Empirical Article

Clinical Psychological Science
2019, Vol. 7(6) 1–15

DOI: 10.1177/2167702619859333
www.psychologicalscience.org/CPS

Posttraumatic Stress Disorder Near and Far: Symptom Networks From 2 to 12 Months After the Virginia Tech Campus Shootings

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Abstract

Posttraumatic stress disorder (PTSD) is unique in its longitudinal focus. To better understand how PTSD develops, we used network analysis in a longitudinal sample of survivors of the 2008 Virginia Tech campus shootings. Participants were 212 women who completed surveys at both 2 and 12 months after the shooting. Using within-group permutation tests, we found that overall network strength significantly increased and overall network structure significantly changed. Several symptoms saw marked alterations in their network centrality and relations to other symptoms. Psychological reactivity at reminders was the most central symptom at 2 months but among the least central at 12 months. By contrast, reliving, anhedonia, and physiological reactivity had low centrality at 2 months but high centrality at 12 months. Findings broadly support memory-based and fear-conditioning accounts of PTSD and suggest that automatic situationally cued symptoms, including reliving, thought avoidance, and physiological reactions, become more central to the network over time.

Keywords

network analysis, posttraumatic stress disorder, longitudinal, fear conditioning, open materials

Received 12/19/18; Revision accepted 4/28/19

Why do some people go on to experience persistent and often disabling posttraumatic stress disorder symptoms (PTSD) after acute adversity? Although research has illuminated many risk factors of PTSD (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003), it has largely ignored the distinctive contribution of individual symptoms to the development of PTSD. Indeed, the traditional diagnostic understanding of PTSD treats symptoms as functionally equivalent and interchangeable manifestations of an underlying disorder (Friedman, Resick, Bryant, & Brewin, 2011). Recent advances in network analysis have called these and other assumptions about psychopathology sharply into question (Borsboom & Cramer, 2013; McNally, 2016). From a network perspective, there are sound reasons to think that some symptoms may exert greater influence on the development of PTSD than others and that the role of symptoms changes over time. However, because prior network analyses of PTSD have largely relied on cross-sectional data involving only one time point, the nature of these changes has not been fully investigated. A better understanding of these changes over time may help refine theories of PTSD, improve risk assessment, and facilitate treatment recommendations. In this study, we used network analyses to explore these questions in a longitudinal sample of female participants exposed to

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the 2008 Virginia Tech campus shootings, one of the deadliest mass shootings in U.S. history.

Network Framework

A network approach proposes that the symptoms of PTSD and other mental disorders mutually influence one another in complex chains of interaction. In the case of PTSD, for example, if someone experiences psychological distress when reminded of a traumatic event, he or she may experience aversive physiological arousal at the same time, including a racing heartbeat and sweaty palms. In response, individuals may try to avoid those reminders, either behaviorally (e.g., avoiding the street where a car accident occurred) or by suppressing unwanted thoughts (e.g., avoiding thinking about the traumatic experience). These symptoms may have further consequences on functioning, such as sleep difficulties. Sleep difficulties may amplify other PTSD symptoms (Harvey, 2008), creating a positive feedback loop. From the network perspective, these symptoms are a causal system. They are not caused by a disorder; they are the disorder, mutually reinforcing one another in complex ways, activating, deactivating, and circling back in feedback loops.

The mutually interactive nature of symptoms has long been recognized and treated by clinicians. Accordingly, network analysis has been applied to a growing list of clinical syndromes, including depression (van Borkulo et al., 2015), social anxiety (Heeren & McNally, 2016), eating disorders (Levinson et al., 2017), and grief (Robinaugh, LeBlanc, Vuletich, & McNally, 2014). Moreover, network approaches have provided new insights into psychopathology (McNally, 2016), explaining the overlap in disorders (Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011), the prevalence of comorbidity (Cramer, Waldorp, van der Maas, & Borsboom, 2010), and the core symptoms in a given disorder (Robinaugh, Millner, & McNally, 2016).

Network Analysis: Promise and Limitations

Network theory proposes that symptoms causally interact, producing disorders. However, causal theories are extremely difficult to test, and causality can generally be established only through convergence of multiple types of analysis and theory. One useful type of analysis involves examining correlational structures between symptoms. Although this approach can only provide clues regarding the question of causality, it can test implications of network theory. In psychopathology network analyses, symptoms are described (and graphically depicted) as *nodes*; the associations between nodes are described as *edges*. When one symptom node shows strong positive associations to other symptom nodes in the network, that symptom is strongly associated with activation of the overall network. Researchers have examined networks between persons and within persons; both methods generated useful future research questions regarding network theory. However, it is important to note that network theory (i.e., symptoms causally interact) is not the same as a given network analysis (i.e., symptoms in a sample show a correlational structure).

Network Analysis and Theories of PTSD

A network theory of psychopathology necessarily implies a developmental progression in which the network of psychopathological nodes (or symptoms) is activated and maintained. For this reason, the symptoms of PTSD are unusually amenable to network analysis because they result from an exogenous cause, a traumatic event (American Psychiatric Association, 2013). One consequence of this etiology is that PTSD takes time to develop, as indicated by the requirement that 1 month must elapse before PTSD can be diagnosed (American Psychiatric Association, 2013). Virtually all theories of PTSD propose that symptoms arise and gather coherence over time either through the strengthening of a fear network (Foa & Rothbaum, 1998), appraisal processes (Ehlers & Clark, 2000), the increased availability of trauma memories (Rubin, Berntsen, & Bohni, 2008), or the inability to form a coherent trauma memory (Brewin, Dalgleish, & Joseph, 1996).

Given this explicit developmental component, the role of some PTSD symptoms in the coalescing network should change over time. However, the precise nature of these changes is unclear. Theoretical accounts of PTSD suggest a number of plausible developmental changes in the network. For example, in accounts that emphasize memory processes (Brewin et al., 1996; Rubin et al., 2008), intrusive involuntary memories and flashbacks should exert increasing influence on the network over time. By contrast, in accounts that emphasize the development of fear conditioning or emotional processing (Foa, Zinbarg, & Rothbaum, 1992), conditioned physiological reactions to reminders and avoidance should exert increasing influence on the network over time. Finally, in cognitive accounts that emphasize appraisal processes (Ehlers & Clark, 2000), psychological distress at reminders, which reflects higher order cognitive processes related to emotional upset, should exert increasing influence on the network over time as the person appraises the reminder more negatively (though psychological distress also has an automatic component that is not a result of conscious appraisal).

