

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

by

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A dissertation submitted to the Graduate Faculty of  
Auburn University  
in partial fulfillment of the  
requirements for the Degree of  
Doctor of Philosophy

Auburn, Alabama  
August 7, 2021

Keywords: posttraumatic stress, cognitive processing therapy, acceptance and commitment therapy, network analysis

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## Abstract

Two prominent conceptual models of PTSD are the cognitive model, used in the development of cognitive processing therapy (CPT; Resick & Schnicke, 1992), and the functional contextualist model, underlying acceptance and commitment therapy (ACT; Hayes, Strosahl, & Wilson, 1999). Despite the evidence supporting both models, they fundamentally differ in the constructs that are theorized to pose risk and maintenance for PTSD. Network analysis was used in the current study to examine the dynamic interactions among cognitive (relating to CPT) and functional contextualistic (relating to ACT) variables and PTSD symptoms. Specifically, for both the cognitive and functional contextualist models, a Gaussian graphical model and a directed acyclic graph were estimated, resulting in a total of four networks. A sample of 722 trauma-exposed adults were included in the final sample. Results from the cognitive networks highlighted the importance of maladaptive beliefs about threat, strong negative feelings, strong negative beliefs, feeling distant, startle, and hypervigilance in maintaining the co-occurrence of PTSD symptoms and maladaptive posttraumatic beliefs. Additionally, the cognitive networks revealed that PTSD symptoms are more likely to lead to posttraumatic beliefs, rather than the reverse direction being true. Results from the functional contextualist networks identified numerous associations amongst variables that may be particularly important, including associations between experiential avoidance and avoidance of trauma-related thoughts and memories, between risky behaviors and lack of contact with values, between cognitive fusion and strong negative beliefs, and among trouble concentrating, lack of present-moment awareness, and inaction. Findings from this study may help identify potentially important causal relationships that can be tested further with longitudinal research.

## Acknowledgments

I would like to take this opportunity to thank those who have supported and mentored me along this academic journey. First and foremost, I extend my sincere gratitude to my mentor, Dr. Joe Bardeen. Your commitment to your students' productivity is second to none. More importantly, I thank you for modeling what it means to be a compassionate clinician, a dedicated teacher, and a skilled researcher. I would also like to thank Dr. Frank Weathers, Dr. Dan Syvantek, and Dr. Richard Macatee for their willingness to serve on my committee and provide helpful feedback on this project. This project was possible in part due to the hard work of Travis Rogers, my labmate and friend, who was instrumental in data collection efforts.

While this dissertation marks a crucial milestone in my career, I would not be at this point without the years of loving support from my family and friends. Thank you to my parents, who maintained unwavering faith that I could achieve any goal, no matter how improbable it seemed. You instilled a confidence in me that was an absolute requisite for getting through graduate school. And finally, I am indebted to my peers at Auburn who have passed along their sage advice and answered my endless questions without complaint. Your helpfulness and constant cheerleading made all the difference during these past five years.

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

Posttraumatic stress disorder (PTSD) emerged as a diagnosis in the *Diagnostic and Statistical Manual—3rd Edition (DSM-III)*; American Psychological Association [APA], 1980) to address the impairing stress response for Veterans and survivors of interpersonal violence (Andreasen, 2004), and recent estimates suggest the lifetime prevalence rate of PTSD is approximately 8% (Kilpatrick et al., 2013). A number of treatments have been developed or adapted to treat individuals with PTSD. While several treatments have been considered effective in reducing PTSD symptoms, each treatment differs in the conceptualization of factors that confer risk and maintenance of the disorder. An improved understanding 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, Strosahl, & Wilson, 1999).

### **Conceptualizations of PTSD**

**The cognitive model.** The cognitive model that was used to develop CPT for PTSD came from the cognitive theory of depression (Beck, 1979). The cognitive theory of depression purports that distorted thought patterns (i.e., negative automatic thoughts) about the self, others, and the world leads individuals to interpret ambiguous or neutral information as negative (e.g., interpreting a lack of response from a friend as “No one likes me”). The interpretation of neutral information as negative is a negative bias. Individuals with this negative bias are less likely to engage in activities that (1) are positively reinforcing and (2) provide information to correct their



negative bias. In turn, negatively biased cognitions lead to the symptoms that characterize major depressive disorder. Using the example from above, an individual who says “No one likes me” may limit him/herself from social engagement, thus limiting the opportunity to receive positive reinforcement from socializing, as well as restricting the opportunity to learn that the belief “No one likes me” is not correct. Instead, by limiting social engagement, the negative belief is reinforced and the cycle of depression (e.g., low mood, negative thoughts, restriction in behaviors) continues. Cognitive therapy for depression works to identify and re-evaluate the veracity of those negative automatic thoughts. Resick and colleagues noted that survivors of rape had a similar negative thinking styles, but unlike depressive thoughts, the negative cognitions were anchored to the past—the traumatic event—rather than the present. The creators of CPT referred to these maladaptive beliefs stemming from the traumatic event as “stuck points” (Resick, Monson, & Chard, 2017).

Extending the cognitive model to PTSD, the theory suggests that if pre-trauma beliefs about self, others, and world are inconsistent with information from the traumatic event, people may act in one of three ways: 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 (overaccommodation; Resick & Schnicke, 1992; Zalta, 2015). For example, a survivor of a rape may have held the pre-trauma belief that “People should be trusted.” After the assault, perpetrated by a male acquaintance, she overaccommodates this belief to be “No man can be trusted.” Conflicts between pre- and post-trauma beliefs (i.e., assimilation or overaccommodation) generate heightened psychological distress. Additionally, rigid overaccommodated beliefs, like the negative thought patterns in the cognitive model of

depression, create a biased lens through which neutral information is interpreted. Using the example from above, the belief that no men can be trusted may motivate the survivor to avoid men, thereby (1) positively reinforcing her belief that men cannot be trusted (the survivor maintains safety, which is attributed to the avoidance of men) and (2) limiting opportunities to obtain new information that may run counter to that belief. The maladaptive post-trauma beliefs thus set off a set of hypervigilant (e.g., scanning environments with men for signs of danger) and avoidance (e.g., avoiding situations in which men are present) behaviors that characterize PTSD (Resick & Schnicke, 1992; Zalta, 2015). In an effort to extend cognitive therapy to account for the unique beliefs that exist in PTSD (i.e., consistent themes of self-blame, distrust, safety; beliefs tethered to past event[s]), Resick and colleagues developed CPT.

CPT is considered one of the gold standard treatment packages for treating PTSD, and the efficacy of CPT has been extensively studied (Asmundson et al., 2019; Monson et al., 2006; Resick, Nishith, Weaver, Astin, & Feuer, 2002). Although originally developed to treat survivors of rape (Resick & Schnicke, 1992), it is a validated treatment for individuals who experience a wide variety of traumatic events, including combat, motor vehicle accidents, loss of loved ones, and witnessing of atrocities (Beck & Coffey, 2005; Monson et al., 2006; Schulz, Resick, Huber, & Griffin, 2006). Just as the goal of cognitive therapy for depression (i.e., Beck, 1979) is to progressively re-evaluate the accuracy of negative beliefs about self, others, and the world based on objective evidence, CPT works to target maladaptive trauma-related beliefs (or stuck points). As such, the majority of sessions in CPT focus on retrieving and challenging maladaptive cognitive schema (Resick et al., 2017; Resick & Schnicke, 2002). For the case example above, sessions might focus on gathering evidence for and against the belief that no man could be trusted. Over time, ideally, the client's belief would change to a more balanced, accurate belief

of “Some men are no good, but this is not true of all men” (Resick & Schnicke, 1993). Common themes of beliefs targeted in CPT include maladaptive beliefs about safety, trust, power/control, and esteem (Resick et al., 2017).

*Evidence of risk factors for PTSD in the cognitive model.* Based on the cognitive model used to inform CPT, assimilated and overaccommodated beliefs are risk factors for PTSD. Indeed, evidence suggests that stuck points are associated with greater PTSD severity. For example, Sobel, Resick, and Rabalais (2009) examined accommodated, overaccommodated, and assimilated clauses in impact statements before and after CPT. The impact statement is an assignment delivered at the beginning and end of treatment in which the client identifies why the traumatic event happened to him or her as well as the ways in which the event has impacted beliefs about self, other, and the world. The percentage of accommodated clauses (i.e., adaptive beliefs) was significantly negatively associated with PTSD symptom severity before treatment, indicating that those who were able to accommodate beliefs about the trauma were experiencing less PTSD symptoms. Interestingly, however, overaccommodated and assimilated clauses were not significantly positively related to PTSD symptom severity, which would be consistent with the cognitive model.

