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A Network Analysis of Two Conceptual Approaches to the Etiology of PTSD

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Abstract

Two prominent conceptual models of posttraumatic stress disorder (PTSD) are the cognitive model, associated with cognitive processing therapy (CPT; Resick & Schnicke, 1992), and the functional contextualist model, underlying acceptance and commitment therapy (ACT; Hayes et al., 1999). Network analysis was used to examine dynamic interactions among cognitive (relating to CPT) and functional contextualistic (relating to ACT) variables and PTSD symptoms in a sample of 722 trauma-exposed adults. Results from the cognitive networks highlighted the importance of maladaptive beliefs about threat in maintaining the co-occurrence of PTSD symptoms and cognitive variables. Additionally, PTSD symptoms were more likely to lead to cognitive variables, rather than the reverse direction. Results from the functional contextualist networks identified numerous associations amongst variables that contribute to the co-occurrence of PTSD symptoms and psychological inflexibility. Findings from this study may help generate causal hypotheses that can be tested further using a longitudinal study design.

Keywords: PTSD, network analysis, cognitive processing therapy, acceptance and commitment therapy, functional contextualism
A Network Analysis of Two Conceptual Approaches to the Etiology of PTSD

Posttraumatic stress disorder (PTSD) is associated with severe dysfunction and substantial economic and social burden (Brady et al., 2000; Kessler et al., 2005). As such, several treatments have been developed to alleviate the suffering associated with PTSD, all of which differ in their conceptualization of factors that confer risk for and maintenance of PTSD. Exploration of how relevant factors from different conceptual models relate to PTSD symptoms helps (1) inform testable hypotheses of the etiology of PTSD, (2) identify core risk and maintenance factors that may be particularly influential in the etiology of PTSD, and (3) inform treatment selection based on an individual’s presenting risk factors. Among treatments that differ substantially in their conceptualization of PTSD are cognitive processing therapy (CPT; Resick & Schnicke, 1992) and acceptance and commitment therapy (ACT; Hayes et al., 1999).

Conceptualizations of PTSD

Cognitive Processing Therapy. CPT was developed based on several prominent theories of PTSD, including information processing theory (Foá et al., 1989), the cognitive theory of anxiety (Beck & Emery, 1985), and theory on schema-discrepant information (Hollon & Garber, 1988). CPT emphasizes the role that schema discrepant information plays in the development of PTSD symptoms and emphasizes directly challenging cognitions associated with these discrepancies. Resick and Schnicke (1992) hypothesized that PTSD symptoms are caused by conflicts between prior beliefs and new trauma-related information. For example, someone who previously held the belief that the world is a safe place is confronted with discrepant information upon being raped in their own home. These discrepancies are referred to as “stuck points” in CPT (Resick et al., 2017). According to Resick and Schnicke (1992), people may respond to these discrepancies in one of three ways following a traumatic event: alter (often minimize) their
memory of the event in order to maintain existing schema (i.e., assimilation), alter beliefs enough to incorporate information from the traumatic event (i.e., accommodation), or over-correct by drastically changing pre-trauma schemas (i.e., overaccommodation). While accommodation is thought to be adaptive, assimilation and overaccommodation are thought to increase the likelihood that one will develop PTSD. Much like Beck and Emery’s (1985) cognitive approach, distorted beliefs that develop as a result of assimilation and overaccommodation are directly challenged in CPT. These distorted beliefs tend to fall into the following three categories: beliefs about self (“I’m to blame for what happened”), others (“Nobody can be trusted”), and the world (“I’m not safe anywhere”).

In support of the cognitive model of PTSD, the number of accommodated (adaptive) beliefs at the outset of treatment is associated with lower PTSD symptom severity (Sobel et al., 2009). Additionally, negative appraisals of the sequelae of a traumatic event (e.g., “Nothing good can happen to me anymore”) demonstrate cross-sectional (Dunmore et al., 1997; 1999; Wenninger & Ehlers, 1998) and longitudinal (Dunmore et al., 2001; Ehring et al., 2008) positive associations with PTSD severity. Taken together, this evidence provides support for maladaptive beliefs about self, others, and the world as risk factors for PTSD. In addition to posing risk for PTSD, maladaptive beliefs appear to be one mechanism by which PTSD symptoms are maintained (Dunmore et al., 1997; 1999), such that CPT reduces maladaptive cognitions, which in turn, reduces PTSD symptoms (Gallagher & Resick, 2012; Gilman et al., 2012). However, it remains unclear how maladaptive beliefs lead to changes in PTSD at the symptom level.

**Acceptance and Commitment Therapy.** Functional contextualism is the philosophical framework upon which ACT is based (Anderson & Simmons, 2008; Hayes et al., 1988; Jacobson, 1997). In a functional contextualist framework, processes (e.g., the functions of a
thought in a given situation) are emphasized over form (e.g., thought content). For example, in a functional contextualist conceptualization of PTSD, avoidance of thoughts or memories associated with the traumatic event may serve different functions for different individuals in different situations. However, when an individual is using behaviors, such as avoidance, rigidly across different contexts, it can lead to emotional distress. The primary goal of ACT, therefore, is to increase psychological flexibility. To do so, six core domains of psychological inflexibility (i.e., the ACT “inhexaflex”) are targeted in treatment: cognitive fusion (CF), self-as-content, experiential avoidance (EA), lack of contact with present moment, lack of contact with values, and inaction (Hayes et al., 1999; McHugh, 2011).

CF is defined as strong attachment to one’s thoughts such that they are interpreted as literally true (Hayes et al., 2013). When CF is high, the individual may begin to see themselves as the culmination of private events. In ACT, this is known as viewing self-as-content (e.g., seeing oneself as their thoughts) rather than viewing self-as-context. Furthermore, when CF is high and private events are uncomfortable, individuals may have a tendency to attempt to avoid those private events. This unwillingness to maintain contact with internal experiences is known as EA (Hayes et al., 2013). The more EA is utilized, the more the individual may begin to live outside of the present moment—stuck in either the past or the future. Lack of contact with the present moment (1) deemphasizes values outside of private events and (2) limits opportunities to engage in valued action. Together, these domains of the inhexaflex maintain distress, under the functional contextualist model.

To date, studies have not examined the associations between specific components of the inhexaflex and specific PTSD symptoms or symptom clusters. However, several hypotheses can be made based on theory. For instance, trauma-related intrusive thoughts may be the result of
increased attempts to avoid thinking about the index event (Blackledge, 2004; Shiperd & Beck, 1999; 2005). As such, intrusion symptoms (i.e., cluster B) and EA should be positively associated. Next, CF, lack of values clarity, and inaction may be related to negative alterations in cognition and mood symptoms (i.e., cluster D). Specifically, CF may exacerbate negative beliefs related to oneself, others, or the world. Relatedly, greater clarity in values and engagement in valued action might increase contact with sources of positive reinforcement, thus reducing symptoms within cluster C (avoidance) and D (negative alterations in cognition).

**Summary of cognitive and functional contextualist models.** The cognitive and the functional contextualist models differ in how the etiology of PTSD is conceptualized. Whereas the content of thoughts is of central importance in the cognitive model, the process and functions of internal (e.g., thoughts, beliefs) and external (e.g., overt behaviors) events are more important in the functional contextual model. It is important to consider how these different models characterize the etiology of PTSD for several reasons. First, theory guides hypotheses. If one is operating under a cognitive framework, hypotheses about the development, maintenance, and treatment of PTSD will focus on the role of maladaptive cognitions and their literal content. If, however, one accepts a functional contextualist model, one’s hypotheses will focus on the function (rather than form) of internal events and the flexibility of external behaviors. Second, a more precise understanding of maintaining factors can provide valuable guidance in selecting and sequencing treatment targets. For example, if CF is shown to be most strongly related to PTSD symptoms within the functional contextualist framework, it would stand to reason that ACT for PTSD should first focus on cognitive defusion. Note that this may differ across disorders; thus, it is important to consider risk factors within a framework by the disorder of interest. Third, clinicians can use such information to determine the most appropriate course of
treatment. For instance, if dysphoric mood and anhedonia are particularly bothersome to an individual, a treatment that explicitly targets factors most closely related to dysphoria and anhedonia (e.g., engagement in value-based behaviors) may be more appropriate than a treatment that directs clinical attention elsewhere.