On the other hand, general distress symptoms, such as sleep difficulties, anhedonia, and feeling distant or cut off from others, should provide scaffolding for network activation generally and thus serve as maintaining factors, likely irrespective of time (Marshall, Schell, & Miles, 2010). Network analysis is uniquely positioned to examine these developmental changes and thus may help refine our theoretical accounts of PTSD and inform more targeted treatment, including early intervention and prevention.

Few prior network analyses of PTSD have examined these changes, usually because they have been based on a single snapshot of PTSD symptoms. In addition, prior research has often used samples exposed to heterogeneous types of trauma, which serves to introduce variation or noise (e.g., Armour, Fried, Deserno, Tsai, & Pietrzak, 2017; Fried et al., 2018; Mitchell et al., 2017). Although one prior network study focused on survivors of the 2008 Virginia Tech campus shootings (Sullivan, Smith, Lewis, & Jones, 2018), this study was cross-sectional, precluding an examination of network change. Equally important, the researchers' PTSD assessment, the 10-item Harvard Trauma Screening Questionnaire, omits key PTSD symptoms, including physiological reactions to reminders, reliving or flashbacks of the event, and lack of interest or anhedonia.

To our knowledge, the only prior PTSD network analysis with a longitudinal component is a study of people admitted to a trauma hospital for a serious injury (Bryant et al., 2017). This study found that associations among reexperiencing symptoms and overall network connectivity became stronger at 12 months compared with immediately after trauma, suggesting those symptoms progressively strengthen. Nevertheless, this study had limitations. The baseline assessment was conducted within 24 hr of admission (7 days on average after the injury), well before PTSD can be diagnosed. The participant sample also included widely varying traumatic injuries and a substantial proportion of individuals with a mild traumatic brain injury (43%).

Present Study

In the present study, we examined a normative sample of 212 female students exposed to the 2008 Virginia Tech campus tragedy, one of the deadliest civilian shootings in U.S. history. Participants were enrolled at Virginia Tech at the time of the shooting and were assessed at 2 and 12 months after the shootings, allowing us to examine how their PTSD symptom networks evolved over time. The normative sample allowed us to capture the full range of PTSD symptoms, which is advantageous for identifying network relationships, and it also allowed us to assess how PTSD symptoms develop in a nonclinical sample. In previous network studies of PTSD, the traumatic events were often highly heterogeneous, varying in their duration, severity, or arousal characteristics, or were highly distal from the traumatic event (as much as 20 years). A single-blow incident reduces variation in symptoms attributable to event type (e.g., motor vehicle accident and physical assault) and tightly links assessments to the event so that participants are all equidistant in time from the traumatic experience.

Our goal was to illuminate how PTSD networks change over time. Because we reasoned that this development should bear a relation to theories of PTSD, we compared changes in symptom networks with theoretical frameworks for PTSD. Thus, we explored whether changes in symptom-to-symptom relationships were more consistent with memory models (Brewin et al., 1996; Rubin et al., 2008), a fear-conditioning–emotional-processing model (Foa et al., 1992), or a cognitive-appraisal-based model (Ehlers & Clark, 2000). Because of our longitudinal design, we also explored whether different theoretical accounts offer insights at early and later stages of the development of the PTSD network. To address these issues, we used newly developed permutation techniques to assess within-group changes from 2 to 12 months, focusing on whether networks become stronger, change in overall structure, exhibit differences in the most central symptoms, and change in terms of the strongest symptom-to-symptom relationships or edge weights over time (van Borkulo et al., 2016). To validate our network results and assess the prognostic utility of individual symptoms, we also examined the predictive capacity of each symptom at 2 months in relation to overall PTSD symptoms at 12 months.

Method

Participants

Participants were drawn from a sample of 368 women who completed at least one postshooting survey (at 2 months, 6 months, and 12 months). The initial sample was 843 female students who had completed a multiuniversity survey of sexual victimization during the same semester as the shooting or the previous semester. For the current study, participants were 212 women who provided complete data at both the 2-month and the 12-month postshooting surveys. Participants were 19.4 years of age on average (SD = 1.4 years, range = 18–27 years) when they completed the initial (preshooting) survey. Eighty-six percent characterized their ethnicity as White/European American, 6% as Asian American, 3% as Black/African American, and 2% as

Latina, 2% as multiethnic, and 1% did not indicate their ethnicity or marked other.

Procedure

Complete study procedures have been detailed elsewhere (e.g., Grills-Taquechel, Littleton, & Axsom, 2011). To summarize, women 18 years and older who were enrolled in a psychology course at Virginia Tech initially received course credit to take part in a multiuniversity online survey of women's negative sexual experiences conducted before the shooting. As part of this survey, measures were completed regarding psychological distress, sexual victimization history, and social support. At approximately 2, 6, and 12 months after the shooting, participants were e-mailed an invitation to participate in an online survey related to risk and resilience in response to the shooting. Only participants who completed one of the two prior postshooting surveys were contacted about the 12-month postshooting survey. All surveys were approved by the university institutional review board at Virginia Tech and a university committee designed to ensure ethical research conduct (Coping, Adjustment, and Resilience Among College Women Following the Mass Shooting at Virginia Tech, VT IRB 07-283).

Measures

PTSD symptoms. The PTSD Symptom Scale, Self-Report (PSS-SR; Foa, Riggs, Dancu, & Rothbaum, 1993) was administered at both postshooting surveys to assess PTSD symptoms in connection to the campus shooting. This measure consists of 17 items designed to map on to the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV*; American Psychiatric Association, 1994) reexperiencing, avoidance/numbing, and arousal PTSD symptoms. At each survey, participants were asked to rate how often they had a symptom in the past week in connection to the shooting on a 4-point scale anchored by 0 (*not at all or only one time*) and 3 (*five or more times per week/almost always*). Scores can range from 0 to 51, and a cutoff score of 14 for probable PTSD is recommended (Coffey, Gudmundsdottir, Beck, Palyo, & Miller, 2006).

Exposure to the shootings. Participants were asked 11 yes/no questions regarding their direct exposure to aspects of the shooting (e.g., if they were on campus during the shooting, heard gunfire, witnessed the gunman, knew anyone wounded). On the basis of a sum score of items (in which 1 = yes and 0 = no), participants reported a moderate degree of exposure to the shootings (M = 5.23, SD = 2.84; median = 6.0).

Data analysis

Network estimation. We used the qgraph package (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012) for the R software environment (Version 3.5.1; R Core Team, 2018) to compute networks of PTSD symptoms. To ensure a parsimonious network model, we employed the least absolute shrinkage and selection operator (LASSO) technique. In addition to partialling out all other correlations, the LASSO technique regularizes the model using the overall covariance matrix to shrink nonsignificant coefficients to zero, which avoids problems with conventional forward or backward removal techniques. The hyperparameter for regularization was set at 0.5, the default setting, to reduce the likelihood of spurious edges.