Additionally, negative appraisals of the sequelae of a traumatic event (e.g., “Nothing good can happen to me anymore”) and, what Dunmore, Clark, and Ehlers (2001) called “trauma sensitive beliefs” (e.g., “There is no place which is safe”) are risk factors for PTSD under the cognitive model. Such cognitions demonstrate cross-sectional (Dunmore, Clark, & Ehlers, 1997; 1999; Wenninger & Ehlers, 1998) and longitudinal (Dunmore et al., 2001; Ehring, Ehlers, & Glucksman, 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, although there is some inconsistency in the research (i.e., Sobel et al., 2009).

*Evidence of maintenance factors for PTSD in the cognitive model.* In addition to posing risk for PTSD, maladaptive cognitive beliefs appear to be one mechanism by which PTSD symptoms are maintained (Dunmore et al., 1997; 1999). Furthermore, CPT appears to reduce PTSD symptoms via reduction in maladaptive cognitions, signaling that the decrease in maladaptive post-trauma beliefs triggers reduction in PTSD symptoms. For example, changes in hopelessness (Gallagher & Resick, 2012; Gilman, Schumm, & Chard, 2012), self-blame and negative beliefs about oneself (Holliday, Link-Malcolm, Morris, & Surís, 2014; Shumm, Dickstein, Walter, Owens, & Chard, 2015), and negative beliefs about the world (Holliday et al., 2014) appear to mediate reduction in PTSD symptoms in CPT. Investigations of impact statements have demonstrated positive associations between changes in overaccommodated (maladaptive) statements throughout treatment and PTSD symptoms, supporting the theory that CPT decreases PTSD symptoms by correcting maladaptive beliefs (Dondanville et al., 2016; Sobel et al., 2009). Although evidence supports the efficacy of CPT in treating PTSD, and some evidence points to mechanisms of change in CPT, it remains unclear how the proposed mechanisms (i.e., maladaptive cognitions/beliefs) lead to changes in PTSD at the symptom level. For example, it is possible that changes in maladaptive cognitions/beliefs act to reduce symptoms of negative alterations in cognition and mood (cluster D; APA, 2013), which then, in turn, decrease other symptoms. However, evidence of associations of symptom/symptom clusters and cognitive model variables (e.g., overaccommodated beliefs) is mixed. Lancaster, Rodriguez, and Weston (2011) found evidence that maladaptive beliefs about self, others, and the world were more associated with cluster D symptoms than they were with cluster B (intrusions) or C

(avoidance) symptoms. In a randomized control trial of CPT, Macdonald, Monson, Doron-Lamarca, Resick, and Palfai (2011) found that avoidance symptoms evidenced the greatest decrease over time, compared to a waitlist control, suggesting that changes in maladaptive cognitions (the proposed active mechanism of CPT) first acts to reduce avoidance symptoms. Nishith, Resick, and Griffin (2002) found that CPT reduced avoidance and numbing at a constant rate throughout treatment. Thus, it remains unclear how maladaptive post-trauma beliefs are related to PTSD at the symptom or cluster level.

**Functional contextualism.** Functional contextualism is the philosophical framework upon which ACT is based, and it is likened to radical behaviorism and behavior analysis (Anderson & Simmons, 2008; Hayes, Hayes, & Reese, 1988; Jacobson, 1997). Functional contextualism is grounded in the claim that psychological phenomena can only be meaningfully understood with consideration to the context in which it occurs. As such, psychological phenomena are viewed as ongoing processes. The emphasis on processes (e.g., the functions of a thought in a given situation) fundamentally differs from cognitive therapies which emphasize form (e.g., thought content) and do not give as much credence to the context in which the behavior (overt or covert) occurs (Hayes, Villatte, Levin, & Hildebrandt, 2011). Anderson and Simmons (2008) provide an example from bulimia nervosa. They describe that the act of purging may have the function of reducing negative affect following a binge in one individual, whereas another individual may purge to reduce worry resulting from non-eating life events (e.g., relationship stress). Functional contextualism would focus on the function of purging, rather than the purging behavior itself. Extending to PTSD, avoidance of thoughts or memories associated with the traumatic event may serve different functions for different individuals, in different contexts. For example, one person who is at work may avoid thoughts and memories associated

with a traumatic event because they typically, for her, trigger physiological reactions (e.g., shaking, shortness of breath) and she does not want her colleagues to think she is “crazy.” For another, avoidance of trauma-related thoughts and memories may be a function of a more pervasive unwillingness to experience negative affect, which would be triggered by the trauma-related thoughts and memories. Thus, the same (covert) behavior is maintained by different contingencies across individuals and environments. The functional contextualist perspective is thus grounded in principles from behavior analysis, such that the behavior that is being shaped is defined by its relationship with other variables (e.g., the relationship between avoidance, the environmental context, and the antecedents and consequences of avoidance).

Where functional contextualism parts from traditional behavioral analysis is the emphasis on private events (e.g., thoughts) as being subject to behavioral principles (e.g., reinforcement). Therefore, some argue that functional contextualism is akin to radical behaviorism, in which private events and overt behaviors (i.e., observable actions) are treated identically. Because private events are subject to behavioral principles in functional contextualism, the context in which they occur is emphasized. For example, a belief that the world is completely dangerous is not inherently maladaptive from a functional contextualist perspective. Instead, emphasis is on how the belief is or is not helping the individual achieve the goal at hand, given the environmental and private context the individual is in. This stands in contrast to cognitive theories, which may denote such a belief as irrational and problematic without significant consideration to the context.

Both behavior analysis and functional contextualism emphasize “the criterion for truth or adequacy” (p.102; Hayes et al., 1988), such that analysis of the event is grounded in some goal, rather than “analysis for analysis’ sake” (p.102; Hayes et al., 1988). That goal, for both behavior

analysis and functional contextualism, is defined as prediction and influence of behavior. Functional contextualism, therefore, emphasizes the workability (e.g., function) of psychological variables (Hayes, 2004; Ruiz, 2010). It is feasible to alter how a thought functions in an environment by altering one's relationship or reaction to a thought (Hayes, 2004). In contrast, cognitive theories emphasize a more subjective truth—rational thinking. Taken together, a functional contextualism conceptualization of PTSD emphasizes the role maladaptive processes (e.g., experiential avoidance, cognitive fusion; see below) play in maintaining PTSD symptoms, with specific regard for the idiographic function of symptoms and processes across different individuals and contexts (Blackledge, 2004; Follette, Palm, & Hall, 2004).

ACT is grounded in the philosophy of functional contextualism and the theory of relational frame theory (RFT; Hayes, Barnes-Holmes, & Roche, 2001). RFT asserts that language guides behavior through arbitrary contextual control. This ability to infer function of events through language is learned (Hayes, Luoma, Bond, Masuda, & Lillis, 2006). Specifically, language is the operant that becomes shaped through the relational frames. For example, if an individual with PTSD has the thought “I cannot handle going back [to the scene of the traumatic event]”, s/he creates relational frames wherein passing by the scene initiates (a) thoughts about being in danger and (b) the fear response even though the scene is no longer dangerous; that is, the scene and danger are related through verbal frames (Fletcher & Hayes, 2005; Hayes, 2004).

ACT grew out of functional contextualism and RFT. ACT aims to target the potentially maladaptive interaction between cognition and language that prevents individuals from changing or working toward actions that are consistent with their values (i.e., valued-action), versus being driven by distress and avoidance (Hayes et al., 2006). Cognitive fusion (CF)—strong attachment to one's thoughts such that they are interpreted as literally true (Hayes, Levin, Plumb-Villardaga,

Villatte, & Pistorello, 2013)—captures the overregulation of behavior by cognition (Gillanders et al., 2014). When behaviors are rigidly guided by cognition, an individual is guided by the arbitrary, and potentially incorrect, relations between language and behavior (e.g., believing the scene is dangerous because of the thought “I can’t handle going back”). When CF is high, thoughts are more likely to be seen as literally true, and moreover, the individual may begin to see him/herself as the culmination of private events. In ACT, this is known as viewing self-as-content (e.g., thoughts) rather than viewing self-as-context. Furthermore, when CF is high and private events are negative, individuals may have a tendency to attempt to avoid those private events. This unwillingness to maintain contact with internal experiences is known as experiential avoidance (EA; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). 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 (Hayes et al., 2006). Lack of contact with the present moment (1) deemphasizes values outside of private events and (2) limits opportunities to engage in valued-action. As Hayes et al. (2006) describes it, “it can be more important to defend a verbal view of oneself (e.g., being a victim; never being angry; being broken; etc.) than to engage in more workable forms of behavior that do not fit that verbalization” (p.7). The processes above are known as the core processes of psychological inflexibility (“inhexaflex”; CF, EA, self-as-content, lack of contact with present moment, lack of contact with values, inaction). ACT aims to introduce psychological flexibility through the activation of core processes of psychological flexibility: cognitive defusion, acceptance, self-as-context, contact with the present, values, and committed action (Hayes et al., 1999; McHugh, 2011). The more an individual can utilize these processes, the more psychological flexibility is achieved.