A network analytic approach provides a novel way to examine relationships between symptoms and conceptual variables in a manner that can help elucidate etiology of a disorder under a given conceptual framework. Unlike latent variable modeling, network analysis examines relationships at the symptom-level. Intervention studies that examine mechanisms of change across a treatment are the gold standard for improving our understanding of conceptual models of disorders. However, they are resource-intensive, and as such require strong theories about proposed risk and maintenance factors prior to testing. Thus, there is a gap between the generation and testing of hypotheses regarding the etiology and maintenance of psychopathology. Network analysis can provide an important step in generating such testable hypotheses. Though network analysis has mainly been used to examine the dynamic interactions among symptoms within a given psychological disorder (Epskamp et al., 2018; McNally, 2016), there has been a call to examine networks that include variables beyond symptoms, such as risk and protective factors (Jones et al., 2017).

The Present Study

The aim of the current study was to examine how PTSD symptoms relate to the risk and maintenance factors identified in the two theoretical conceptualizations of PTSD described above (i.e., cognitive and functional contextualist models) using two network approaches that are well-suited for use with cross-sectional data: the Gaussian graphical model (GGM) and the directed acyclic graph (DAG). GGM networks allow for feedback loops, which are important in
exploring how symptoms and other variables perpetuate each other in a cyclical manner (McNally, Heeren et al., 2017). However, it does not propose directionality. In contrast, DAG networks use arrowed edges to represent predicted directionality (McNally, Mair et al., 2017), but feedback loops are not allowed. Thus, the limitations of the GGM are addressed in the DAG and vice versa. Tandem interpretation of GGM and DAG results allows for more nuanced explanation of results (Bartels et al., 2019; Heeren et al., 2020; McNally, Heeren, et al., 2017).

Network analysis can be exploratory in nature; however, several hypotheses were posited based on guiding theory and existing evidence. Some of the hypotheses below predict relationships between etiological variables and PTSD symptom clusters rather than symptoms themselves. This is because of the paucity of evidence about symptom-level associations. An overarching aim of this study is to provide an initial understanding of relationships between PTSD symptoms and etiological variables from the cognitive and functional contextualist models. Specific hypotheses were as follows. In the GGM cognitive network, (H1) cluster D symptoms (negative alterations in cognition and mood) would have the strongest bridge to etiological variables derived from the cognitive model. This hypothesis is based on research suggesting such variables from the cognitive model may be most highly related to cluster D symptoms (Lancaster et al., 2011). Given the thematic overlap between the cognitive variables, no predictions were made regarding the differential downstream effects that each of the cognitive variables might have on PTSD symptoms in the DAG cognitive network. In the GGM functional contextualist network, it was hypothesized that multiple paths would connect functional contextualist variables and specific PTSD symptoms. Specifically, (H2) EA was expected to be most strongly connected to cluster B (intrusive symptoms), given the paradoxical nature of EA on PTSD wherein avoidance increases long-term distress (e.g., Kumpula et al., 2011). (H3) EA
was expected to be strongly connected to cluster C (avoidance symptoms) as well, given their theoretical overlap. (H4) Inaction and lack of contact with values were expected to be connected to cluster D symptoms, based on evidence linking behavioral activation to reduced symptoms of depressed mood (Dimidjian et al., 2006). (H5) CF was predicted to relate to cluster D symptoms as well, given evidence of the cross-sectional and longitudinal role of CF in depressive symptoms (e.g., Cookson et al., 2020). (H6) Lack of contact with the present moment was expected to be connected to multiple PTSD symptoms, including avoidance, anhedonia, and blame per the findings of King et al. (2013). Lastly, in the functional contextualist DAG network, (H7) EA was expected to be the most influential etiological variable, given its strong conceptual overlap with core symptoms of PTSD (i.e., avoidance).

Method

Participants

Participants \((N = 999)\) were recruited through Amazon’s Mechanical Turk (MTurk), an online platform in which individuals can participate in research for monetary compensation. Evidence suggests that MTurk data are of high quality (Buhrmester et al., 2011; Paolacci et al., 2010; Shapiro et al., 2013) and that participants are more diverse than undergraduate samples (Buhrmester et al., 2011). Quality control methods were employed (i.e., using participants with at least 95% approval ratings from requesters and at least 50 previous human intelligence tasks; Peer et al., 2014). Participants were also required to be fluent in English, to reside in the United States, and to be at least 18 years old. Participants were compensated $1.75 for completing the battery of questionnaires, an amount consistent with MTurk studies of similar length (Fergus & Dolan, 2014). Power analyses for network analysis do not yet exist, but it is generally recommended that the number of participants exceed the number of parameters (Epskamp et al.,
Therefore, a sample of at least 400 individuals who had experienced a Criterion A event was needed.

Criterion A was assessed using a method outlined by Bardeen and Benfer (2019) in which all endorsed events were considered for Criterion A status. Based on this procedure, 277 individuals were removed. Of those, 226 did not experience a Criterion A traumatic event per its current definition (American Psychiatric Association [APA], 2013), and 51 provided narratives consistent with computer-generated responses (e.g., text copied from a website; following guidance by Yarrish et al., 2019). The final sample of 722 participants (53.5% female) had an average age of 37.02 years ($SD = 11.17$; range: 18-64). The majority of the sample reported their race as White (72.7%), followed by Black or African-American (16.3%), Asian (4.8%), Middle Eastern or North African (0.3%), and Native Hawaiian or other Pacific Islander (0.1%). Three percent identified as an unlisted race, and the majority of the sample (86.7%) identified themselves as not Hispanic or Latino/a.

**Self-Report Measures**

*Life Events Checklist for DSM-5 Extended Version (LEC-5; Weathers, Blake et al., 2013)*

The LEC-5 is a self-report measure of lifetime exposure to traumatic events. Participants indicated whether they experienced each of 17 potentially traumatic events through direct exposure, witnessing the event, learning about the event, or experiencing the event as part of their job. Participants provided narrative descriptions and responded to clarification questions for each event endorsed in order to accurately capture exposure to a Criterion A event (Bardeen & Benfer, 2019).

*PTSD Checklist for DSM-5 (PCL; Weathers, Litz et al., 2013)*
The PCL is a 20-item measure of *DSM-5* PTSD symptoms that fall into four symptom clusters: criteria B (intrusions), C (avoidance), D (negative alterations in cognitions and mood), and E (alterations in arousal and reactivity; APA, 2013). Participants indicated the extent to which they have been bothered by each symptom in the past month on a scale of 0 (*Not at all*) to 4 (*Extremely*). Higher scores indicate greater PTSD symptoms. As per Bardeen and Benfer (2019), participants responded to the PCL in a trauma-general fashion (i.e., without respect to an index trauma). The PCL has demonstrated adequate psychometric properties, including internal consistency, and convergent and discriminant validity (Blevins et al., 2015).

**Multidimensional Psychological Flexibility Inventory (MPFI; Rolffs et al., 2018)**

The MPFI is a 60-item measure that assesses the six domains of psychological flexibility and the six domains of psychological inflexibility. Analyses within the current study used only the 30 items reflecting the core components of inflexibility (lack of contact with the present moment, lack of contact with values, inaction, self-as-content, cognitive fusion, and experiential avoidance), as described in ACT (Hayes et al., 1999). Respondents indicated how much they have experienced aspects of psychological inflexibility in the past two weeks on a scale of 1 (*Never true*) to 6 (*Always true*), with higher scores indicating greater levels of the measured construct. The inflexibility scales of the MPFI have demonstrated adequate psychometric properties, including internal consistency (Dubler, 2018), construct validity (Rolffs et al., 2018), and sensitivity to change during ACT treatment (Dubler, 2018).