We used qgraph to visually represent the strength of network relationships. In network graphs, thicker lines between nodes indicate stronger partial correlations (edge weights); the absence of a line indicates no correlation. To specify the arrangement of nodes in the network, we used the Fruchterman-Reingold algorithm, which positions nodes in a manner that facilitates the viewing of network edges. To facilitate comparisons and guard against inappropriate inferences based on visualization, we used the average layout function implemented in qgraph (Epskamp et al., 2012).

Network centrality. To determine the importance (or centrality) of symptoms in our networks, we first focused on the strength metric (Opsahl, Agneessens, & Skvoretz, 2010) because of its theoretical relevance to and reliability in psychopathology networks (Fried et al., 2018). Strength reflects the sum of the absolute value of edge weights for a given node (regularized partial correlations). A symptom with higher strength has stronger relationships with other symptoms (edge weights) and thus is more central to the network. An extension of the strength metric is expected influence (EI; Robinaugh et al., 2016). EI calculates the sum of edge weights but retains the negative value (or sign) of the weight. EI is identical to the strength index when there are no negative edges but can diverge substantially from the strength index when there are negative edges. EI provides a more accurate index of node centrality when negative edges are present (Robinaugh et al., 2016).

Network stability. To determine the reliability of the centrality indices, we used techniques that employ bootstrapped subsets of the data that estimate the percentage of data that can be dropped while still retaining a .7 correlation with the original data. Conventionally, this percentage should not fall below 25% and should ideally be

50% (Epskamp, Borsboom, & Fried, 2018). We used 1,000 bootstrapped subsets to assess the stability of the centrality indices. The case dropping bootstrap indicated that EI possessed adequate stability at 2 months (0.36) and at 12 months (0.28). However, strength (0.13, 0.21), betweenness (0.00, 0.05), and closeness (0.13, 0.21) did not. As a result, we used EI to determine centrality.

Network comparisons from 2 to 12 months. To investigate change in PTSD networks from 2 to 12 months, we used the network comparison test (NCT; van Borkulo et al., 2017), a permutation test that repeatedly calculates differences in networks in randomly regrouped individuals, providing bootstrapped confidence intervals that test differences between two networks. The NCT can be used to make between-group comparisons, but it also can be used to compare within-group network changes over time. In the present study, we used permutation tests to examine within-group change in (a) overall network strength, (b) overall network structure, (c) symptom-node centrality, and (d) edge-weight strength. As a way of validating our network results and investigating the prognostic utility of individual symptoms, we used a final analytic step to regress each symptom score at 2 months, in a multivariate model, on total PTSD scores at 12 months. These exploratory analyses allowed us to control for 2-month PTSD at the symptom level and assess change in PTSD symptoms at the total level, providing an estimate of the predictive capacity of each symptom on later elevations in overall PTSD symptoms.

Results

Sample exposure and PTSD symptoms

All participants reported exposure to the shootings. A majority reported knowing someone who had been killed (68.4%), being on campus when the shootings occurred (65.1%), seeing the police on campus (59.4%), and being locked down in a campus building (51.4%). Some participants reported knowing someone who was wounded (36.8%), losing a friend in the shooting (34%), seeing people who were wounded (15.6%), and hearing gunfire (14.6%). PTSD symptoms in connection to the shooting were frequently reported; 96.0% reported at least one PTSD symptom at 2 months, and 91.5% reported at least one symptom at 12 months. A total of 33% of participants scored above the cutoff for probable shooting-related PTSD at each time point (Coffey et al., 2006).

PTSD networks

Figure 1 provides a visual depiction of the PTSD LASSO networks at each wave of data collection using an average of their layouts. The magnitude of the correlation is indicated by the thickness of the line and the direction by the color of the line (blue for positive; red for negative). Because negative edges were present in our LASSO networks (2 months = 28.1%, 12 months = 12.8%) and only expected influence revealed adequate stability, we relied on EI as our measure of centrality (Robinaugh et al., 2016). At both time points, the sample had a modest degree of symptom variance (see Table 1). Given the modest degree of variance for some symptoms, we first examined potential floor effects as a factor in centrality. That is, a symptom with lower levels of endorsement could show weaker centrality simply because it has lower levels of variability. To address this possibility, we correlated strength centrality with the standard deviation for each symptom. A moderate or strong correlation would suggest a systematic relationship between the variance and the centrality of a symptom. However, the strength index was weakly inversely related to the standard deviation in zero-order and rank-order correlations at 2 months ($r = -.12$; $r_s = -.07$) and at 12 months ($r = -.12$; $r_s = -.02$), suggesting that differential variance is not driving node strength centrality for a given symptom.

Comparisons of networks from 2 to 12 months

Change in network strength and structure. Theoretical accounts imply that PTSD networks should undergo change. We first examined this change in terms of overall network strength and overall network structure. Using a permutation test with 10,000 iterations, we found that the PTSD networks both got stronger and changed in the structure of their edge weights from 2 to 12 months. Specifically, overall global strength increased significantly from 2 months (global strength = 7.20, variance = 12.46) to 12 months (global strength = 7.69, variance = 12.26, $p = .028$), and network structure changed ($p = .018$), indicating that the strength of specific edge weights showed significant change from 2 to 12 months. We next investigated the specific nature of these network changes.

Change in expected influence centrality. We examined change in symptom node EI centrality from 2 to 12 months. As shown in Figure 2, some symptoms retained high EI centrality at both time points, and others saw substantial change, becoming either more or less central. At 2 months, the node with the highest EI, by far, was psychological reactivity at reminders. Other nodes with high centrality at 2 months were feeling distant, startle, and avoidance of situations. At 12 months, however, several of the most central nodes had changed position. For example, reliving or flashbacks was the second least