Although ACT is transdiagnostic by nature, specific adaptations for PTSD have been developed (e.g., Walser & Westrup, 2007). Though, to date, there has been just one rigorous study evaluating the efficacy of ACT in treating PTSD (i.e., Lang et al., 2017), the conceptual framework of ACT appears to be particularly well-suited for treating individuals experiencing symptoms of PTSD. In an ACT conceptualization of PTSD, each component of the inhexaflex contributes to the rise and maintenance of PTSD symptoms, and thus, each domain is targeted in ACT to improve psychological flexibility, thus decreasing symptoms. Evidence suggests that various components of the inhexaflex are related to PTSD.

***Evidence of the functional contextualist model and PTSD.*** To begin, EA is proposed as a core underlying feature of PTSD, and it has some content overlap with the PTSD symptom of avoidance of thoughts and feelings related to the traumatic event (APA, 2013). EA is associated with risk, maintenance and severity of PTSD symptoms (Kumpula, Orcutt, Bardeen, & Varkovitzky, 2011; Thompson & Waltz, 2010; Tull, Gratz, Salters, & Roemer, 2004). One theory is that EA paradoxically reduces distress in the short-term but exacerbates long-term psychological distress by limiting opportunities to obtain new information that might serve to provide counterevidence to maladaptive thoughts about the trauma (Bardeen, 2015).

Additionally, intrusive thoughts about the event may paradoxically be the result of increased attempts to avoid thinking about the event. The paradox of EA increasing negative thoughts has been noted as a characteristic of PTSD, compared to trauma-exposed controls (Shiperd & Beck, 1999; 2005), and suppression (a specific strategy that may be used to engage in experiential avoidance) of trauma-related thoughts predicts higher levels of intrusion symptoms (Davies & Clark, 1998; Salkovskis & Campbell, 1994). Thus, while the goal of utilizing EA is to avoid,

suppression may actually increase intrusive trauma-related memories, thoughts, or feelings (i.e., cluster B; Blackledge, 2004).

In the context of PTSD, CF may play a particularly important role in exacerbating negative beliefs such as self-blame and overly negative thoughts about oneself, others, or the world (i.e., cluster D; APA, 2013). If these negative cognitions are fused to the self (e.g., viewing oneself as truly to blame), a cascade of negative emotions and behaviors could emerge. Despite strong theoretical rationale for relations between CF and cluster D symptoms, these associations do not appear to have been empirically tested, although higher CF is associated with higher PTSD symptom severity, broadly (Bardeen & Fergus, 2016; Gerhart et al., 2016; Nitzan-Assayag, Aderka, & Bernstein, 2015).

An individual who is tethered either to the past or the future is engaging in a lack of contact with the present moment. Those who have experienced a traumatic event may be at particular risk for feeling tethered to the past because the memories associated with the traumatic event(s) that happened to them in the past influence current behaviors. Similarly, individuals who are suffering from psychological distress, such as PTSD, may be excessively preoccupied with thoughts of how they will continue to suffer in the future (Walser & Westrup, 2007). Evidence suggests that increased mindfulness, which increases one's contact with the present moment, may be a protective factor for PTSD among those who have a trauma history (Smith et al., 2011), and it could represent an antidote to dissociation—a known risk factor for the development of PTSD (Ozer, Best, Lipsey, & Weiss 2003). Additionally, mindfulness-based treatments show promise as an effective treatment for PTSD (Kearney, McDermott, Malte, Martinez, & Simpson, 2012; Niles et al., 2012; Polusny et al., 2015), and may be particularly useful in targeting symptoms of avoidance, anhedonia, and self-blame (King et al., 2013).

Individuals who stay tethered to the past or future may also have difficulty distinguishing the self from internal stimuli (e.g., thoughts, feelings, bodily sensations) because of a general difficulty staying present and aware of private events (Westrup & Walser, 2007). This maladaptive tendency is known as viewing self-as-content (versus the adaptive counterpart, self-as-context). Survivors of trauma may have difficulty distinguishing their identity from the events they have experienced. This could be especially true for those who have a chronic trauma history dating back to their childhood (Westrup & Walser, 2007). According to Boals & Murrell (2016), self-as-content is related to the construct of event centrality, such that those who view the traumatic event as more central to identity may be more likely to tie identity to their thoughts and feelings, especially as they relate to the trauma(s). Event centrality appears to be an important predictor of PTSD severity (Brown, Antonius, Kramer, Root, & Hirst, 2010; Robinaugh & McNally, 2011). However, associations between event centrality/self-as-content and specific symptoms has yet to be examined.

The final components of the inhexaflex—lack of values clarity and inaction—are interlinked such that identification of values can facilitate committed action consistent with those values (Hayes et al., 2006). Though values and valued-action are less studied within the PTSD literature, it stands to reason that greater clarity in values and engagement in valued action (i.e., behaviors that align with one's values) may decrease PTSD symptoms. Engagement in valued-action might increase sources of positive reinforcement. For example, an individual with PTSD who lost contact with the value of being a good friend may be withdrawn from others. By reconnecting with the value and engaging in actions consistent with being a good friend, he increases the opportunities for positive reinforcement via social interaction. In this way, clarity of values and engagement in valued-action may first act to decrease the negative alterations in

mood symptoms (e.g., anhedonia, feeling cut off from others); that is, engagement in valued-action may act similar to behavioral activation in depression by increasing sources of positive reinforcement. An additional potential effect of increased contact with values and valued-action is a decrease in avoidance symptoms. Survivors of trauma may engage in a pattern of avoidance so generalized that activities that were once enjoyed are now avoided. For example, a survivor of a motor vehicle accident may avoid driving, then cars, then leaving the house altogether, which would decrease her opportunities to engage in valued-action. Thus, an increase in clarity of values and valued-action may serve as motivation to reduce avoidance of external reminders. However, the relationships between clarity of values, valued-action, and (1) cluster D symptoms (i.e., negative alterations in cognition and mood; APA, 2013) and (2) avoidance of external reminders do not appear to have been empirically examined in the literature to date.

Taken together, the conceptual framework of ACT (i.e., [in]hexaflex), appears to provide utility in understanding the etiology of PTSD. The role of EA in PTSD may be particularly relevant to risk and maintenance of PTSD (Orsillo & Batten, 2005; Walser & Hayes, 2006). However, it remains to be seen how the interconnected constructs in the inhexaflex relate to specific PTSD symptoms.

**Summary of cognitive and functional contextualist models.** Both the cognitive model and the functional contextualist model have given rise to two treatments for PTSD that have been deemed efficacious (in the case of CPT) or demonstrated promise (in the case of ACT). However, the models fundamentally differ in the conceptualization of the etiology of PTSD. In the cognitive model, the content of thoughts and beliefs are of central importance for understanding the pathogenesis of PTSD. In the functional contextualist model, the process and functions of internal (e.g., thoughts, beliefs) and external (e.g., overt behaviors) events contribute

to psychological (in)flexibility, which then generates emotional distress. Both models may have validity in accounting for PTSD symptoms, but given that they differ so greatly, it is unlikely that the etiology of PTSD is the same under these two models. PTSD, like many anxiety and mood disorders, is comprised of symptoms that mutually reinforce one another in a causal way. The way in which the symptoms reinforce one another likely differs from individual to individual. For example, one person's symptoms may be particularly sensitive to changes in intrusive symptoms. This is akin to a patient whose distress worsens when intrusive symptoms are heightened. Another patient with PTSD, however, may be more sensitive to alterations in sleep. With insomnia, a cascade of difficulties with concentration, low mood, and isolation from others may become activated. Similarly, the etiology may look different depending on the risk and maintenance factors that are considered. When EA is considered as a risk and maintenance factor, avoidance symptoms may be core to PTSD, because of the likely strong relationship between EA and PTSD avoidance symptoms.

It is important to consider how different conceptual 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 be grounded in the role of maladaptive cognitions. Thus, a more precise understanding of the maintaining factors can help stimulate more precise hypotheses. Second, an understanding of maintaining factors within a given conceptual framework helps to improve treatments. Hypothetically, in a functional contextualist conceptualization, CF may be most related to PTSD symptoms. As such, it would stand to reason that ACT for PTSD should first focus on cognitive defusion. Note that this may differ across disorders. When using ACT to treat someone with major depressive disorder, for example, the relationship between valued-action and depressive

symptoms may be strongest, potentially suggesting that that risk factor should be targeted first. Thus, it is important to consider risk factors within a framework by the disorder of interest. Third, an improved understanding of maintaining factors across different conceptual models may aid in the choice of treatment packages. In the examples from above, if insomnia is particularly bothersome to an individual, the treatment package with risk and maintenance factors most closely related to insomnia should be selected. While it is fruitful to understand the maintaining factors under different conceptual models, the analytic tools used to test the theoretical models in relation to symptomatology have been limited to date.