**Posttraumatic Maladaptive Beliefs Scale (PMBS; Vogt et al., 2012)**

The PMBS is a 15-item measure comprised of three subscales: Threat of Harm (e.g., “The world is very dangerous”), Self-Worth and Judgment (e.g., “I have lost respect for myself”), and Reliability and Trustworthiness of Others (e.g., “Most people are basically caring”). Items are
rated on a scale of 1 (*Not at all true of you*) to 7 (*Completely true of you*), with higher scores indicating greater maladaptive beliefs. The PMBS subscales have demonstrated adequate internal consistency and good concurrent and criterion validity (Fergus & Bardeen, 2017; Shiperd & Salters-Pedneault, 2018; Vogt et al., 2012).

**Data Analytic Plan**

Main analyses were conducted in R using RStudio (Version 1.1.419, 2009-2016). Four networks were estimated: two GGM networks and two DAG networks. The two GGM networks included the PCL symptoms and either the MPFI inflexibility items (referred to as the functional contextualist network) or the PMBS cognitive items (referred to as the cognitive network). The two DAG networks included the same respective measures. For all four networks, the conceptual variables (either MPFI items or PMBS items) were collapsed into subscale nodes to maximize statistical power. Code for the current study analyses is included in supplementary material.

**Gaussian Graphical Model Networks**

For the two GGM networks, the graphical LASSO (i.e., *lasso*; least absolute shrinkage and selection operator) was utilized, which shrinks trivial edges to zero (Knefel et al., 2016). Specifically, we used the EBIC model selection with a hyperparameter gamma of 0.5. The estimated networks utilized the Fruchterman-Reingold algorithm to aid in the visual interpretation of the network (Fruchterman & Reingold, 1991). The *qgraph* package was used to estimate the GGM networks.

**Network Inference.** In addition to providing a visual network, a quantitative centrality metric of expected influence was calculated. Expected influence sums edge weights while maintaining the positive/negative sign of each edge in order to account for the potential presence
of negative edges (Robinaugh et al., 2016). Variables with higher expected influence are more influential in the overall network.

**Bridge Influence.** Bridge influence was calculated for each GGM network. First, two clusters of variables, termed communities, were specified: the PTSD symptoms and the conceptual variables (e.g., inflexibility variables in the functional contextualist network). These communities were determined based on the constructs they belong to, rather than derived from a community detection approach. Next, similar to the use of expected influence metric, the sum of edge weights (maintaining sign of each edge) from one node to all other nodes outside of its assigned community was calculated. For example, EA bridge influence is calculated as the sum of edges from EA to PTSD symptoms; the higher the bridge influence score, the more influential EA is in connecting to PTSD symptoms (Jones et al., 2019; Ross et al., 2018).

**Network Accuracy and Stability.** Network stability was calculated to determine the reliability of estimated networks (Epskamp et al., 2018; Fried & Cramer, 2017). The `bootnet` package was used to calculate bootstrapped edge weights and associated confidence intervals (CIs). Edges with 95% CIs that include zero are not reliably different from other edges in the network. Additionally, correlation stability (CS)-coefficients were calculated for expected influence. CS-coefficients indicate the proportion of cases that can be removed while still retaining a relatively high correlation (i.e., .70) with the original strength metrics. CS-coefficients above .25 are acceptable (Epskamp et al., 2018). Finally, bootstrapped difference tests were conducted to identify significantly different edge weights, expected influence estimates, and bridge expected influence estimates.

**Directed Acyclic Graphs**
DAG networks were computed using the \textit{bnlearn} package and \textit{hill-climbing} algorithm, which continuously adds, removes, and reverses direction of edges until optimal fit per the Bayesian information criterion (BIC) is achieved (McNally, Heeren, et al., 2017). Given that the cognitive and functional contextualist variables are conceptualized as risk factors for, rather than consequences of, PTSD, the DAG was originally specified such that PCL variables could not precede risk factors (i.e., cognitive and functional contextualist variables) in the model. To ensure network stability, a two-step process was used. First, only edges that were present in 85% of the 10,000 bootstrapped networks (based on Sachs et al., 2005) were retained. Second, the direction of each edge was estimated. If the same direction of an edge was present in 51% or more of the 10,000 bootstrapped networks, that directional edge was retained (McNally, Heeren et al., 2017; McNally, Mair et al., 2017). The DAG network was estimated in such a way that the edge thickness represents directional probability, meaning that a thicker edge depicts a higher probability that a directional relationship exists. The further upstream a variable in the DAG is (i.e., toward the top of the network), the greater influence it has on the overall network. Therefore, etiological variables that had the greatest downstream effects were deemed to be the most influential.

\textbf{Results}

\textbf{GGM Networks}

\textit{Network Estimation}

Data were normally distributed (skew range = -0.1 – 1.2; kurtosis range = 1.7 – 3.2; Byrne et al., 2010; Curran et al., 1996). The visual depiction of the cognitive and functional contextualist GGM networks are illustrated in Figure 1\textsuperscript{1}. See Table 1 and Supplemental Table 1 for variable labels and descriptive statistics, respectively. For both networks, PTSD symptoms
within each cluster generally clustered with one another, with the exception of cluster E symptoms, which appeared to have clustered in pairs (more so in the cognitive network).

Additionally, trauma-related amnesia (PCL8) did not cluster with other cluster D symptoms in the functional contextualist network. Overall, in the cognitive network there were 81 non-zero edges, the vast majority of which (76 edges) were positive. The three strongest edges in the cognitive GGM network were between: avoidance of trauma-related thoughts and avoidance of reminders (0.53), hypervigilance and startle (0.48), and reliability and trustworthiness of others and self-worth and judgment (0.39). These edges were significantly different from nearly all other edges in the network (supplementary Figure S1a). In the functional contextualist network, there were 82 non-zero edges, the vast majority of which (79 edges) were positive. The three strongest edges in the functional contextualist GGM network were avoidance of trauma-related thoughts and avoidance of reminders (0.52), hypervigilance and startle (0.50), and inaction and lack of contact with values (0.46). These edges were significantly different from nearly all other edges in the network (supplementary Figure S2a). Overall, network estimation was similar across networks.

**Network Inference and Bridge Symptoms**

A graph depicting node expected influence for the cognitive and functional contextualist GGMs is located in the supplementary material (Figure S3). In the cognitive network, PMBS-Threat had the highest bridge expected influence of the cognitive variables, and its bridge expected influence was significantly different from all other variables in the network (supplementary Figure S1b). Of the PCL variables, strong negative beliefs (PCL9) and feeling distant (PCL13) had the highest bridge expected influence. However, it is important to note that these values were only significantly different from one other PCL variable—engagement in risky
behaviors (PCL16; see Figure S1b). This reflects the small magnitude of bridge expected influence for PCL9 and PCL13. Thus, the hypothesis that cluster D symptoms would bridge between cognitive and PTSD variables was supported, but should be interpreted with caution, given the small magnitude of bridge expected influence for these variables.

In the functional contextualist network, EA and lack of present moment awareness (NotPres) had the highest bridge expected influence (nearly equal values) of the functional contextualist variables. Five of the six MPFI variables acted as bridges to the PCL variables (all but self-as-content [SelfCont]), indicating a complex relationship amongst variables. Of the PCL variables, trouble concentrating (PCL19) had the highest bridge expected influence. Bridge expected influence for both GGM networks is depicted in the supplementary material (Figure S4). Significant difference testing revealed that none of these variables had bridge expected influence values that were significantly different from most other variables in the network, with a few exceptions (see Figure S2b). Similar to the GGM, the magnitude of the bridge expected influence may have been too small to be significantly different, and thus, bridge expected influence results should be interpreted with caution.

Contrary to study hypotheses, EA (MPFI-EA) did not have any direct connections to intrusion symptoms (cluster B). However, consistent with predictions, EA shared a strong association with avoidance of trauma-related thoughts and memories (i.e., one of two avoidance symptoms; cluster C). This was the only bridge by which EA was associated with PTSD symptoms. Partially consistent with predictions, CF (MPFI-CF) was associated with strong negative beliefs (one of seven cluster D symptoms); however, lack of contact with values (MPFI-NVl) and inaction (MPFI-Inc) were not directly associated with any cluster D symptoms. Finally, lack of contact with the present moment (MPFI-NtP) was not associated with the
predicted PTSD symptoms of avoidance, anhedonia, or blame. Instead, it was only associated with trouble concentrating (PCL19).