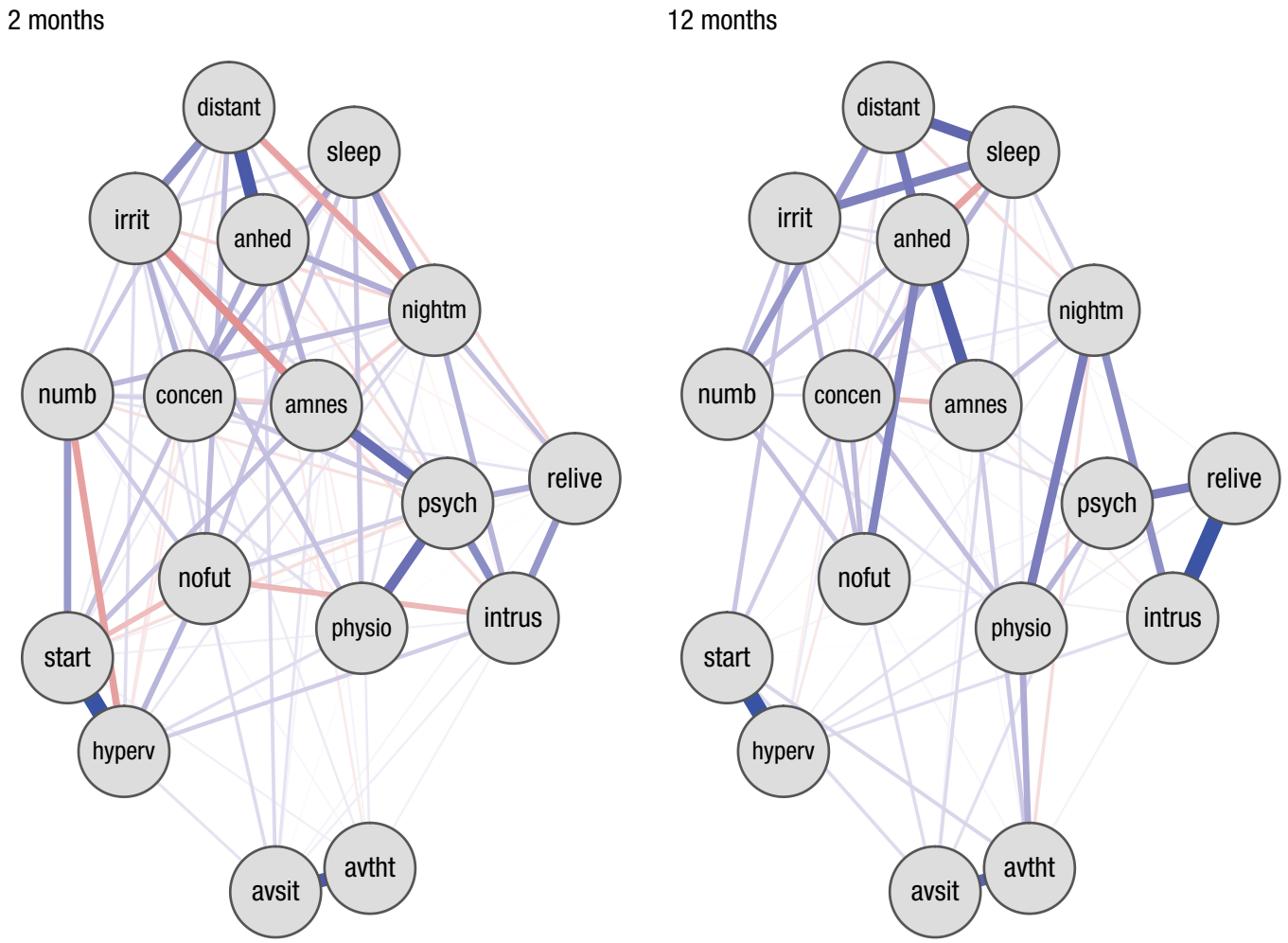

Fig. 1. Least absolute shrinkage and selection operator networks for posttraumatic stress disorder symptoms at 2 and 12 months. Intrus = intrusive thoughts; nightm = distressing dreams; relive = reliving or flashbacks; psych = psychological reactivity; physio = physiological reactions; avtht = avoidance of thoughts; avsit = avoidance of situations; amnes = amnesia; anhed = anhedonia; distant = feeling distant; numb = feeling numb; nofut = no future; sleep = sleep difficulties; irrit = irritability or anger; concent = concentration difficulties; hyperv = hypervigilance; start = exaggerated startle.

central node at 2 months but one of the most central nodes at 12 months. Likewise, physiological distress, intrusive thoughts or images, avoidance of thoughts, and anhedonia all had modest EI centrality at 2 months but became considerably more central at 12 months. Conversely, the most central symptom at 2 months, psychological distress at reminders, was one of the least central at 12 months. Finally, amnesia also increased in its EI centrality from 2 to 12 months, but it remained the symptom node with the least EI centrality of all symptoms at both time points.

To assess the reliability of changes in EI centrality, we used a permutation test with 10,000 iterations for each symptom node. As shown in Table 2, anhedonia ($p = .029$) and avoidance of thoughts ($p = .044$) increased significantly, whereas reliving or flashbacks showed a marginal increase ($p = .057$) from 2 to 12 months. In addition, psychological reactivity showed a significant decrease ($p = .036$) from 2 to 12 months in EI centrality. These results suggested that several symptom nodes underwent change from 2 to 12 months. However, when we corrected for multiple tests using the false discovery rate (Benjamini & Hochberg, 1995), a stringent procedure given the large number of tests, there were no node differences ($ps > .23$).

Change in edge weights. We next examined how edge weights differed from one another and whether they changed over time. We first looked at whether some edges within each network were significantly stronger than other edges in the network. To assess whether a given edge differed significantly from other network edges, we used bootstrapped confidence intervals calculated from permutation tests with 10,000 iterations (van Borkulo et al., 2016). On the basis of these results, we also calculated an *edge difference percentage* (EDP), a metric

Table 1. Mean PTSD Symptoms at 2 and 12 Months After the Shooting ($N = 212$)

Symptom variable	2 months	12 months
Intrusive thoughts or images	0.86 (0.86)	0.68 (0.82)
Distressing dreams	0.36 (0.66)	0.29 (0.61)
Reliving or flashbacks	0.44 (0.71)	0.35 (0.64)
Psychological reactivity	1.28 (0.89)	1.00 (0.87)
Physiological reactivity	0.49 (0.76)	0.41 (0.72)
Avoidance of thoughts	1.08 (1.03)	0.87 (0.97)
Avoidance of situations	0.59 (0.87)	0.66 (0.94)
Amnesia	0.20 (0.51)	0.22 (0.60)
Anhedonia	0.26 (0.59)	0.35 (0.68)
Feeling distant or cut off	0.49 (0.79)	0.45 (0.75)
Feeling numb	0.50 (0.80)	0.46 (0.81)
No future	0.48 (0.80)	0.58 (0.91)
Sleep difficulties	0.93 (1.04)	0.88 (1.02)
Irritability or anger	0.68 (0.83)	0.45 (0.77)
Concentration difficulties	0.82 (0.97)	0.87 (1.01)
Hypervigilance	0.95 (1.09)	1.02 (1.05)
Exaggerated startle	0.99 (1.09)	0.92 (1.02)

Note: Values are means with standard deviations are in parentheses. PTSD = posttraumatic stress disorder.

developed for this study that divided the count of significant differences with other edges by the total number of other edges recovered in the network. This provided a metric for the degree to which an edge differed from other edges. For example, an edge that is significantly different from every other edge in the network would have an EDP of 100%.