### **Limits of Traditional Approaches to Testing Theoretical Conceptualizations of Latent Variable Modeling.**

Modeling variables as latent constructs is common practice in the realm of psychopathology; however, there are a number of limitations to this approach. First, latent variable modeling (e.g., item response theory, structural equation modeling, latent profile analysis, latent class analysis) aligns with the reflective model, which has been subject to criticism. The reflective model of psychopathology proposes that observables (i.e., symptoms) are caused by or reflective of a latent construct (i.e., the disorder; Borsboom, 2008). The reflective model suggests that some underlying construct causes the disorder, and thus, the symptoms. That is, there is some underlying thing, which we call “PTSD”, causing the symptoms that constitute intrusions, avoidance, negative alterations in cognitions and mood, and alterations in arousal and reactivity. Under the reflective model, a disorder arises and remits all at once, as if turning a light switch, because all symptoms are thought to be directly caused by (or reflected by) the latent construct. Of course, this is not how psychologists conceptualize disorders clinically; symptoms within a disorder rarely change at a parallel rate. For example,

someone may remit in avoidance symptoms, but not in the symptom of startle; although both are symptoms of PTSD, they do not necessarily entirely coincide. In the literature that utilizes latent variable modeling (and thus the reflective model of psychopathology), PTSD is often treated as one latent construct, calculated as a total score of PTSD symptoms as measured via self-report or clinical interview. While there are no statistical problems with the approach, the theoretical underpinning is problematic.

A second limitation of latent variable modeling concerns the modeling of covariance among indicators. Although some approaches to latent variable modeling (e.g., structural equation modeling [SEM]) model covariance among indicators, this is actually in conflict with the reflective model, which posits that “covariance among the symptoms must not arise from any interactions among them; the symptoms must be locally independent” (McNally, 2016, p. 838). That is, under the reflective model, covariance among symptoms should be fully explained by the latent construct; however, latent variable modeling approaches often violate this theoretical assumption. Thus, the statistical and theoretical foundations appear in conflict (Kruis & Maris, 2016) which limits interpretability. In the case where latent variable modeling approaches do not model covariance among indicators, there are also concerns because indicators may share an association that is not fully accounted for by the latent construct. For example, insomnia and concentration difficulties likely share a relationship that is not fully accounted for by PTSD.

Finally, latent variable modeling is limited in that it cannot easily (i.e., without intensive longitudinal research) identify particularly important or influential indicators. It is understood that psychological variables, such as symptoms, likely reinforce one another over time, and that some symptoms may be more influential than others (McNally, 2016). Without intensive longitudinal data, like ecological momentary assessment or cross-lagged panel model designs, it

is difficult to identify feedback loops that strengthen the maintenance of the disorder. Even when longitudinal designs are used, the relationships are often modeled at the latent construct-level, rather than the symptom- or variable-level. When PTSD is modeled as one latent construct made up of a summed symptom total, or dimensions comprised of subscale scores, information about *how* the symptoms within PTSD may operate differently is missing. Such nuanced information about the relationships between symptoms may partially explain inconsistent findings across the psychopathology literature, such that it may be inappropriate to sum or combine heterogeneous symptoms (McElroy, Fearon, Belsky, Fonagy, & Patalay, 2018). Because of the demanding nature of longitudinal designs, a substantial portion of the PTSD literature is unable to speak to the most influential symptoms or variables in the etiology of the disorder.

**Intervention research.** Research designs that examine changes in risk factors throughout intervention (i.e., mediation) are another valid method of testing theoretical conceptualizations of disorders. As described by Laurenceau, Hayes, and Feldman (2007), by assessing the changes in potential mediators of change (e.g., risk factors/treatment targets) within a treatment package, we are better able to understand the etiology of the disorder. For example, under a cognitive conceptualization of PTSD, changes in cognitions should mediate PTSD symptom change throughout CPT. Indeed, this appears to be partially supported (Gallagher & Resick, 2012; Gilman et al., 2012; Sobel et al., 2009). Additionally, consistent with a functional contextualist conceptualization of PTSD, changes in psychological inflexibility appear associated with changes in PTSD symptoms following an ACT intervention (Meyer et al., 2018). However, intervention research is better at confirming a priori conceptualizations of pathology rather than exploring potential risk factors because intervention research is extremely costly and time



consuming. Prior to using an intervention to test a conceptualization, a more feasible exploration of the role of risk and maintenance factors is needed.

**Summary of traditional approaches to testing theoretical conceptualizations of PTSD.** Taken together, while latent variable modeling is pervasive in the PTSD literature, and the insights garnered from this line of research have generated important contributions to our understanding of the etiology of the disorder, latent variable modeling has limitations. Most notably, it is based on the reflective model of psychopathology, which has been subject to criticism for the lack of consideration given to the role of dynamic interactions among symptoms, which more accurately captures the way in which disorders emerge (i.e., through reinforced relationships among symptoms versus through a singular activation of the disorder). Furthermore, latent variable modeling approaches typically do not provide nuanced understanding of how indicators (e.g., variables, symptoms) interact with one another in feedback loops, and if they do, it is through use of time-intensive methodological designs. Intervention research, which often utilizes latent variable modeling approaches, is a considerable methodological step above cross-sectional latent variable modeling because it can test hypotheses about factors that maintain psychopathology. However, it is a resource-intensive approach that often uses hypotheses generated from latent variable modeling studies. Thus, to date, there has been an unfilled gap between the generation and testing of hypotheses of the etiology of psychopathology. Network analysis can provide an important step in generating testable hypotheses regarding the etiology of disorders.

### **An Alternative Approach: Network Analysis**

Network analysis is the statistical method used to estimate and visually depict interactions between observables/symptoms. The “network approach” refers to theoretical

framework in which latent constructs (e.g., mental disorders) are defined by the interaction among indicators (e.g., symptoms; McNally, 2016). As a brief overview, network analysis estimates associations (called “edges”) between observables (e.g., symptoms; called “nodes” in network analysis; Cramer, Waldorp, van der Maas, & Borsboom, 2010). Correlation matrices serve as the input data. These data can be cross-sectional (see Epskamp, Borsboom, & Fried, 2018 for tutorial) or longitudinal (e.g., cross-lagged panel network models, Rhemtulla, van Bork, & Cramer, in press; vector autoregressive models using time-series data [VAR]; Fried & Cramer, 2017), undirected (i.e., without regard to causality; Markov random fields, Epskamp et al., 2018) or directed (e.g., directed acyclic graphs [DAG]; McNally, 2016). The obtained network allows for additional analysis and intuitive graphical representation, through techniques derived from graph theory (Epskamp et al., 2018). One of the most used indices from network graphs is node (symptom) centrality. Node centrality, as measured via node strength, indicates the sum of weighted edges for that node, and thus it indicates how directly connected one node is to the network as a whole. Higher levels of strength indicate that the node is more central (i.e., integral) to the network (Armour, Fried, Deserno, Tsai, & Pietrzak, 2017; Epskamp et al., 2018). Symptom centrality is a common point of focus in network studies in part because deactivating central symptoms (i.e., reducing the severity at which a symptom is experienced) may be the most probabilistically fruitful approach to disrupting, and therefore reducing, symptomatology of a disorder network (Fried et al., 2018).

Many have noted the relative strengths of network analysis as compared to latent variable modeling. In comparison to the limitations noted above, the network approach more accurately captures the way we conceptualize psychological constructs. In PTSD, the network approach models PTSD using the 20 symptoms from the four symptom clusters (i.e., intrusions, avoidance,

negative alterations in cognition and mood, alterations in arousal and reactivity; from the *Diagnostic and Statistical Manual—Fifth Edition [DSM-5]*; APA, 2013) which reinforce one another. Thus, under the network approach, PTSD is defined as the interaction among the 20 symptoms. An additional strength of the network approach is that, unlike the reflective model, the network approach assumes covariance among nodes, and thus is consistent with the statistical framework of network analysis, in which symptoms are modeled as covarying (Borsboom, 2017; Bringmann & Eronen, 2018; Epskamp, Rhemtulla, & Borsboom, 2017). That is, theory and methodology align in the network approach and network analysis, whereas they are inconsistent in the reflective model and latent variable modeling. Finally, network analysis allows for a more nuanced understanding of which symptom(s) are associated with activation of other symptoms. For example, in a network analysis of 2,783 individuals, physiological and psychological reactivity and feelings of detachment appeared to be the most central symptoms in the PTSD network (Fried et al., 2018). This suggests that if reactivity and detachment were disrupted (i.e., reduced) the network would lose connectivity (i.e., global strength), which is akin to disorder symptom severity weakening.