**Network Accuracy and Stability**

Bootstrapped results of the cognitive GGM network indicated that 35 of the 81 non-zero edges were truly non-zero (i.e., 95% confidence intervals did not include zero; supplementary Figure S5). Graphs depicting significant differences across cognitive network metrics (i.e., edge weights, expected influence, and bridge expected influence) are depicted in Figure S1a-b. In the functional contextualist GGM network, 40 of the 82 non-zero edges were truly non-zero (Figure S5). Graphs depicting significant differences across functional contextualist network metrics (i.e., edge weights, expected influence, and bridge expected influence) are depicted in Figure S2a-b. The CS-coefficient for expected influence in both networks exceeded the 0.25 threshold put forth by Epskamp et al. (2018; cognitive network = .44, functional contextualist network = .44). Graphs of CS-coefficients for expected influence for both GGM graphs are depicted in supplementary Figure S6. Overall, the networks were considered stable and interpretable.

**DAG Networks**

Originally, the DAG models were specified such that the PCL variables could not precede cognitive and functional contextualist variables (e.g., no PCL variable would flow to a cognitive variable). This was proposed because (1) based on theory, the cognitive and functional contextualist variables are risk factors and thus should temporally precede PTSD symptoms and (2) it would simplify comparative interpretation across the cognitive and functional contextualist networks. However, the cognitive network was only partially directed using this specification, such that not all nodes could be connected through directional arcs, using the data. Therefore, this specification was removed for the cognitive model. The proposed model did compute for the
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The results of the DAG networks, without the directional specifications for the cognitive network, and with the directional specifications for the functional contextualist network, are depicted in Figure 2.

In the cognitive DAG network, unwanted memories (PCL1) and blame (PCL10) were the only variables not acted upon by another variable in the network. As mentioned above, PTSD symptoms had downstream effects on the cognitive variables, rather than the reverse. More specifically, feeling distant (PCL13) and hypervigilance (PCL17) led to maladaptive beliefs about threat (PMBS-Threat), which then led to maladaptive beliefs about self-worth and judgment (PMBS-SWJ) and reliability and trustworthiness of others (PMBS-RTO). The three strongest edges (i.e., highest probability that directionality exists) were from: threat (PMBS-Threat) to reliability and trustworthiness of others (PMSBS-RTO), feeling distant (PCL13) to threat, and hypervigilance (PCL17) to startle (PCL18).

In the functional contextualist DAG, four edges bridged the constructs. EA (MPFI-EA) had direct downstream effects on avoidance of trauma-related thoughts and memories (PCL6), lack of present-moment awareness (MPFI-Not Pres) had downstream effects on trouble concentrating (PCL19), and lack of contact with values (MPFI-No Values) had downstream effects on risky behaviors (PCL16). The DAG also illustrates the important role of blame (PCL10), unwanted memories (PCL1), strong negative feelings (PCL11), and lack of contact with values (MPFI-No Values) in the co-occurrence of PTSD symptoms and psychological inflexibility, as evidenced by these variables appearing at the top of the network. Contrary to predictions, EA was not the most influential variable in the DAG network, as determined by its position in the network; lack of contact with values was the most influential. However, the edge from EA to avoidance of trauma-related thoughts and memories was one of the thickest in the
DAG, indicating greater probability that this directional relationship would exist with longitudinal data. The other two strongest edges were from lack of present-moment awareness (MPFI-Not Pres) to trouble concentrating (PCL19) and lack of contact with values (MPFI-No Values) to engagement in risky behaviors (PCL16).

**Discussion**

The purpose of the current study was to examine, via network analysis, how PTSD symptoms relate to risk and maintenance factors from two theoretical conceptualizations of PTSD: the cognitive model and the functional contextualist model. As predicted, in the cognitive GGM network, cluster D PTSD symptoms acted as a bridge between PTSD symptoms and cognitive variables. Specifically, strong negative beliefs and feeling distant were strong bridges to cognitive variables. Also in partial support of predictions, in the functional contextualist GGM network, EA shared a strong association with avoidance of trauma-related thoughts and memories, but not trauma-related external reminders. Contrary to hypotheses, EA was not uniquely associated with any of the intrusion symptoms. It might be that EA is only directly related to avoidance of trauma-related thoughts and memories, and that all other associations are indirect (i.e., mediated) via this relationship. This appears to be probable based on existing results, but it needs to be tested in longitudinal designs. The most surprising finding was that the data did not fit the theoretical assumption for the cognitive DAG model that maladaptive cognitive beliefs precede PTSD symptoms. However, the data did not fit this assumption. That is, results suggested that it is more likely that PTSD symptoms temporally precede cognitive variables, than vice versa.

Interpretation of the GGM and DAG in parallel may help generate hypotheses about directional relationships among study variables. The cognitive GGM network revealed several
potential feedback loops that should be subject to testing (e.g., using longitudinal research
designs). First, maladaptive beliefs about threat, strong negative beliefs, strong negative feelings,
and feeling distant were all associated with one another. Turning to the DAG, the results suggest
that if the symptom of strong negative feelings is activated, this loop is likely to become
activated. In the DAG, the cascading order of symptom flow is from strong negative feelings to
strong negative beliefs and feeling distant, which then leads to maladaptive beliefs about threat.
The DAG must be acyclic (i.e., no feedback loops), and thus, maladaptive beliefs cannot
maintain or exacerbate PTSD symptoms in this model. However, based on the associations
observed in the GGM, it may be that maladaptive beliefs about threat reinforces (i.e., leads back
to) any one of those symptoms to generate a reinforced feedback loop. Mood-related symptoms
(i.e., strong negative beliefs, strong negative feelings) may lead individuals to retreat from their
relationships, thus limiting contact with typical activities. This type of behavioral avoidance may
then, in turn, reinforce a belief that staying isolated from others reduces threat (i.e., maladaptive
belief about threat). Such a feedback loop is consistent with the emotional processing theory,
which posits that avoidance maintains maladaptive beliefs about threat (see Rauch & Foa, 2006
for a review).

A second potential feedback loop emerged in the cognitive GGM between maladaptive
beliefs about threat, hypervigilance, and startle. The DAG suggests that hypervigilance may lead
to both startle and maladaptive beliefs about threat. Given the associations observed in the GGM,
either maladaptive beliefs about threat or startle reinforces (1) hypervigilance and (2) either
maladaptive beliefs about threat or startle. The vigilance-avoidance hypothesis of cognitive
biases that are thought to underlie anxiety and fear-related disorders (e.g., social anxiety disorder
[Vassilopoulos, 2005], specific phobias [Pflugshaupt et al., 2005]) and PTSD [see Bardeen, 2020]
for a review]) provides a framework for a chain in which reflexive orienting of attention toward threat (i.e., hypervigilance) increases sympathetic nervous system arousal (e.g., enhanced startle; Weierich et al., 2008). According to this hypothesis, following initial threat detection, fearful individuals quickly disengage and shift attention away from threat stimuli to reduce the increased physiological arousal associated with attending to such stimuli, which, over time, reinforces maladaptive beliefs about threat. These beliefs may then reinforce alterations in arousal and reactivity (e.g., startle, hypervigilance) over time, and thus, maintain this potential feedback loop. In support of this possible feedback loop, in a post-hoc DAG (described below; Supplementary Figure 4), maladaptive beliefs about threat led to cluster E symptoms (i.e., alterations in arousal and reactivity, such as hypervigilance). Taken together, there may be a reciprocal relationship between hypervigilance, startle, and maladaptive beliefs about threat.