At 2 months, the five strongest edges were significantly different from the considerable majority of other network edges, including hypervigilance–startle (edge = .72, EDP = 95.9%), avoid situations–avoid thoughts (edge = .55, EDP = 84.5%), anhedonia–distant (edge = .55, EDP = 75.3%), psychological reactivity–amnesia (edge = .44, EDP = 68.0%), and psychological reactivity–physiological reactivity (edge = .39, EDP = 60.8%). At 12 months, several edge weights had changed in key respects. For example, reliving–intrusions was not a particularly strong edge at 2 months (edge = .28), but it became a dominant edge at 12 months (edge = .61, EDP = 93.6%), significantly stronger than almost every other edge. Sleep problems–feeling distant also emerged as a strong edge at 12 months (edge = .40, EDP = 64.1%). We also found evidence that a number of the top edges replicated. Hypervigilance–startle was again the strongest edge (edge = .66, EDP = 96.2%), and avoid situations–avoid thoughts, as the fourth strongest, largely replicated (edge = .43, EDP = 60.3%). Anhedonia also retained a link to feeling distant (edge = .34, EDP = 62.8%) as the sixth strongest. At 2 months, three of the strongest six edges involved psychological reactivity,

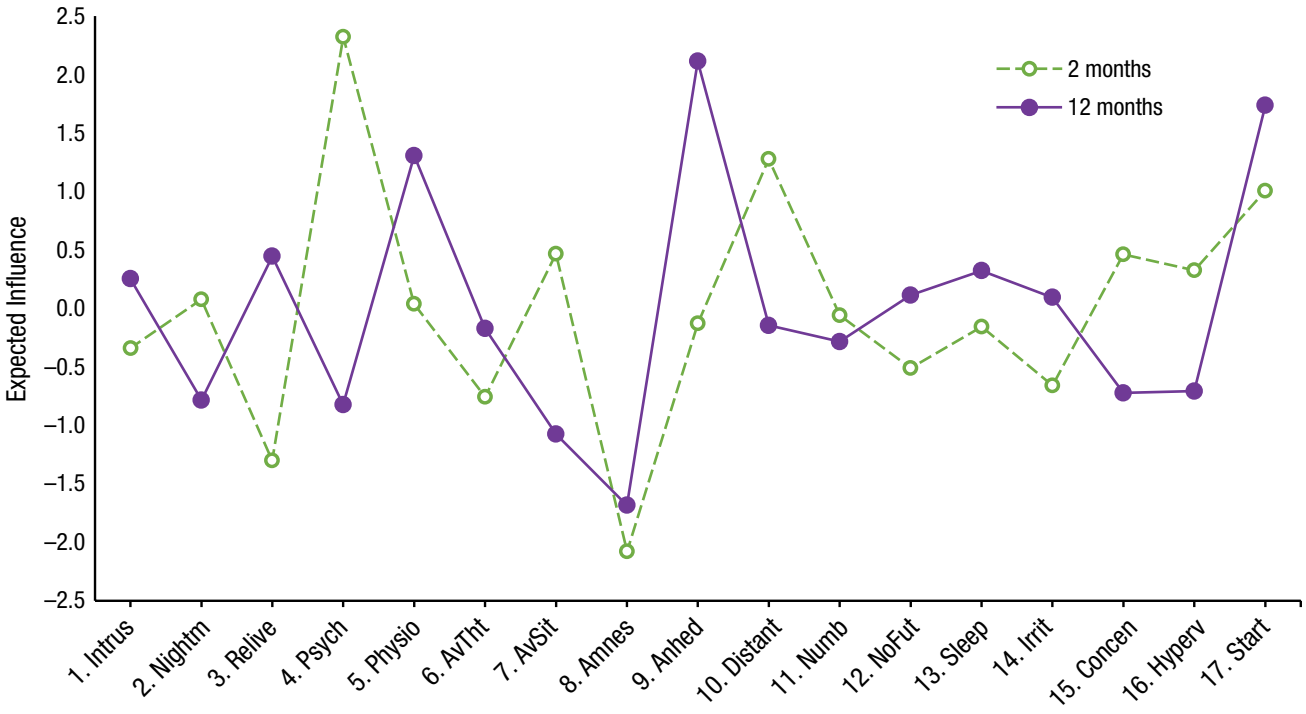

Fig. 2. Comparing expected influence (standardized) at 2 and 12 months after the shooting. Intrus = intrusive thoughts; nightm = distressing dreams; relive = reliving or flashbacks; psych = psychological reactivity; physio = physiological reactions; avtht = avoidance of thoughts; avsit = avoidance of situations; amnes = amnesia; anhed = anhedonia; distant = feeling distant; numb = feeling numb; nofut = no future; sleep = sleep difficulties; irrit = irritability or anger; concent = concentration difficulties; hyperv = hypervigilance; start = exaggerated startle.

Table 2. Change in Expected Influence (EI) for PTSD Symptoms From 2 to 12 Months

Symptom node	EI		EI change	p^a
	2 months	12 months		
Intrusive thoughts or images	0.83 (12)	**0.98 (6)**	0.15	.79
Distressing dreams	0.93 (7)	0.85 (14)	–0.08	.53
Reliving or flashbacks	0.61 (16)	1.01 **(4)**	0.40	.06
Psychological reactivity	1.46 **(1)**	0.84 (13)	–0.62	.04
Physiological reactivity	0.92 (8)	**1.12 (3)**	0.20	.59
Avoidance of thoughts	0.73 (15)	0.93 (9)	0.19	.04
Avoidance of situation	**1.02 (4)**	0.81 (16)	–0.21	.84
Amnesia	0.42 (17)	0.73 (17)	0.31	.16
Anhedonia	0.88 (10)	1.22 **(1)**	0.34	.03
Feeling distant or cut-off	**1.21 (2)**	0.93 (9)	–0.28	.18
Feeling numb	0.90 (9)	0.91 (10)	0.01	.35
No future	0.79 (13)	0.96 (7)	0.17	.16
Sleep difficulties	0.88 (11)	**0.99 (5)**	0.12	.84
Irritability or anger	0.76 (14)	0.96 (8)	0.20	.77
Concentration difficulties	**1.02 (5)**	0.86 (11)	–0.17	.21
Hypervigilance	**0.99 (6)**	0.86 (12)	–0.13	.19
Exaggerated startle	**1.15 (3)**	**1.18 (2)**	0.03	.38

Note: Rank order in parentheses; top six in boldface type. EI = expected influence; PTSD = posttraumatic stress disorder.
[a]The p value is based on a permutation test with 10,000 iterations.

whereas at 12 months, no edges with psychological reactivity were among the top six. Because edge weights are partial correlation coefficients, their magnitude can be interpreted according to effect size conventions. A number of edge weights went from either negligible or small effects to medium and large effects, including sleep–feeling distant, anhedonia–amnesia, intrusions–reliving, physiological reactivity–nightmares, and sleep–irritability (for the top 10 edge weights at each time point and the degree to which they differed from other network edges, see Table S1 in the Supplemental Material available online).