The network approach, therefore, addresses some of the limitations of latent variable modeling, without being as resource intensive as intervention research. However, network analyses are only as good as the variables selected to be in the network, since the analysis relies on the covariance modeled amongst indicators. Thus, there must be strong theoretical rationale for including indicators in a network. Traditional approaches, such as latent variable modeling, may be an important first step in identifying salient variables relevant to a given construct (e.g., risk factors). Following testing of potentially important variables, network analysis can provide a more theoretically sound and nuanced analysis of the etiology of the construct in question.

To date, within the field of psychopathology, network analysis has mainly been used to examine the dynamic interactions between symptoms within a given psychological disorder (Epskamp et al., 2018; McNally, 2016). It has been applied to a number of disorders such as major depressive disorder, anorexia nervosa, and PTSD (e.g., van Borkulo et al., 2015; DuBois, Rodgers, Franko, Eddy, & Thomas, 2017; Spiller et al., 2017). In addition to looking at the symptoms within one disorder, network analysis has proven useful in understanding comorbidity between disorders by examining bridge symptoms. In a network analysis of two co-morbid disorders, the symptoms that connect—or bridge—between disorder A symptoms and B symptoms may provide a first step in understanding the etiology of comorbidity (Borsboom, Cramer, Schmittmann, Epskamp, & Waldrop, 2011; Cramer et al., 2010). This concept of bridge symptoms appears to be used most often to examine co-morbidity between disorders (Borsboom, 2017; Cramer et al., 2010). However, bridge symptoms may also help elucidate relationships between symptoms of a disorder (e.g., PTSD) and psychological constructs (e.g., CF). Recently, there has been a call to examine networks that include variables beyond symptoms, such as risk and protective factors in addition to symptoms (Jones, Heeren, & McNally, 2017).

The next logical step for employment of network analysis is to examine the interactions between symptoms of disorders and identified risk factors within specified conceptual models (Smith et al., 2018). In the most basic sense, this entails computing a network with all symptoms for the disorder of interest as well as all risk factors within the conceptual model of interest. Heeren and McNally (2016) provide an example of this approach. The authors tested a cognitive model of social anxiety disorder (SAD) by examining a network of SAD symptoms, attention bias for social threat, emotional reactivity, and attentional components (alerting, orienting, executive control; measured via the Attention Network Task). Through a combination of the

Gaussian graphical model (GGM; see below for description) and a relative importance network, the authors discovered that orientation of attention toward social threat and behavioral avoidance of social situations were the most central components of the cognitive model of SAD.

Importantly, orienting of attention is not an explicit symptom of SAD, despite its apparent influential role in the disorder. Without the inclusion of non-symptom variables, network analysis may be missing important variables that have direct influence over symptoms (Jones et al., 2017).

There are several positive implications of examining networks of disorder symptoms combined with risk and maintenance factors. First, it provides a novel way to test theoretical conceptualizations, devoid of the limitations mentioned above for more traditional methods. Network analysis investigates the function of observed variables which is more aligned with modern conceptualization of psychopathology. This is especially true in a functional contextualist and radical behavioral conceptualization, which emphasize function of variables over form (Christodoulou, Michaelides, & Karekla, 2019). In the example from Heeren and McNally (2016), long-standing theory suggests that avoidance of fear-eliciting situations blocks opportunities to disconfirm threat-related beliefs, thereby reinforcing fear of social situations; consequently, the networks supported a potential feedback loop between avoidance and alertness, which in turn influenced fear of social situations. Thus, the networks confirmed theory in a way that could not have been assessed in a cross-sectional latent variable model. Second, identifying variables that bridge between the disorder symptoms and conceptual variables provides initial, exploratory hypotheses as to how a risk factor may lead to disorder symptoms. These bridge variables may be particularly fruitful targets in treatment because they are directly related to disorder symptoms. In other words, decreases in those factors may spread deactivation

throughout the network. Although network analysis with cross-sectional data cannot test that claim, the network can highlight potential variables that should be targeted or relationships that should be disrupted (e.g., through intervention) so that the effects on overall symptom presentation can be observed. Heeren and McNally (2016) found that the ability to quickly orient attention was a central bridge between SAD symptoms and constructs of attention and emotional reactivity, such that those who are quicker to orient their attention experience more fear and withdraw from social situations (rather than the reverse direction of the relationship). Therefore, targeting reflexive orienting of attention may have beneficial downstream effects on SAD symptoms. That hypothesis can be tested in intervention research and may not have come to light using traditional latent variable modeling approaches. Third, the network approach may help clinicians select treatment packages based on a client's most impairing symptoms (Christodoulou et al., 2019). For example, if network analysis demonstrated that insomnia was a more central symptom in treatment package A versus B, and trouble sleeping was very impairing for the client, the clinician could have greater confidence that using treatment package A would improve sleep more directly than treatment package B.

Although the implications for this approach are exciting, it is paramount to understand that network analysis, using cross-sectional data, provides just exploratory hypotheses about causality. While we may learn more about the relationship between symptom A and risk factor B, we cannot confidently say that  $B \rightarrow A$  versus  $A \rightarrow B$  without longitudinal data. However, different types of network analysis can help elucidate potential causal relationships, which can then be used to form more well-defined hypotheses for intervention or longitudinal studies (Moffa et al., 2017). The strengths and limitations of those types of networks are discussed below.

**Gaussian graphical model (GGM).** Of the various types of networks, pairwise Markov random field networks (PMRF) have predominated the psychopathology network literature in part because they are well-suited for cross-sectional data. The networks provide undirected (i.e., no causal inferences) edges (i.e., associations) between nodes (i.e., variables) by using a polychoric correlation matrix. The edges can be interpreted as partial correlations. Within the family of PMRF networks is the weighted GGM, which is suited for non-binary, normally distributed data (Epskamp & Fried, 2018). PMRF networks likely also prevail in the psychopathology literature because they allow examination of feedback loops. For example, node A can connect to node B, which can connect to node C, which connects to node A. This is crucial in psychopathology research because we understand that symptoms reinforce one another in cyclical ways (McNally, Heeren, & Robinaugh, 2017). However, the PMRF approach is limited in that it does not speak to directionality (e.g., does node A lead to node B or vice versa?). That being said, PMRF networks are a first step in generating potential causal relationships (Moffa et al., 2017). Adding to the example from above, if node D is not connected to node A, we can now test (via longitudinal, experimental, or intervention methods) the causal relationships  $A \rightarrow B$  and  $B \rightarrow A$ , and we can drop any plans to test  $A \rightarrow D$  or  $D \rightarrow A$ .

**Directed acyclic graph (DAG).** DAGs are rooted in Bayesian statistics and have been described as non-parametric SEM (Rohrer, 2018). In contrast to a parametric SEM, a DAG uses the data to discern parameters (e.g., distributions; Rohrer, 2018). The DAG accomplishes this through an iterative process of adding edges (i.e., associations) between nodes and running those edges in the reverse direction until an optimal fit is obtained (McNally, Mair, Mugno, & Riemann, 2017). In contrast to the weighted GGM, a DAG provides arrowed edges which represent predicted directionality (McNally, Mair et al., 2017). DAGs also differ from weighted

GGMs in that they do not allow feedback loops. That is, if there is a directed arrow from node  $A \rightarrow B$  there cannot be a directed arrow from  $B \rightarrow A$ . The arrow will appear in the direction that is most probabilistic given the data. For example, if  $A \rightarrow B$  in 51% of sampled data, and  $B \rightarrow A$  in 49% of sampled data, the network will depict an association from A to B (Moffa et al., 2017). Jones, Mair, Riemann, Mugno, and McNally (2018) write that DAG networks do “tell us what causal model is most likely to have generated the data, assuming the data were actually generated from a causal model” (p.4), and therefore is a step beyond what the GGM can provide in terms of generating hypotheses of causality. Thus, the limitations of the GGM are addressed in the DAG, and vice versa (McNally, Heeren, et al., 2017).

### **Current Study Aims and Hypotheses**

The aim of the current study was to examine how PTSD symptoms relate to the risk and maintenance factors identified in two theoretical conceptualizations of PTSD (i.e., cognitive and functional contextualist approaches). To do so, two types of networks well-suited for cross-sectional data were employed: the GGM and DAG, which, when interpreted in parallel, maximize the benefits of each approach (i.e., feedback loops in the GGM, probable directionality in the DAG). Each network included the factors associated with the conceptualization (either cognitive or functional contextualism) and PTSD symptoms. Results from these cross-sectional networks, while unable to speak to causal mechanisms, elucidate “admissible causal relationships,” which can then be used to form more well-defined hypotheses for intervention studies (p. 1274; Moffa et al., 2017). Thus, this study represents an important initial step needed prior to testing how factors from the cognitive and functional contextualist models predict and maintain PTSD symptoms. It should be noted that the aim of the study is not to compare the validity of the cognitive versus functional contextualist model as they relate to PTSD. Rather, it



is to demonstrate that risk and maintenance factors for PTSD (as noted from symptom centrality, edges, feedback loops) appear different depending on the conceptualization that is chosen.