There were no potential feedback loops observed that included maladaptive beliefs about self-worth and judgment or reliability and trustworthiness of others, which is consistent with their low bridge expected influence and their positioning at the very bottom of the DAG. That is, they do not appear to be directly influential on any PTSD symptoms. This finding may be surprising at first, given that much of CPT focuses on targeting beliefs in these two areas. However, the results do not suggest that they are unfruitful targets; rather, targeting maladaptive beliefs about threat may directly impact changes in maladaptive beliefs about self-worth and judgment and reliability and trustworthiness of others. This is consistent with the order in which these theme areas are targeted in CPT. Beliefs about safety (directly related to beliefs about threat) are addressed first, followed by beliefs about trust, power/control, and finally, esteem (Resick et al., 2017). Thus, the ordering of CPT appears to acknowledge that targeting beliefs about safety/threat first is important.
Results from the functional contextualist models provided several potential directional relationships to explain the co-occurrence of psychological inflexibility and PTSD symptoms. Overall, there appear to be three salient bridges by which these constructs co-occur. The first bridge operates via the link between EA and avoidance of trauma-related thoughts, memories, and feelings. This relationship appeared influential and strong in both the GGM and DAG. In the functional contextualist DAG, a high probability edge flowed from EA to avoidance of trauma-related thoughts and memories. This is consistent with empirical evidence that pre-trauma EA predicts PTSD symptoms following a traumatic event (Kumpula et al., 2011). It appears, from the DAG, that avoidance of thoughts and feelings associated with the trauma spreads activation to avoidance of reminders (per GGM and DAG). Results of the DAG suggest that, in addition to activation from EA, avoidance of trauma-related thoughts and memories is also activated by intrusion symptoms (i.e., feeling upset at reminders [direct relationship], unwanted memories [indirect relationship via feeling upset at reminders]). The GGM further supports an association between avoidance symptoms (cluster C) and intrusion symptoms (cluster B). Taken together, it appears that a combination of global EA (a pre-trauma risk factor) and intrusion symptoms trigger trauma-related avoidance. Despite this potentially important relationship bridging the two constructs, EA was not highly central in the overall network. It may be that the EA subscale is separate, and perhaps distinct, from the other subscales of the MPFI, which appear to be more closely associated (see GGM). The strong associations between the other five subscales of the MPFI may account for them being more central, and having greater influence, compared to EA.

The remaining bridges included relationships between the other highly interconnected inflexibility constructs and cluster D and E symptoms. Although lack of contact with the present moment was important in these remaining bridges, it did not bridge to any of the predicted PTSD
symptoms (avoidance, anhedonia, blame). These relationships were hypothesized based on a mindfulness treatment that led to reductions in these specific PTSD symptoms (King et al., 2013). It is possible that though engaging in more mindful awareness improves these symptoms, less mindful awareness is not necessarily positively associated with these symptoms pre-treatment. Lack of present-moment awareness, did, however, share associations with trouble concentrating and inaction; associations among these three variables in the GGM suggest a potential feedback loop, and the functional contextualist DAG network suggests that lack of contact with the present moment activates trouble concentrating. It may be that a person who, pre-trauma, has difficulty staying present is more likely to become distracted by trauma-related intrusions and therefore exhibits trouble concentrating. The relationship between inaction and these variables is less clear. Though they are connected in the GGM, inaction is not directly related to these variables in the DAG. Per the DAG, lack of present-moment awareness might activate trouble concentrating and, consistent with the association in the GGM, is related to a lack of clarity in values, which results in inaction. Trouble concentrating then generates a feedback loop (given association in GGM) by activating inaction. A directional relationship from trouble concentrating to inaction makes theoretical sense. A person who has trouble concentrating may not be able to consistently and effectively engage in valued action. However, it is important to note that this interpretation is speculative and makes assumptions that cannot be fully supported by the results in the present study. Therefore, the relationships among inaction, lack of present-moment awareness, lack of clarity in values, and the PTSD symptom of trouble with concentration should be subject to further testing. Although it is unclear how these facets of psychological inflexibility are related to trouble concentrating, trouble concentrating does appear to be an important bridge between constructs.
The final bridge within the functional contextualist networks involved engagement in risky behaviors and lack of contact with values (per the GGM and DAG). Results from the DAG suggest a strong probability that lack of contact with values precedes engagement in risky behaviors. For example, individuals who are not clear in their values (e.g., health, spending time with loved ones) may be more likely to engage in behaviors that put themselves in danger (e.g., drug use, excessive gambling). Alternatively, if someone is very clear in their values, they may be able to experience enough positive reinforcement from engaging in valued action (for which clarity of values is a pre-requisite) that it outweighs the short-lived reinforcement that may be received from engaging in risky behaviors. In support of this hypothesis, clarity of values appears to be a means of reducing risky behaviors such as substance use (Meyer et al., 2018) and non-suicidal self-injury (Cameron et al., 2014).

It is worth noting that blame, strong negative feelings, and unwanted memories were at the top of both DAG networks, suggesting they contribute to the most downstream effects on other variables. A study of traumatized children and adolescents found that cluster D symptoms and unwanted memories similarly emerged at the top of a DAG of PTSD symptoms (Bartels et al., 2019). Given preliminary evidence from this study as well as Bartels et al. (2019), the potentially causal role of blame, strong negative feelings, and unwanted memories in the development of PTSD should be examined in future longitudinal studies.

The most surprising result from the present study was that the proposed risk factors did not precede the PTSD symptoms in the cognitive DAG network. One potential explanation is that the PTSD symptoms were dominant in the network due to there being more variables (20 compared to three cognitive variables). To test this post-hoc explanation, PCL variables were collapsed into clusters (i.e., clusters B-E), and the DAG was re-estimated. Results were generally
unchanged (supplementary Figure S7). PCL symptoms preceded two of the three cognitive variables—maladaptive beliefs about self-worth and judgment, and reliability and trustworthiness of others. However, maladaptive beliefs about threat led to alterations in arousal and reactivity. This is consistent with previous research which demonstrated that beliefs about threat contribute to both the onset and maintenance of PTSD symptoms (Dunmore et al., 1999). Still, the pattern of results does not appear to be fully explained by a difference in number of nodes, as most PTSD symptom clusters still preceded two of the three cognitive variables in the post-hoc analysis.

This finding is puzzling, given that it appears to conflict with the cognitive model of PTSD. According to the cognitive model, maladaptive posttraumatic cognitions (i.e., assimilated and overaccommodated beliefs) lead to the persistence of thoughts and behaviors that develop into PTSD. Additional research is warranted to clarify the trajectory of maladaptive posttraumatic cognitions in relation to PTSD symptoms. In contrast, functional contextualist models of PTSD highlight the potential role of psychological inflexibility leading to PTSD symptoms (Blackledge, 2004), rather than the reverse directional relationship. The results from the current study are consistent with longitudinal evidence that EA serves as a pre-trauma risk factor for the development of PTSD symptoms (Kumpula et al., 2011).

Study findings should be interpreted in light of limitations. Though DAG models are growing in popularity in psychological research (Bartels et al., 2019; Kuipers et al., 2019; McNally, Heeren, et al., 2017; McNally, Mair et al., 2017), these findings should not be interpreted as inferring causality. DAGs provide an important step toward generation of causal hypotheses, beyond what other methods are able to do, but inferences of causality are only indisputable when all confounds have been measured (Moffa et al., 2017). The networks
estimated in this study do not comprise a full set of likely relevant etiological factors. Related to this point, DAG models are based on the assumption that the collected data was generated from a causal model (Jones et al., 2018). Data for the present study were collected almost exclusively from participants whose index events occurred months to years prior to study completion. As such, the nature of relations among study variables does not represent a critical period for understanding the development of the disorder (i.e., in the acute aftermath of the trauma). Thus, caution is warranted in drawing causal inferences from the results of this study. Finally, though stability was adequate, it fell just below recommended thresholds (i.e., CS greater than .50; Epskamp et al., 2018). The sample size may have not been large enough to produce ideal stability. While some guidelines suggest that one participant per parameter is sufficient to have adequate power, others recommend using structural equation modeling guidelines of upwards of 10 participants per parameter (Schreiber et al., 2006). In sum, it will be important to replicate study findings with a larger sample, using longitudinal methods, including shortly after traumatic events.