We next examined edge-weight changes from 2 to 12 months using permutation tests with 10,000 iterations (van Borkulo et al., 2016). We compared edges that were present at either 2 or 12 months (k = 113 edges). These comparisons revealed 11 edges that showed significant change at $p < .05$ (see Table S4 in the Supplemental Material for p values of all edge-weight changes). As shown in Table 3, the edge weight that showed the largest increase was sleep–feeling distant. Other edges that showed a marked and statistically significant increase were irritability–amnesia, intrusions–no future, anhedonia–amnesia, intrusions–reliving, anhedonia–no future, and physiological reactivity–avoid thoughts. Of the 10 largest edge-weight increases, 3 involved physiological reactivity. One of the strongest edge-weight increases was reliving–intrusions. Consistent with node EI analyses, the two largest edge-weight decreases involved psychological reactivity, which decreased markedly in its edges with amnesia and intrusions as well as in its edge with physiological reactivity to reminders. Although these findings were generally consistent with an increased role for reliving, anhedonia, and physiological reactivity, a correction for the false discovery rate using the Benjamini-Hochberg procedure resulted in only two edge-weight changes remaining significant (sleep–feeling distant; irritability–amnesia). The remaining edge changes were not (ps > .32). However, given the large number of comparisons (k = 113), this resulted in a very stringent correction.

Negative edge weights

One unusual feature of the LASSO networks was a high proportion of negative edges at 2 months (28.7%). By contrast, association networks (bivariate relationships) revealed no negative correlations among symptoms at 2 or 12 months. This indicated that negative edges between symptoms emerged only when controlling for the effect of other symptoms. To better understand

Table 3. Edge Weight Increases and Decreases in LASSO Networks From 2 to 12 Months

	Edge weight			
Symptom pair	2 months	12 months	Change	*p*
Increases				
Sleep–feeling distant	0.00	0.40	0.40	< .001
Amnesia–irritability	–0.32	0.05	0.37	.001
Anhedonia–amnesia	0.12	0.47	0.35	.01
Reliving–intrusions	0.28	0.61	0.35	.03
Anhedonia–no future	0.00	0.31	0.31	.03
Physiological–nightmares	0.09	0.33	0.24	.13
Intrusions–no future	–0.20	0.03	0.23	.04
Physiological–concentration	–0.05	0.17	0.25	.09
Sleep–irritability	0.10	0.32	0.22	.10
Physiological–avoid thoughts	0.00	0.21	0.21	.02
Decreases				
Psychological reactivity–amnesia	0.40	0.07	–0.33	.10
Psychological reactivity–intrusion	0.32	0.01	–0.31	.10
Distant–irritability	0.31	0.00	–0.31	.09
Numb–startle	0.27	0.00	–0.27	.17
Amnesia–distant	0.22	0.00	–0.21	.03
Anhedonia–distant	0.55	0.34	–0.21	.06
Anhedonia–nightmares	0.23	0.02	–0.21	.86
Psychological reactivity–physiological	0.39	0.19	–0.20	.13

Note: Change reflects 12 months – 2 months. Table includes edge-weight changes of more than .20 in which at least one edge weight is more than .20 from zero. The *p* value is based on a permutation test with 10,000 iterations. LASSO = least absolute shrinkage and selection operator.

these negative edges, we examined them qualitatively. (See Tables B and C in the Supplemental Material for a full reporting of all negative edges at 2 and 12 months.) At 2 months, the five strongest negative edges were no future–startle (edge = –.32), numbness–hypervigilance (edge = –.27), nightmares–feeling distant (edge = –.26), intrusions–no future (edge = –.27), and startle–no future (edge = –.18). By contrast, at 12 months, negative edges were fewer (12.1%) and involved weaker relationships. The five strongest negative edges at 12 months were anhedonia–sleep (edge = –.24), amnesia–concentration (edge = –.16), nightmares–distant (edge = –.10), nightmares–avoid thoughts (edge = –.09), and anhedonia–hypervigilance (edge = –.06). These findings indicated that negative edges are both more prominent in the early network and often involve symptom pairings of activating (e.g., hypervigilance) and vegetative states (e.g., numbness).

Symptoms as prospective predictors

A final exploratory analysis used symptoms at 2 months to predict overall PTSD symptoms at 12 months. We reasoned that if a symptom plays an increasing role in the PTSD network from 2 to 12 months, it may also be uniquely prognostic of later PTSD symptoms. To examine this possibility, we simultaneously regressed all 2-month symptom scores on total PTSD scores at 12 months, correcting for multiple comparisons using the false discovery rate (Benjamini & Hochberg, 1995). The overall model was significant, $F(17, 194) = 13.19$, $p < .001$, $R^2 = .55$. After adjusting for the influence of other symptoms, the only significant symptom predictors were reliving at 2 months ($B = 2.39$, $SE = .81$, 95% CI = [0.79, 3.90], semipartial $r^2 = .14$, $p = .003$; corrected p value = .051) and anhedonia at 2 months ($B = 2.91$, $SE = 1.07$, 95% CI = [0.80, 5.00], semipartial $r^2 = .13$, $p = .007$; corrected p value = .059). No other symptom scores at 2 months were predictive of later PTSD symptoms (ps > .07; corrected p values > .23; for full results, see Table S5 in the Supplemental Material). In addition to suggesting that these symptoms are uniquely prognostic of later elevations in symptoms, these results were remarkably consistent with the observed changes in network centrality: Reliving and anhedonia showed the two largest increases in expected influence centrality from 2 to 12 months.