Several predictions were made. First, in the cognitive network (GGM only), it was expected that cluster D symptoms (negative alterations in cognition and mood) would act as the bridge between PTSD symptoms and all cognitive variables (i.e., maladaptive beliefs about threat of harm, self-worth and judgment, and reliability and trustworthiness of others). This prediction is based on the theoretical overlap between cluster D symptoms and cognitive variables, as well as some research suggesting cognitive variables may be most highly related to cluster D symptoms (Lancaster et al., 2011). Because the intention was to specify DAG models such that the risk factors (i.e., cognitive variables) would have downstream effects on the PTSD symptoms, rather than vice versa, no specific hypotheses were made about the cognitive DAG network. That is, given the conceptual overlap between all three cognitive variables, there were no predictions as to differential downstream effects that each of these cognitive variables might have on PTSD symptoms. Several hypotheses were made for the functional contextualist (both GGM and DAG) networks. Given the paradoxical nature of EA on PTSD (e.g., Kumpula et al., 2011), it was hypothesized that EA would be most strongly connected to intrusive symptoms (cluster B). It was also hypothesized that EA and avoidance (cluster C) symptoms would share a strong association, given their strong theoretical overlap. Moreover, based on theories of depression that link behavioral activation to reduced symptoms of low mood (Dimidjian et al., 2006), CF and lack of contact with values were expected to be connected to negative alterations in cognitions and mood (cluster D) symptoms. Finally, lack of contact with the present moment was expected to be connected to avoidance, anhedonia, and blame symptoms (per King et al., 2013). Thus, it was posited that there would be multiple bridges by which functional

contextualist and PTSD variables were connected. For the functional contextualist DAG network, EA was expected to be the most influential variable, given its high overlap with core symptoms of PTSD (i.e., cluster C symptoms).

## **Method**

### **Participants**

Participants were recruited through Amazon's Mechanical Turk (MTurk). MTurk is an Internet-based platform in which individuals can participate in research for monetary compensation. Evidence suggests MTurk data are of high quality (Buhrmester, Kwang, & Gosling, 2011; Paolacci, Chandler, & Ipeirotis, 2010; Shaprio, Chandler, & Mueller, 2013) and participants are more diverse than undergraduate samples (Buhrmester et al., 2011). Some have cautioned that Internet-based participants may be more prone to random responding (Goodman, Cryder, & Cheema, 2013). As such, quality control methods (i.e., using participants with at least 95% approval ratings from requesters and at least 50 past human intelligence tasks; Peer, Vosgerau, & Acquisti, 2014) were used. Participants with high approval ratings (i.e., 95% or greater approval rating) have provided data that is more reliable than data from participants using quality control questions (Peer et al., 2014). Participants were eligible to participate if they were fluent in English, resided in the United States, and were at least 18 years old. Participants were compensated \$1.75 for completing the battery of questionnaires, an amount consistent with similar MTurk studies of similar length (Fergus & Dolan, 2014). Power analyses for network analysis do not yet exist, and thus the proposed sample size was based on precedent. A maximum of 325 parameters were to be estimated (325 in the functional contextualist network, 276 in the cognitive network). Afzali et al. (2017) estimated a stable (i.e., greater than .50 correlation stability [CS]; Epskamp et al., 2018) network with a similar number of parameters using 449

individuals. Generally, the number of participants should exceed the number of parameters (Epskamp et al., 2018). As such, a sample of at least 400 individuals who had experienced a Criterion A event (*DSM-5*; APA, 2013) was needed.

Removal of duplicate entries and MTurk participants who did not complete the measures outlined below (e.g., entered the MTurk worker ID, but did not complete any of the measures) resulted in a sample of 999 participants. Coding of Criterion A traumatic event exposure, following the procedure outlined under *Data Reduction*, removed an additional 277 individuals who either did not directly experience a Criterion A traumatic event ( $n = 226$ ) or who provided narratives consistent with computer-generated responses ( $n = 51$ ; e.g., text copied from a website; following guidance by Yarrish et al., 2019). The final sample of 722 participants was nearly half female ( $n = 386$ ; 53.5%) with an average age of 37.02 ( $SD = 11.17$ ; *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%), 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 17-item checklist assessing potentially traumatic events. Participants indicate exposure level for each of the sixteen events (e.g., witnessed it, learned of it happening to a close family member or friend). Additionally, participants can write-in another stressful event not captured by the list. The extended version of the LEC-5 asks for a narrative description as well as clarification questions (e.g., did event involve exposure to actual or threatened death, serious injury, or sexual violence). Consistent to the procedure used by Bardeen and Benfer

(2019), participants provided narrative descriptions and responded to clarification questions for each event endorsed in order to accurately capture exposure to a Criterion A event. Without querying each event, approximately 25% of the sample may be mischaracterized as not having experienced a Criterion A event (Bardeen & Benfer, 2019). Participants then selected the event they consider the worst.

**Multidimensional Psychological Flexibility Inventory (MPFI; Rolffs, Rogge, & Wilson, 2018).** The MPFI is a 60-item measure of the six dimensions of psychological flexibility (present moment awareness, values, committed action, self-as-context, cognitive defusion, and acceptance) and 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 indicate how much they have experienced aspects of psychological in/flexibility 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. Although the MPFI has just recently been developed, the initial psychometric assessment from Rolffs et al. (2018) is promising. The scale was developed from an original pool of 554 items derived from 22 existing ACT-based measures (e.g., Five Facet Mindfulness Questionnaire) and author-generated questions. Through exploratory factor analysis and Item Response Theory, the final 60 items (five items for each of the 12 dimensions) were selected. The inflexibility composite, which is of primary interest for the current study, demonstrated good convergent validity in a healthy community sample (e.g., Multidimensional Experiential Avoidance Questionnaire-Distraction,  $r = .58, p < .05$ ) and divergent (e.g., Self-Compassion Scale-Kindness to Self,  $r = -.36, p < .05$ ; Rolffs et al., 2018). One additional study has utilized the MPFI and found good internal consistency for the inflexibility composite ( $\alpha = .88$ ), as well as sensitivity to change

throughout ACT treatment (Dubler, 2018). See Table 1 for internal consistency and correlations from the current study.

**Posttraumatic Maladaptive Beliefs Scale (PMBS; Vogt, Shiperd, & Resick, 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 good concurrent (e.g., medium- to large-sized correlations with Posttraumatic Cognitions Inventory; Fergus & Bardeen, 2017) and criterion validity (e.g., medium-sized correlation with PTSD symptoms, Vogt et al., 2012). Internal consistency for the PMBS is consistently adequate ( $\alpha > .70$ ; Fergus & Bardeen, 2017; Shiperd & Salters-Pedneault, 2018; Vogt et al., 2012). See Table 1 for internal consistency and correlations from the current study.

**PTSD Checklist for DSM-5 (PCL-5; Weathers, Litz et al., 2013).** The PCL-5 is a 20-item measure of DSM-5 PTSD criteria B (intrusions), C (avoidance), D (negative alterations in cognitions and mood), and E (alterations in arousal and reactivity; APA, 2013). For this study, participants responded to the PCL-5 in a trauma-general fashion (i.e., without respect to an index trauma). Evidence suggests that symptom severity and factor structure of PTSD appear similar regardless of whether symptoms are reported in a trauma-specific or trauma-general manner (Bardeen & Benfer, 2019; Elhai et al., 2009). Moreover, there is an acknowledgment that PTSD can arise from multiple events, and therefore tying symptoms to a singular event may be problematic (Kilpatrick et al., 2013; Smith, Summers, Dillon, & Cogle, 2016). 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 thus indicate greater PTSD symptoms. The PCL-5 has demonstrated adequate internal consistency ( $\alpha > .90$ ), convergent (e.g., large-sized correlations with the Posttraumatic Distress Scale,  $r = .85, p < .01$ ), and discriminant validity (e.g., medium-sized correlation with Personality Assessment Inventory-Antisocial Features,  $r = .39, p < .01$ ; Blevins, Weathers, Davis, Witte, & Domino, 2015). See Table 1 for internal consistency and correlations from the current study.