In spite of limitations, this study provides an important first step in employing network analysis to examine two conceptual models of PTSD—the cognitive and functional contextualist models. Use of network analysis serves to address some of the limitations of traditional methods (e.g., latent variable modeling) in generating testable hypotheses about how PTSD develops and is maintained under these two frameworks. Results from this study suggest that PTSD symptoms develop prior to maladaptive posttraumatic beliefs in the cognitive model. Maladaptive beliefs about threat may be a particularly important treatment target that impacts other maladaptive beliefs (i.e., about self-worth and judgment and trustworthiness of others), which in turn, may affect PTSD symptoms. In the functional contextualist model, results suggest that psychological
inflexibility may play a potential causal role in the development of PTSD symptoms. There were several bridges that explained the co-occurrence of psychological inflexibility and PTSD, giving rise to several possible causal chains that should be further examined using longitudinal research designs, including bridging relationships between EA and trauma-related avoidance of thoughts and memories, between lack of contact with values and engagement in risky behaviors, and among lack of contact with the present, trouble concentrating, and inaction. These findings should be considered as initial evidence for formulating causal hypotheses about PTSD etiology under the cognitive and functional contextualist models.
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Using directed acyclic graphs in epidemiological research in psychosis: An analysis of the role of


Footnote

1A dense network (i.e., more edges) was originally selected in which small edges may represent false positives. Dense networks can be unstable and inaccurate. As such, a thresholding rule in which edge-weights are required to be larger than those in the final model and EBIC computation models was employed (i.e., argument threshold = TRUE in qgraph package). Thus, the network may favor specificity over sensitivity. While there may be true edges missing from the graph, the edges that are present are more likely to be true (Epskamp et al., 2018).
Funding statement: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.
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<tr>
<th>Abbreviation in Network</th>
<th>Full Variable Name</th>
<th>Associated Construct</th>
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<td>1</td>
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<td>2</td>
<td>Dreams</td>
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<td>Strong Negative Beliefs</td>
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<td>Blame</td>
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<td>Reliability and Trustworthiness of Others</td>
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<td>PMBS-SWJ</td>
<td>Self-worth and Judgment</td>
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<td>MPFI-Inc/Inaction</td>
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<td>MPFI-EA/EA</td>
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Figure 1

Cognitive GGM (Left) and Functional Contextualist GGM (Right) Networks

Note. See Table 1 for label names. Solid lines represent positive associations and dashed lines represent negative associations. Thicker lines indicate stronger associations.
Figure 2

Directed Acyclic Graphs for Cognitive (Left) and Functional Contextualist (Right) Networks

Note. Cognitive graph is without model specification and functional contextualist is with model specification) networks. Thicker lines indicate greater probability of directionality. See Table 1 for label names.
Table S1.

**Descriptive Statistics and Zero-Order Correlations of Study Variables**

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<th>Variable</th>
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<td>3. MPFI-SelfCont/SIC</td>
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<td>.72</td>
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*Significant at p < .05
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</table>

Note. *denotes correlation was not significant at \( p < .01 \); all other correlations were significant at \( p < .01 \). MPFI = Multidimensional Psychological Flexibility Inventory; EA = Experiential Avoidance subscale; Not Pres/NtP = Lack of Present Moment Awareness subscale; SelfCont/SIC = Self-as-Context subscale; CF = Cognitive Fusion subscale; NVl/No Values = Lack of Contact with Values subscale; Inact/Inaction = Inaction subscale; PMBS = Posttraumatic Maladaptive Beliefs Scale; Thr/Threat = Threat of Harm subscale; SWJ = Self-Worth and Judgment subscale; RTO = Reliability and Trustworthiness of Others subscale; PCL = PTSD Checklist for DSM-5 total score.
CONCEPTUAL APPROACHES TO ETIOLOGY OF PTSD

Figure S1a

*Bootstrapped Difference Tests for Non-Zero Edges in Cognitive GGM*

Note. α = .05; black boxes indicate significant differences and light gray boxes indicate non-significant differences. See Table 1 for variable names.
CONCEPTUAL APPROACHES TO ETIOLOGY OF PTSD

Figure S1b

*Bootstrapped Difference Tests for Bridge Expected Influence (Left) and Expected Influence (Right) in the Cognitive GGM*

Note. $\alpha = .05$; black boxes indicate significant differences and light gray boxes indicate non-significant differences. See Table 1 for variable names.
CONCEPTUAL APPROACHES TO ETIOLOGY OF PTSD

Figure S2a

*Bootstrapped Difference Tests for Non-Zero Edges in Functional Contextualist GGM*

*Note.* $\alpha = .05$; black boxes indicate significant differences and light gray boxes indicate non-significant differences. See Table 1 for variable names.
Figure S2b

*Bootstrapped Difference Tests for Bridge Expected Influence (Left) and Expected Influence (Right) in the Functional Contextualist GGM*

*Note.* $\alpha = .05$; black boxes indicate significant differences and light gray boxes indicate non-significant differences. See Table 1 for variable names.
Figure S3

One-Step Expected Influence for Cognitive GGM Network (Left) and Functional Contextualist GGM Network (Right)

Cognitive Graph

Functional Contextualist Graph

Note. See Table 1 for label names.
Figure S4

One-Step Bridge Expected Influence for Cognitive GGM Network (Left) and Functional Contextualist GGM Network (Right)

Cognitive Graph

Functional Contextualist Graph

Note. See Table 1 for label names.
CONCEPTUAL APPROACHES TO ETIOLOGY OF PTSD

Figure S5

*Bootstrapped Confidence Intervals (Gray) for Edge Weights in the Cognitive (Left) and Functional Contextualist (Right) GGM Networks*

Cognitive Graph

Functional Contextualist Graph
**Figure S6**

*Correlations Between Expected Influence Values in the Sampled and Case-Dropped Sample*

Cognitive Graph  
Functional Contextualist Graph

*Note.* Lines represent the average correlation and red regions indicate the 95% confidence interval.
Figure S7

Directed Acyclic Graph for the Cognitive Variables with PTSD Symptoms Collapsed into Clusters

Note. Thicker lines indicate greater probability of causality. Intrs = intrusions (cluster B), NegAlt = negative alterations in cognitions and mood (cluster C), Avoid = avoidance (cluster C), Hypervig = alterations in arousal and reactivity (cluster E), Threat = threat of harm, RTO = reliability and trustworthiness of others, SWJ = self-worth and judgment. Dashed lines indicate non-significant edges.
CONCEPTUAL APPROACHES TO ETIOLOGY OF PTSD

# Supplementary Code

```r
library(psych)
library(lavaan)
library(qgraph)
library(glasso)
library(bootnet)
library(huge)
library(networktools)
library(huge)
library(bnlearn)
library(ggplot2)

## import data
#setwd(masked)
data <- read.csv(masked)
attach(data)

# Cognitive Graph Network
CPTdata <- data[c(25:44, 18:20)]

CPTcor <- cor_auto(CPTdata)
EBIC_CPT <- EBICglasso(CPTcor, n = 722, threshold = TRUE)
clustersCPT <- list("B" = c(1:5), "C" = c(6:7), "D" = c(8:14), "E" = c(15:20), "PMBS" = c(21:23))
CPTcommunities <- c(rep("PTSD", 20), rep("CPT", 3)) # identify communities as PCL and PMBS groups

# Black and white graphs
CPT_NetworkGraphBW <- qgraph(EBIC_CPT, layout = "spring", theme = "gray", title = "Cognitive Graph", legend.cex = .35)
pdf(file = "CPT network BW.pdf") # Fig 1
plot(CPT_NetworkGraphBW)
dev.off()

# Examine highest edge weights
CPT_NetworkGraphBW$Edgelist[['weight']]
CPT_NetworkGraphBW$Edgelist[['from']]
CPT_NetworkGraphBW$Edgelist[['to']]

# Centrality; only using expected influence given negative edges
CPTexpecInf <- expectedInf(CPT_NetworkGraphBW) # 1 and 2 step look similar so we only report 1-step
plot(CPTexpecInf$step1)
ggsave("CPT expec inf 1 step.png") # Fig S3
```
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#examine bridge influence
CPTcommunity_structure<-c(rep("PTSD",20), rep("PMBS",3))
CPTbridge<--bridge(CPT_NetworkGraphBW, communities=CPTcommunity_structure, useCommunities = "all",
  directed = NULL, nodes = NULL)
CPT_plot_bridge<-plot(CPTbridge, include="Bridge Expected Influence (1-step)"
plot(CPT_plot_bridge)
ggsave("CPT_bridgecentrality.png")  #Fig S4

