computes the strength of associations between symptoms, uncovers connections that have eluded clinical observation (e.g., between anger and concentration problems), and identifies those symptoms most central to a disorder. Hence, the computational power of network analyses provides tools hitherto unavailable to even the most seasoned clinicians.

Readers familiar with the history of PTSD may ask whether network theorists are merely reinventing a conceptual wheel fashioned already by scholars such as Horowitz (1986) and Young (1995, 2004). Others acquainted with the philosophy of biology may ask whether the causal system perspective merely reinvents Boyd's (1991) homeostatic property cluster kinds that clinicians have celebrated as a nonessentialist way of framing mental disorders as natural kinds (Kendler et al., 2011; McNally, 2011, pp. 203–208). There is merit in these questions. In fact, Horowitz did postulate an oscillatory, mutually reinforcing process between reexperiencing symptoms of PTSD and those of emotional numbing and avoidance, and Young (2004) has elucidated the "inner logic" (p. 128) of PTSD, implying causal interactions among symptoms, not local independence among them. And although Boyd's concept concerned a nonessentialist interpretation of *species* as a cluster of properties that coheres despite environmental perturbations, thereby evincing homeostasis, its relevance to disorders as networks holds. Yet the causal systems perspective brings to bear powerful computational methods for deepening our understanding of psychopathology, thereby building on the conceptual insights of these scholars.

Others may wonder how network analysts can hope to extract causal inference from mere correlational, cross-sectional data. To be sure, controlled experiments provide the gold standard for supporting causal inference in science. However, progressively sophisticated network methods, combined with data from other sources, can support a causal abductive inference or inference to the best explanation (Harman, 1965). Indeed, specialists in network analysis continue to develop ever more sophisticated computational methods that promise to strengthen our causal inferences about the causes of mental disorders (van Borkulo et al., 2014).

Limitations and future directions

Limitations in our study point to future directions in network research on PTSD and other mental disorders. First, the claim that mental disorders are causal systems is a plausible theoretical conjecture resting on the implausibility of the axiom of local independence. However, it is one thing to assert that disorders are causal systems and another thing to confirm the precise causal relations among the symptoms themselves. Indeed, with cross-sectional correlational data, the best that one can do is to eliminate spurious candidates for causal relations, as we did via computation of concentration networks. Longitudinal data consisting of repeated symptom assessments over time would clarify the temporal priority of symptoms so vital to confirming causal relations (Bringmann et al., 2013). Such studies are a high priority for future research.

Second, we elucidated the network structure in adult Chinese survivors of a massive earthquake. It is unclear whether the same networks would emerge in victims of other traumatic events. Although the original rationale for the PTSD diagnosis was that diverse traumatic events can produce the same syndrome, there are numerous ways to qualify for the diagnosis. Indeed, Galatzer-Levy and Bryant (2013) calculated that there are 79,794 distinct combinations of *DSM-IV* symptoms that enable one to qualify for the PTSD diagnosis. Matters become more complicated in *DSM-5* as there are now 636,120 ways to qualify for the disorder (Galatzer-Levy & Bryant, 2013). To be sure, not all these symptom combinations are equally likely to occur in people exposed to trauma. Nevertheless, these findings suggest that there may be multiple network structures for PTSD.

Third, our network analyses rest solely on questionnaire data rather than on symptoms reported in clinical interviews or on other sources of information concerning symptoms (e.g., physiological; Shin et al., 1999). Accordingly, our inferences about the network structure of PTSD must be qualified as pertaining to adult Chinese survivors of a natural disaster who reported their symptoms on a standardized questionnaire. Network studies involving sources of data other than self-report are warranted.

In conclusion, conceptualizing mental disorders as causal systems provides an alternative to the traditional categorical and dimensional models. To illustrate the approach, we presented network analyses of PTSD symptoms in adult survivors of a major earthquake that provide clues to the causal system of PTSD.

Author Contributions

R. J. McNally wrote the first draft, and R. J. McNally, D. J. Robinaugh, G. W. Y. Wu, and D. Borsboom wrote subsequent

drafts. R. J. McNally, D. J. Robinaugh, and M. K. Deserno ran the data analyses and created the figures, and L. Wang translated the instruments and secured funding for the clinical research center where the data were collected. G. W. Y. Wu and L. Wang supervised data collection, and D. Borsboom pioneered the network approach to psychopathology.

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