Discussion

Theorists and clinicians have long proposed that PTSD symptoms develop and mutually influence one another over time. In the present study, we used network analysis to better understand the development of PTSD among a sample of female participants exposed to the Virginia Tech campus shootings. A number of findings emerged. We found that the centrality of several key symptoms changed substantially over time. Psychological reactivity was by far the most central symptom at 2 months, but it was one of the least central symptoms at 12 months. The opposite pattern was observed for reliving, anhedonia, and physiological reactivity to reminders, which had modest to minimal centrality in the network at 2 months but were among the most central symptoms at 12 months. In direct tests of change across time points, we found that psychological reactivity significantly declined from 2 to 12 months in its node centrality, whereas anhedonia and thought avoidance significantly and reliving marginally increased in centrality. We also found that reliving and anhedonia at 2 months uniquely predicted overall PTSD scores at 12 months, which suggests that these symptoms may have specific utility as prognostic risk markers in assessments of PTSD. Taken together, these findings suggest that the relatively more automatic and situationally cued symptoms, including reliving, intrusions, thought avoidance, and physiological reactions, become more central to the PTSD network, whereas psychological reactivity to reminders becomes less central to the network.

Nevertheless, it should be noted that PTSD network analyses have not converged on a consistent set of symptoms with the strongest centrality (Forbes, Wright, Markon, & Krueger, 2017b). Although this lack of convergence is perhaps unsurprising given the considerable methodological and sample variation in prior PTSD network studies, it raises questions about our capacity to draw generalizable inferences about centrality from psychopathology networks. We found that our results converged in some respects with prior network analyses and diverged in others. A prior network analysis of the Virginia Tech shootings found high centrality for intrusions, anger, sleeping difficulties, and concentration difficulties (Sullivan et al., 2018). However, this study did not measure key symptoms (reliving, physiological reactivity, and anhedonia). By contrast, our findings comported in key respects with a recent multisite examination of the replicability of PTSD networks (Fried et al., 2018). Fried and colleagues (2018) found that a composite of psychological and physiological reactivity had the highest centrality and startle and anhedonia also had high node centrality. We found a largely similar degree of centrality for each of these symptom nodes. However, Fried and colleagues combined psychological reactivity and physiological reactivity into one symptom node in order to align different PTSD measures. Our results suggest psychological and physiological reactivity diverge in centrality over time, and it is therefore critical to measure them separately. A final point is that amnesia had the lowest centrality at both time points, which is consistent with all prior PTSD network analyses (Armour et al., 2017; Bryant et al., 2017; Fried et al., 2018; McNally et al., 2015).

Together, these findings provide evidence that some aspects of PTSD networks change, whereas others remain stable over time. Broadly, this finding is compatible with the only prior network analysis of PTSD symptoms including multiple time points (Bryant et al., 2017), which also identified changes in the centrality of symptoms and an increase in overall network connectivity across time. However, the findings from the present analysis differed in a number of key respects. Whereas Bryant and colleagues (2017) found that reexperiencing symptoms, including intrusions and physiological reactivity, were highly central directly following the traumatic injury, we found that among the reexperiencing cluster of symptoms, only psychological reactivity at reminders was a central component of the network at 2 months. Unusually, Bryant and colleagues also found that concentration difficulties had the highest level of strength centrality at both time points, a finding that is unique among prior PTSD network studies and at variance with the present findings (Bryant et al., 2017; Fried et al., 2018; McNally et al., 2015; Mitchell et al., 2017; Sullivan et al., 2018). One plausible explanation for the high centrality of concentration difficulties is the high proportion of participants with traumatic brain injuries (Bryant et al., 2017). A final difference is that unlike Bryant and colleagues, we found that general distress symptoms, including sleep difficulties and anhedonia, were among the most central symptoms at 12 months and that anhedonia increased in centrality over time. These differences likely reflect, at least in part, that the Bryant and colleagues study assessed participants within 7 days of their injury, at a time when the memory of the experience was undoubtedly fresh and when reexperiencing symptoms (e.g., intrusive thoughts) would likely be normative reactions to the event.

Although theories of PTSD converge in many respects and thus offer few competing predictions, we found suggestive evidence that different theoretical accounts may be operative at different stages of the development of PTSD. If we consider psychological distress at reminders as reflecting higher order appraisal processes, then a cognitive account can most clearly explain the dominant influence of this symptom early in the PTSD network (Ehlers & Clark, 2000). However,

the present results suggest that appraisal processes are primarily central soon after the event, perhaps before a causal system of PTSD symptoms has coalesced. On the other hand, reliving symptoms increased in their overall network centrality from 2 to 12 months and became considerably more powerfully linked to intrusions. This increase in the centrality of reliving, a component of involuntary memory, is broadly in line with memory models of PTSD. Multirepresentation models suggest that the trauma memory of individuals with PTSD is poorly integrated with autobiographical memory and thus overly active in response to situational cues (Brewin et al., 1996). Given the low centrality of amnesia, the results also supported a mnemonic account of PTSD as driven primarily by an excess of remembering, not an inability to access memory (Rubin et al., 2008). Memories of the trauma may be further strengthened by the fear-conditioning effect of physiological reactivity to reminders (Foa et al., 1992), which increased in its relation to avoidance of thoughts, marginally increased in relation to concentration, and was the third most central symptom overall at 12 months. Indeed, these results are roughly consistent with the idea that fear conditioning can enhance the long-term potentiation of associative amygdala-based memories of the trauma (Rogan, Stäubli, & LeDoux, 1997).

The current findings raise a number of questions. Why would reliving, thought avoidance, and physiological reactivity primarily emerge as central later on? One explanation integrates memory-based, cognitive, and fear-conditioning accounts of PTSD (Ehlers & Clark, 2000; Foa et al., 1992). In a cognitive model, negative appraisals of a potentially traumatic experience and one's initial reactions (during and after trauma) are directly linked to distress and aversive physiological arousal. Indeed, one of the strongest edge weights at 2 months was psychological distress–physiological reactions, but this edge declined considerably at 12 months, though not statistically significantly. At the same time, the more automatic and situationally cued symptoms of reliving and thought avoidance increased significantly in expected influence. If these conditioned memory-based responses predominate over time, they would be more difficult to inhibit, activating other symptoms in the PTSD network (Falconer et al., 2008). Edge weight differences further supported this possibility. Among the largest edge-weight changes from 2 to 12 months was intrusions–reliving, which went from a small to a large effect, indicating that intrusions became more vivid. Another edge that showed significant change was physiological reactions–avoidance of thoughts, indicating that avoiding thoughts became more strongly linked with physiological arousal.