##Stability [using method/code from McNally, Mair, Mugno, & Riemann, 2017 suppl code]
####bootstrap glasso network
glassofit2<--estimateNetwork(CPTdata, default="EBICglasso",threshold=TRUE)

#####edge weight accuracy
set.seed(123)
glassoboot1<--bootnet(glassofit2, nBoots=1000,nCores = 4,statistics =
c("edge","expectedInfluence","bridgeExpectedInfluence", "strength"),communities =
CPTcommunity_structure)
summary(glassoboot1)
plot(glassoboot1, labels = FALSE, order = "sample", statistics = "edge") #plot 95% CI for edge strengths
ggsave("CPT stability.png") #Fig S5

####difference tests
plot(glassoboot1, "edge", plot = "difference", onlyNonZero = TRUE, order = "sample",theme(axis.text.x =
element_text(size=20), axis.text.y=element_text(size=20))
ggsave("CPT edge weight diff.pdf", width=15, height=15, units="in") #Fig S2a
plot(glassoboot1,"expectedInfluence", plot="difference")
ggsave("CPT expec infl diff.png") #Fig S2b
plot(glassoboot1,"bridgeExpectedInfluence", plot="difference")
ggsave("CPT bridge expec infl diff.png") #Fig S2b

#dark gray boxes represent significant differences; light gray boxes represent non-significant differences

#####corstability for EI
corstabCPT<--bootnet(glassofit2, nBoots=1000, type="case", nCores=8, statistics = "expectedInfluence")
CScoeff<--corStability(corstabCPT) #CS for EI = .44
plot(corstabCPT, statistics="expectedInfluence")
ggsave("CPT cor stab EI.png") #Fig S6

informatics ACT GGM NETWORK

ACTdata<-data[c(25:44, 12:17)]
names(ACTdata)<-c("PLC1", "PLC2", "PLC3", "PLC4", "PLC5", "PLC6", "PLC7", "PLC8",
  "PLC9", "PLC10", "PLC11", "PLC12", "PLC13", "PLC14", "PLC15", "PLC16", "PLC17",
  "PLC18", "PLC19", "PLC20", "EA", "Not Pres", "SelfCont", "CF", "No Values", "Inaction")
clustersACT<--list("B"=c(1:5),"C"=c(6,7), "D"=c(8:14), "E"=c(15:20), "MFPI"=c(21:26))
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group_col <- c("#72CF53", "#53B0CF","#FFB026","#ED3939","#CC6ACF")
ACTcor <- cor_auto(ACTdata)
EBIC_ACT <- EBICglasso(ACTcor, n = 722, gamma = .75, threshold = TRUE)

#black and white graphs
ACT_NetworkGraphBW <- qgraph(EBIC_ACT, layout="spring", theme="gray", title = "Functional Contextualist Graph", legend.cex=.35)
plot(ACT_NetworkGraphBW)

pdf(file="ACT network BW.pdf") #Fig 1
plot(ACT_NetworkGraphBW)
dev.off()

#examine ACT edge weights

ACT_NetworkGraphBW$Edgelist[['weight']]
ACT_NetworkGraphBW$Edgelist[['from']]  
ACT_NetworkGraphBW$Edgelist[['to']]  

#centrality; only using expected influence given negative edges
ACTexpecInf <- expectedInf(ACT_NetworkGraphBW) #1 and 2 step look similar so we only report 1-step
plot(ACTexpecInf$step1)
ggsave("ACT expec inf 1 step.png")

#examine bridge influence

ACTcommunity_structure <- c(rep("PTSD",20), rep("MPFI",6))
ACTbridge <- bridge(ACT_NetworkGraphBW, communities=ACTcommunity_structure, useCommunities = "all", 
                      directed = NULL, nodes = NULL)
ACT_plot_bridge <- plot(ACTbridge, include=("Bridge Expected Influence (1-step)")) #1 step and 2 step look similar so we only report 1 step
  ggsave("ACT_bridgecentrality.png") #Fig S3

#centrality stability
qwerty##bootstrap graph lasso network
ACTglassofit2 <- estimateNetwork(ACTdata, default="EBICglasso",threshold=TRUE)

qwerty###edge weight accuracy
set.seed(123)
ACTglassoboot1 <- bootnet(ACTglassofit2, nBoots=1000,nCores = 4,statistics = c("edge","expectedInfluence","bridgeExpectedInfluence","strength"),communities = ACTcommunity_structure)
summary(ACTglassoboot1)
plot(ACTglassoboot1, labels = FALSE, order = "sample", statistics = "edge") #Fig S5
  ggsave("ACT stability.png")
### difference tests
plot(ACTglassoboot1, "edge", plot = "difference", onlyNonZero = TRUE, order = "sample")
ggsave("ACT edge weight diff.png", width=15, height=15) #Fig S2a
plot(ACTglassoboot1,"expectedInfluence", plot="difference")
ggsave("ACT expec infl diff.png") #Fig S2b
plot(ACTglassoboot1,"bridgeExpectedInfluence", plot="difference")
ggsave("ACT bridge expec infl diff.png") #Fig S2b

### corstab for EI
corstabACT<-bootnet(ACTglassofit2, nBoots=1000, type="case", nCores=8, statistics = "expectedInfluence")
CscoeffACT<-corStability(corstabACT) #CS for EI = .44
plot(corstabACT, statistics="expectedInfluence")
ggsave("ACT cor stab EI.png") #Fig S6

#-------------------------------------------
# used McNally, Heeren, Robinaugh, 2017 supplemental code as a guide; see their code for more details
install.packages("bnlearn")
BiocManager::install("Rgraphviz")
#set variables to numeric
for (i in c(1:23)){
  CPTdata[,i] <- as.numeric(CPTdata[,i])
}

#specify that CPT variables must precede PCL variables
Blacklist <- matrix(c("PCL1","Threat","PCL2","Threat","PCL3","Threat","PCL4","Threat","PCL5","Threat","PCL6","Threat","PCL7","Threat","PCL8","Threat","PCL9","Threat","PCL10","Threat","PCL11","Threat","PCL12","Threat","PCL13","Threat","PCL14","Threat","PCL15","Threat","PCL16","Threat","PCL17","Threat","PCL18","Threat","PCL19","Threat","PCL20","Threat","PCL1","SWJ","PCL2","SWJ","PCL3","SWJ","PCL4","SWJ","PCL5","SWJ","PCL6","SWJ","PCL7","SWJ","PCL8","SWJ","PCL9","SWJ","PCL10","SWJ","PCL11","SWJ","PCL12","SWJ","PCL13","SWJ","PCL14","SWJ","PCL15","SWJ","PCL16","SWJ","PCL17","SWJ","PCL18","SWJ","PCL19","SWJ","PCL20","SWJ","PCL1","TRO","PCL2","TRO","PCL3","TRO","PCL4","TRO","PCL5","TRO","PCL6","TRO","PCL7","TRO","PCL8","TRO","PCL9","TRO","PCL10","TRO","PCL11","TRO","PCL12","TRO","PCL13","TRO","PCL14","TRO","PCL15","TRO","PCL16","TRO","PCL17","TRO","PCL18","TRO","PCL19","TRO","PCL20","TRO"),2,byrow=TRUE)
colnames(Blacklist) <- c("from","to")
Blacklist

#fit Bayesian network; 50 random re-starts, 100 perturbations per restart
set.seed(123)
fitCPT1 <- hc(CPTdata, restart = 50, perturb = 100, blacklist = Blacklist)
fitCPT1

bnlearn::score(fitCPT1, data = CPTdata) # global network score
astr <- arc.strength(fitCPT1,CPTdata, "bic-g") # connection strength
astr[order(astr[,3]), ] # sorted edge strength from strongest to weakest
strength.plot(fitCPT1, astr, shape = "ellipse")