### **Data Analytic Plan**

**Data reduction.** Data reduction was completed using SPSS (IBM SPSS, Version 23.0). Criterion A status was clarified through a review of follow-up questions and narratives from the extended LEC-5. The self-selected “worst event” was assessed for Criterion A status first. Events marked as *happened to me directly* or *witnessed it* and endorsed that *my life was in danger*, *someone else’s life was in danger*, or the event *involved sexual violence* met initial Criterion A, and those events were subject to further review of the narrative to identify discrepancies or patterns of inattention in responding (e.g., event is a serious transportation accident, but participant noted that it involved sexual violence). If the self-selected worst event did not meet Criterion A status, other endorsed events were assessed in the same way (consistent with Bardeen & Benfer, 2019). Participants who endorsed at least one Criterion A event were included in the final sample. All remaining data analysis were conducted in R using RStudio (Version 1.1.419, 2009-2016).

**Network estimation.** A total of four networks were estimated. First, two GGM networks were estimated: one with MPFI and PCL-5 variables (henceforth referred to as the functional contextualist network) and one with PMBS and PCL-5 variables (cognitive network). Additionally, a directed acyclic graph (DAG) was estimated for both the functional contextualist

and cognitive networks. All four networks were estimated using the same sample. Use of the same sample for the functional contextualist and cognitive networks aids in comparison of PTSD symptom patterns between conceptualizations. For both networks, the variables from the MPFI and PMBS were collapsed into subscale nodes. For example, the functional contextualist network contained the 20 PTSD symptom nodes and six nodes for each dimension of psychological inflexibility. This approach (1) improves power by reducing the number of parameters to be estimated and (2) more accurately depicts the nature of the question the current study aims to answer. For example, the focus in the present study was on how each of the PTSD symptoms interacts with other PTSD symptoms and dissociable dimensions of psychological inflexibility, not on how each of the 60 MPFI items interact with one another and with the 20 PTSD symptoms.

***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, Tran, & Lueger-Schuster, 2016). The estimated networks utilized the Fruchterman-Reingold algorithm to aid in the visual interpretation of the network. With this tool, the thickness of edges is representative of the strength of the association. Green edges indicate a positive association, whereas red edges indicate a negative association (Fruchterman & Reingold, 1991; McNally, 2016). The *qgraph* package was used to estimate the GGM networks.

***Network inference.*** In addition to providing a visual network, quantitative centrality was calculated for the cognitive and functional contextualist GGM networks. A traditional metric of centrality is node strength--or the absolute-value sum of edge weights (visually depicted as edge thickness) from that node (Armour et al., 2017; Constantini et al., 2015). Nodes with high strength are more central, or important, to the overall network. However, some have raised

concerns about the use of node strength when some negative edges exist in a network (Robinaugh, Millner, & McNally, 2016). In response, it has been suggested that expected influence, or the sum of edge weights while maintaining the positive/negative sign of each edge, is a better indicator of centrality for networks with negative edges (Robinaugh et al., 2016). Thus, expected influence was used in the present study as the metric of centrality.

*Bridge influence.* To examine the variables that are most influential in bridging between PTSD symptoms and the respective conceptual frameworks, bridge influence was calculated for each GGM network. First, two clusters of variables, termed communities in network analysis, were identified: the PTSD symptoms and the conceptual variables (e.g., inhexaflex variables in the functional contextualist network). 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 is calculated. For example, EA bridge influence is calculated as the sum of edges from EA to PTSD symptoms. This is referred to as 1-step bridge influence. Whereas 1-step bridge influence only takes direct relationships into account, 2-step bridge influence uses the same procedures to calculate indirect influence. For example, if EA was only linked to CF, which was associated with seven PTSD symptoms, 2-step bridge influence for EA would be calculated as the sum of all seven of those edge weights (Jones, Ma, & McNally, 2019; Ross, Murphy & Armour, 2018).

*Network accuracy and stability.* Network stability is an important step in determining the reliability of estimated networks (Fried & Cramer, 2017). Following the methods put forth by Epskamp et al. (2018), stability for both GGM networks was calculated. Specifically, 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 strength. CS-coefficients indicate the proportion of cases that can be removed while still retaining a relatively high correlation (i.e., 0.7) with the original strength metrics. CS-coefficients above .25 are acceptable (Epskamp et al., 2018).

**Directed acyclic graphs.** DAG networks were computed using the *bnlearn* package and *hill-climbing* algorithm, which continuously adds, removes, and reverses direction of edges until optimal fit (i.e., Bayesian information criterion [BIC]) is achieved (McNally, Heeren, et al., 2017). This process has several options for maximizing stability and minimizing the iterations of the final network. First, one can establish the proportion of networks in which an edge must be present (amongst the bootstrapped networks) in order to be retained in the final DAG network. Second, one can rule out directional relationships based on theory (McNally, Mair et al., 2017). Based on precedent (e.g., Bernstein, Heeren, & McNally, 2017; McNally, Mair et al., 2017), 1,000 bootstrapped samples were averaged for the final network. To minimize the number of potentially spurious edges, the model was specified such that an edge must appear in 85% of the bootstrapped networks in order to be retained in the final network (consistent with McNally, Heeren, et al., 2017). Additionally, 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. The resultant DAG network depicted directional probabilities as edge thickness (McNally, Mair et al., 2017).

## Results

Descriptive statistics for the final sample ( $N = 722$ ) are presented in Table 2. Data were not skewed (skewness range: -0.10 - 1.24). Thus, nonparanormal transformation was not applied.

## **GGM Networks**

**Network estimation.** The visual depiction of the cognitive and functional contextualist GGM networks are illustrated in Figure 1. For both networks, PTSD symptoms within each cluster generally clustered with one another (e.g., cluster B symptoms clustered together). However, within the cognitive network, this was not the case for cluster E symptoms, which appear to have clustered in pairs (i.e., irritability [PCL15] and risky behaviors [PCL16]; hypervigilance [PCL17] and startle [PCL18]; trouble concentrating [PCL19] and insomnia [PCL20]). Somewhat consistently, in the functional contextualist network, hypervigilance (PCL17) and startle (PCL18) did not cluster well with other cluster E symptoms. Additionally, trauma-related amnesia (PCL 8) did not cluster with other cluster D symptoms. Overall, in the cognitive network there were 81 non-zero edges, the vast majority of which (76 edges) were positive. In the functional contextualist network, there were 82 non-zero edges, the vast majority of which (79 edges) were positive. Thus, overall network estimation was similar across networks.

**Network inference and bridge symptoms.** Node expected influence for cognitive and functional contextualist GGM networks is depicted in Figure 2. In the cognitive network, the strongest nodes were feeling distant (PCL13), flashbacks (PCL3), strong negative feelings (PCL11), and trouble experiencing positive emotions (PCL14). In the functional contextualist network, the strongest nodes were lack of contact with values (MPFI-NV1), inaction (MPFI-Inc), flashbacks (PCL3), trouble concentrating (PCL19), unwanted memories (PCL1), and risky behaviors (PCL16).

One- and two-step bridge expected influence for both GGM networks is depicted in Figure 3. In the cognitive network, PMBS-Threat had the highest one and two-step bridge

expected influence of the cognitive variables. Of the PCL variables, strong negative beliefs (PCL9) and feeling distant (PCL13) had the highest one and two-step bridge expected influence. Thus, the hypothesis that cluster D symptoms would bridge between cognitive and PTSD variables was supported; the two PCL variables with the highest bridge expected influence (both one- and two-step) were from cluster D.

In the functional contextualist network, EA (MPFI-Avd) and lack of present moment awareness (MPFI-NtP) had the highest one and two-step bridge expected influence (nearly equal values) of the functional contextualist variables. Of the PCL variables, trouble concentrating (PCL19) had the highest one and two-step bridge expected influence. Contrary to study hypotheses, EA (MPFI-Avd) 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 one cluster D symptom (strong negative beliefs); however, lack of contact with values (MPFI-NVI) was 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; Figure 4a). In the functional contextualist GGM network, 40 of the 82 non-zero edges were truly non-zero (Figure 4b). Centrality from subsets of bootstrapped networks (using a case-dropping procedure) indicated that node strength remained fairly stable across subsets for

both the cognitive and functional contextualist networks (Figures 4c, 4d). The strength of the CS-coefficient for both networks exceeded the 0.25 threshold put forth by Epskamp et al. (2018; cognitive network = .28, functional contextualist network = .36). Thus, the networks were considered stable and interpretable.

### **DAG Networks**

In the original data analytic plan, it was proposed that the DAG models would be 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, and thus the model could not be computed in the proposed manner. In other words, using the precedent that an edge had to appear in 85% of bootstrapped results, none of the cognitive variables preceded PCL variables. The same was true when the threshold was relaxed to 50% of bootstrapped results. Therefore, the proposed specification that PCL variables could not precede cognitive variables was removed. The proposed model did compute for the functional contextualist DAG network. 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 5.