A key point is that these network changes occurred in comparisons of the same participants at two time points, suggesting that these changes capture the developing PTSD network. For this reason, the present findings bear implications for PTSD treatment, psychoeducation, and risk assessment. Prolonged exposure, widely viewed as the frontline psychotherapeutic treatment for PTSD, explicitly targets conditioned automatic associations to reminders, a treatment focus supported by the present findings. However, the present results also suggest that exposure-based approaches are counterindicated as a blanket intervention and should be reserved for those with elevated symptom levels (Bryant, Harvey, Dang, Sackville, & Basten, 1998). Indeed, as has been widely shown, blanket emotional debriefing interventions are ineffective and potentially harmful (McNally, Bryant, & Ehlers, 2003). Furthermore, these findings suggest that early interventions may demonstrate greater efficacy if they employ cognitive techniques, such as cognitive reappraisal, that seek to modify the interpretation or appraisal of the traumatic event and initial PTSD symptoms (Ehlers & Clark, 2000). Such techniques may interrupt the potential conditioning effects of negative appraisals on automatic physiological and reliving symptoms. By the same token, the present results suggest that the early PTSD network is characterized by psychological distress, a likely normative response to the event that would not require intervention. Although psychoeducation in and of itself is not efficacious as an intervention (Kearns, Ressler, Zatzick, & Rothbaum, 2012), it would be important to normalize psychological distress after an acutely traumatic event as a common and likely transient human experience.

When we examined 2-month symptoms as prospective predictors of total 12-month symptoms, we found evidence that some early symptoms may be risk markers for the later development of PTSD. Reliving and anhedonia at 2 months were the only symptoms that uniquely predicted total PTSD symptoms at 12 months when adjusting for all other symptoms at 2 months. This result converged with the network change analyses to a remarkable degree: Reliving and anhedonia showed the two largest increases in expected influence centrality from 2 to 12 months. Given that these two symptoms were also among the least infrequently endorsed at 2 months, these findings suggest that the presence of reliving and anhedonia symptoms may help clinicians to distinguish normative stress responses from those that pose risk of subsequent elevations and a more persistent course of PTSD symptoms.

One unexpected finding was a relatively high proportion of negative edges at 2 months. Negative edges were completely absent in the bivariate concentration

networks at 2 and 12 months. Thus, only in the presence of other symptoms did negative relationships emerge. Why would conditional negative edges emerge when prior PTSD network analyses have not observed them to the same degree? One possibility is conditional negative edges may primarily occur soon after a traumatic event, when transient PTSD symptoms would be less likely to have syndromal coherence (Shalev, 2002). Indeed, we would expect both a weaker network (less overall connectivity) and more negative edges early on because the causal system would be less organized. Negative edges should furthermore involve an activating and a vegetative or dysphoric symptom because these states are plausibly inversely related when you adjust for the influence of other symptoms. That is, if you feel hypervigilant, you are less likely to feel numb at the same time unless you have other symptoms that contribute to numbness. Consistent with this possibility, one of the strongest negative edges was numbness–hypervigilance, and the top five negative edges all involved an activating and a vegetative or dysphoric symptom, including amnesia–startle, nightmares–feeling distant, and intrusions–no future. However, these negative edges became less numerous and weaker in strength at 12 months. The reduced proportion of negative edges at 12 months implies that as the network forms a causal system, the likelihood of negative edges is reduced. Nevertheless, it will be important for future research to replicate the presence of negative edges under these specific methodological conditions (among normative samples soon after an event).

Limitations

A number of factors constrain the conclusions from this study. A key limitation is that network relationships are correlational and cannot be presumed to reflect causal processes. They could be the result of other unmeasured variables or causes or unobserved latent variables that underlie those relationships (Forbes et al., 2017a). Another limitation is that we found few significant differences when we corrected for multiple tests. However, because we conducted a large number of tests, resulting in a substantially corrected p value, the failure to reject the null hypothesis for most node centrality or edge differences does not imply that the networks are identical. Indeed, global tests indicated that the networks differed significantly in structure and strength. Instead, it indicates that our current sample did not have sufficient power to reliably detect the specific differences between edges or node centralities after corrections. As a result, the uncorrected results should be interpreted in terms of generating hypotheses for future investigation in higher-powered samples.

A further limitation is that cross-sectional between-person network structures cannot be interpreted as identical to the longitudinal within-person network structure of a given individual (Bos et al., 2017; Molenaar, 2004). Experience sampling methods can identify intrainidividual variation using idiographic networks (Epskamp, van Borkulo, et al., 2018; Fisher, Reeves, Lawyer, Medaglia, & Rubel, 2017), a critical necessity for future research. Nevertheless, between-person, cross-sectional networks can examine conditional dependence relations that provide valuable clues to causal relationships (Epskamp, van Borkulo, et al., 2018). Other limitations of the present study include a non-treatment-seeking sample of women with variable levels of direct exposure to the trauma. Another key point is that these results characterize the development of distressing symptoms in a sample that included a broad range of symptom levels but that was not limited to persons with a PTSD diagnosis. Thus, the present results may not be generalizable to treatment-seeking or other highly distressed samples. By the same token, given that our sample was all exposed to the same event, it is unclear if the network changes we observed would be observed for other types of traumatic events. A final point is that some participants may have received mental-health treatment between 2 and 12 months, and such treatment could have influenced network relationships.

Conclusion

Network analyses offer a compelling theoretical challenge to the traditional ontological view of psychopathology (Hofmann, Curtiss, & McNally, 2016). The present data suggest that they can also inform our theoretical understanding of specific forms of psychopathology by identifying how symptom relationships differ early and late in the development of a mental-health syndrome. Future network research should use within-person methodologies, larger and higher-powered samples with more frequent assessments, and examine factors that may protect against or serve to maintain clinical syndromes, such as information processing biases, beliefs about the self, and social, functional, and environmental factors (Jones, Heeren, & McNally, 2017). Ultimately, a more fine-grained understanding of the developmental progression of PTSD and other mental disorders can offer crucial guidance in effective assessment and treatment.

Action Editor

Stefan G. Hofmann served as action editor for this article.

Author Contributions

A. D. Mancini, H. L. Littleton, and A. E. Grills developed the manuscript concept. A. D. Mancini performed the data analyses with assistance from P. J. Jones. A. D. Mancini had primary responsibility for drafting the manuscript, and H. L. Littleon drafted a section of the manuscript. H. L. Littleton, A. E. Grills, and P. J. Jones provided critical revisions. All of the authors approved the final manuscript for submission.

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Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

Funding

This study was supported by National Science Foundation Grant BCS-0737940.

Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702619859333

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