# Bootstrap to stabilize
# Learn 10000 network structures
set.seed(123)
CPTdagboot <- boot.strength(CPTdata, R = 10000, algorithm = "hc", algorithm.args = list(restart = 5, perturb = 10, blacklist = Blacklist), debug = TRUE)
head(CPTdagboot)

# Filter the ones with a strength larger than 0.85 and a direction probability > 0.5
CPTdagbootStrong <- bootnet[which(CPTdagboot$strength > 0.85 & CPTdagboot$direction > 0.5), ]

# Build the average network using a 0.85 threshold (Sachs et al., 2005, Science)
CPTavgnet1 <- averaged.network(CPTdagboot, threshold=.85)  # Graph is only partially directed. Stop here and remove Blacklisting

# Repeat the above, but without Blacklisting
# Fit a first Bayesian network, based on 50 random re-starts and 100 perturbations for each re-start.
set.seed(123)
fitCPT1noB <- hc(CPTdata, restart = 50, perturb = 100)  # hc gives directed graph
fitCPT1noB

bnlearn::score(fitCPT1noB, data = CPTdata)  # Global network score
astrnoB <- arc.strength(fitCPT1noB, CPTdata, "bic-g")  # Connection strength
astrnoB[order(astrnoB[,3]), ]  # Sorted edge strength from strongest to weakest
strength.plot(fitCPT1noB, astrnoB, shape = "ellipse")

# Now we stabilize the network across multiple samples through bootstrapping:
# Learn 10000 network structures
set.seed(123)
CPTdagbootnoB <- boot.strength(CPTdata, R = 10000, algorithm = "hc", algorithm.args = list(restart = 5, perturb = 10), debug = TRUE)
head(CPTdagbootnoB)

# Filter the ones with a strength larger than 0.85 and a direction probability > 0.5
CPTdagbootStrongnoB <- CPTdagbootnoB[which(CPTdagbootnoB$strength > 0.85 & CPTdagbootnoB$direction > 0.5), ]

# Build the average network using a 0.85 threshold (Sachs et al., 2005, Science)
CPTavgnet1noB <- averaged.network(CPTdagbootnoB, threshold=.85)
CPTavgnet1noB
bnlearn::score(CPTavgnet1noB, data = CPTdata)  # Global network score
CPTastr1noB <- arc.strength(CPTavgnet1noB, CPTdata, "bic-g")  # Compute edge strengths
strength.plot(CPTavgnet1noB, CPTastr1noB, shape = "ellipse")

# Use .85 threshold, edge strengths are determined by direction probability
CPTboottabnoB <- CPTdagbootnoB[which(CPTdagbootnoB$strength > 0.85 & CPTdagbootnoB$direction > 0.5), ]  # Edge strengths from net2, edge presence from net1
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CPTboottabnoB
CPTastr4noB <- CPTboottabnoB  ## table with direction probabilities
CPTastr4noB$strength <- CPTastr4noB$direction

pdf(file="CPT DAG no B.pdf")  #Figure 2
strength.plot(CPTavgnet1noB, CPTastr4noB, shape = "ellipse")
dev.off()

#examine CPT DAG edge weights based on probability
CPTastr4noB[order(CPTastr4noB[,4]),]

#-----------------------------ACT DAG -----------------------------#

#set variables to numeric
for (i in c(1:26)){
  ACTdata[,i] <- as.numeric(ACTdata[,i])
}

#specify that ACT variables must precede PCL variables

colnames(ACTBlacklist) <- c("from", "to")
ACTBlacklist
## Fit a first Bayesian network, based on 50 random re-starts and 100 perturbations for each re-start.

```r
set.seed(123)
fitACT1 <- hc(ACTdata, restart = 50, perturb = 100, blacklist = ACTBlacklist)  ## hc gives directed graph

bnlearn::score(fitACT1, data = ACTdata)  ## global network score

ACTAsstr <- arc.strength(fitACT1, ACTdata, "bic-g")  ## connection strength
ACTAsstr[order(ACTAsstr[,3]),]  ## sorted edge strength from strongest to weakest

## Now we stabilize the network across multiple samples through bootstrapping:
## Learn 10000 network structures (takes ~5 min, we keep the number of restarts and perturbations considerably low)

set.seed(123)
ACTdagboot <- boot.strength(ACTdata, R = 10000, algorithm = "hc", algorithm.args = list(restart = 5, perturb = 10, blacklist = ACTBlacklist), debug = TRUE)

head(ACTdagboot)
```

```r
## filter the ones with a strength larger than 0.85 and a direction probability > 0.5
ACTdagboot[ACTdagboot$strength > 0.85 & ACTdagboot$direction > 0.5, ]
```

```r
## build the average network using a 0.85 threshold (Sachs et al., 2005, Science)
ACTavgnet1 <- averaged.netw(ACTdagboot, threshold=.85)

bnlearn::score(ACTavgnet1, data = ACTdata)  # 2496.39

ACTAsstr1 <- arc.strength(ACTavgnet1, ACTdata, "bic-g")  ## compute edge strengths
ACTDAGstrength <- ACTAsstr1[order(ACTAsstr1[,3]),]  # order edge strengths

strength.plot(ACTavgnet1, ACTAsstr1, shape = "ellipse")

# use .85 threshold, edge strengths are determined by direction probability

ACTboottab <- ACTdagboot[ACTdagboot$strength > 0.85 & ACTdagboot$direction > 0.5, ]

ACTboottab

ACTAsstr4 <- ACTboottab  ## table with direction probabilities

ACTAsstr4$strength <- ACTAsstr4$direction  ## use the direction probabilities for edge width

pdf(file="ACT DAG Blacklist.pdf")

strength.plot(ACTavgnet1, ACTAsstr4, shape = "ellipse")  # Figure 2

dev.off()

# Examine ACT DAG edge weights based on probability

ACTAsstr4[order(ACTAsstr4[,4]),]

# CPT DAG with PCL clusters

CPTdata2 <- data[c(18:24)]

names(CPTdata2) <- c("Threat", "SWJ", "TRO", "Reexp", "Avoid", "NegAlt", "Hypervig")  # clusters were calculated as sum scores in original dataset
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CPTcor2 <- cor_auto(CPTdata2)

for (i in c(1:7)){
    CPTdata2[,i] <- as.numeric(CPTdata2[,i])
}

set.seed(123)
fitCPT1b <- hc(CPTdata2, restart = 50, perturb = 100)  ## hc gives directed graph
fitCPT1b

bnlearn::score(fitCPT1b, data = CPTdata2)          ## global network score
astrb <- arc.strength(fitCPT1b, CPTdata2, "bic-g")  ## connection strength
astrb[order(astrb[,3]), ]  ## sorted edge strength from strongest to weakest

# Now we stabilize the network across multiple samples through bootstrapping:
## Learn 10000 network structures
set.seed(123)
CPTdagboot2 <- boot.strength(CPTdata2, R = 10000, algorithm = "hc", algorithm.args = list(restart = 5, perturb = 10), debug = TRUE)
head(CPTdagboot2)

# build the average network using a 0.85 threshold (Sachs et al., 2005, Science)
CPTavgnet1b <- averaged.network(CPTdagboot2, threshold = .85)
CPTavgnet1b
bnlearn::score(CPTavgnet1b, data = CPTdata2)
CPTastr1b <- arc.strength(CPTavgnet1b, CPTdata2, "bic-g")  ## compute edge strengths
strength.plot(CPTavgnet1b, CPTastr1b, shape = "ellipse")

# use .85 threshold, edge strengths are determined by direction probability
CPTboottab2 <- CPTdagboot2[CPTdagboot2$strength > 0.85 & CPTdagboot2$direction > 0.5, ]
CPTboottab2
CPTastr4b <- CPTboottab2  ## table with direction probabilities
CPTastr4b$strength <- CPTastr4b$direction  ## use the direction probabilities for edge width

pdf(file="CPT DAG no Blacklist clusters.pdf")  # figure s8; dashed lines represent non-significant arcs/edges
strength.plot(CPTavgnet1b, CPTastr4b, shape = "ellipse")
dev.off()}