From examination of the cognitive DAG network, it becomes clear that unwanted memories (PCL1) and blame (PCL10) hold an important role in the rise of PTSD symptoms and maladaptive posttraumatic beliefs. These are the only two variables that are not acted upon by another variable in the network. One might think of them as the symptoms that have the greatest

influence over the entire network. As mentioned above, the unexpected finding from the cognitive DAG network is that the cognitive variables appear to be consequences, rather than antecedents, of PTSD symptoms. PTSD symptoms had downstream effects onto 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).

As stated above, the functional contextualist DAG ran with the specification that PTSD variables should not precede functional contextualist variables. The resultant DAG depicts the two constructs as somewhat separated, with just three edges bridging the constructs. Specifically, EA (MPFI-Avoid) has direct downstream effects on avoidance of trauma-related thoughts and memories (PCL6), lack of present-moment awareness (MPFI-Not Pres) has downstream effects on trouble concentrating (PCL19), and lack of contact with values (MPFI-No Values) has downstream effects on risky behaviors (PCL16). With the exception of these three bridges, directional relationships stayed within their respective constructs. 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. It was hypothesized that EA would be the most influential variable in the network. While it was not the most influential variable, 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.

## **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. Tandem interpretation of results from the GGM and DAG networks is meant to generate informed hypotheses about potential directional relationships between the risk factors and PTSD symptoms. Some study hypotheses were supported. Specifically, in the cognitive GGM network, cluster D PTSD symptoms did appear to act as a bridge between PTSD symptoms and cognitive variables. Also as predicted, in the functional contextualist GGM network, EA shared a strong association with cluster C symptoms. However, several hypotheses were not supported; the most surprising finding was that, contrary to expectations, the cognitive variables do not precede PTSD symptoms in the DAG model. That is, results suggest that the hypothesis that PTSD symptoms temporally precede cognitive variables is more likely to be supported than the reverse direction.

### **Interpretation of Results**

Despite the surprising results of the cognitive DAG network, interpreting the results of both the GGM and DAG in parallel may still help generate hypotheses about directional relationships amongst variables. In the cognitive GGM network, cluster D symptoms—feeling distant, strong negative feelings, and trouble experiencing positive emotions, specifically—were the most central symptoms, along with flashbacks. These PTSD symptoms may be particularly important treatment targets in the context of the cognitive model of PTSD, such that decreasing these symptoms might prove most fruitful in reducing PTSD symptoms and co-occurring posttraumatic maladaptive beliefs. The cognitive GGM network also 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 are all associated with one another. Given that some of these variables were among the most central in the network, this may be a particularly salient feedback loop. 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. One interpretation of this loop is that mood-related symptoms (i.e., strong negative beliefs, strong negative feelings) might 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). However, behavioral avoidance was not directly associated with any of these variables in the GGM or DAG, and it did not have high expected influence and bridge expected influence (both 1- and 2-step).

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. Then, given the associations observed in the GGM, either maladaptive beliefs about threat or startle reinforces the other variables in the loop (i.e., [1] hypervigilance and [2] either maladaptive beliefs about threat or startle). The vigilance-

avoidance hypothesis of cognitive biases that are thought to that 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, Treat, & Hollingworth, 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. While the avoidance of threat stimuli may be effective in reducing this physiological arousal, gradually it reinforces beliefs that threat is omnipresent (i.e., maladaptive beliefs about threat) by failing to provide the opportunity for new learning. Over time, if maladaptive beliefs about threat are continuously activated, these beliefs may reinforce the alterations in arousal and reactivity (e.g., startle, hypervigilance), and thus, maintain this potential feedback loop. For example, a person who holds the maladaptive belief that the world is completely dangerous is likely going to be hypervigilant of his or her surroundings and more easily startled. In support of this possible feedback loop, in a post-hoc DAG (described below; Figure 6) in which PTSD symptoms were collapsed into clusters, maladaptive beliefs about threat led to cluster E symptoms (i.e., alterations in arousal and reactivity, such as hypervigilance). Thus, taken together, there may be a reciprocal relationship between hypervigilance, startle, and maladaptive beliefs about threat.

There were not any potential feedback loops observed that included maladaptive beliefs about self-worth and judgment or reliability and trustworthiness of others. This is in line with the observation that these two constructs had much lower bridge expected influence than maladaptive beliefs about threat, and it is consistent with the observation that these two



constructs appeared 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. In CPT, beliefs about safety (directly related to beliefs about threat) are first addressed, 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 four 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. With the model specification of PCL variables not preceding functional contextualist variables, a strong (i.e., high probability) edge flowed from EA to avoidance of trauma-related thoughts and memories. This is consistent with empirical evidence that EA, measured prior to a traumatic event, predicts PTSD symptoms following that event (Kumpula et al., 2011). It appears, from the DAG, that avoidance of thoughts and feelings associated with the trauma spreads activation to the other avoidance symptom (per GGM and DAG; avoidance of reminders). 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 are characterized by the relationships between the remainder of the inhexaflex constructs, which are highly interconnected, and cluster D and E symptoms. The second bridge involves a potential feedback loop among trouble concentrating, lack of present-moment awareness, and inaction. There are associations amongst these three variables in the GGM. The DAG 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 exhibit trouble concentrating. The relationship between inaction and these variables (trouble concentrating, lack of present-moment awareness) is less clear. Though they are connected in the GGM, inaction is not directly related to these variables in the DAG. If lack of present-moment awareness led to inaction, the DAG would not present this directional relationship because it would introduce a feedback loop. That is, the directional flow from inaction to CF to self-as-content, depicted in Figure 5, might be stronger than a relationship from lack of present-moment awareness to inaction, and therefore it is not depicted in the DAG. However, given the association between these variables in the GGM, this relationship might exist. It is plausible that, consistent with the

DAG, lack of present-moment awareness activates 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. Overall, trouble concentrating appears to be an important bridge between constructs.

In a third bridge, CF was associated with strong negative beliefs, per the GGM. However, this direct relationship was not present in the DAG. There was also no indirect relationship (i.e., mediator present) between these variables in the DAG. This could signify that the relationship exists in a direction that was not allowable in the DAG because it would introduce feedback loops (i.e., from CF to strong negative beliefs). A relationship from CF to strong negative beliefs might make theoretical sense, such that someone who is more fused to his or her thoughts is likely to experience negative thoughts about themselves as literal truth rather than transient cognitive events of relatively little value. This in turn, is likely to increase the likelihood that these negative thoughts increase in intensity and frequency and develop into firmly held beliefs, which subsequently increases the likelihood of experiencing prolonged negative affect and psychopathology (e.g., anxiety and depression; Gillanders et al., 2014). Some evidence suggests that CF prospectively predicts PTSD symptoms, adding support to the possibility that there is a directional relationship from CF to strong negative beliefs (Nitzan-Assayag et al., 2015).

The final bridge by which PTSD symptoms and psychological inflexibility appear to co-occur is through an association between 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. The link from lack of contact with values to engagement in risky behaviors is a logical chain, such that 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). In other words, if someone is very clear in their values, they may be able to experience enough positive reinforcement from engaging in valued-action (for which clarification in values is a prerequisite) that it outweighs the short-lived reinforcement that may be received from engaging in risky behaviors. In support of this hypothesis, clarification 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, Reed, & Gaudiano, 2014).

It is worth noting that, across both DAGs, blame emerged at the top of both networks. In fact, the top variables were similar across both the cognitive and functional contextualist networks (i.e., blame, strong negative feelings, unwanted memories). Variables that are at the top of the DAG contribute the most to downstream effects on other variables in the network. A study of traumatized children and adolescents found that cluster D symptoms and unwanted memories similarly emerged at the top of a DAG estimated from the 20 PTSD symptoms (Bartels et al., 2019). While previous longitudinal research has examined the relative importance of different PTSD symptoms at the cluster level, results have been inconsistent. In some studies, the hyperarousal symptom cluster (under *DSM-IV-TR*; APA, 2000), compared to other symptom clusters, has been shown to be the strongest predictor of subsequent PTSD symptoms (e.g., Marshall, Schell, Glynn, & Shetty, 2006), while acute avoidance symptoms have been shown to be the strongest predictor of later PTSD symptom severity in other studies (Difede & Barocas, 1999). One limitation of these studies may be that they used cluster scores instead of individual

symptoms to examine the trajectory of PTSD symptoms. Given preliminary evidence from this study as well as Bartels et al. (2019), the potentially causal role of blame, strong negative beliefs, and unwanted memories in the development of PTSD should be examined in future longitudinal studies to clarify whether these symptoms have a particularly salient influence over other PTSD symptoms over time.

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 because there were many more PCL variables (20) compared to cognitive variables (three) in the model, the PTSD symptoms were more dominant in the overall network. That is, the PCL variables each contribute more to the overall shared variance (making up 87% of the cognitive network), and therefore, they were the most important in the DAG. To test this post-hoc explanation, PCL variables were collapsed into their clusters (i.e., intrusions [cluster B], avoidance [cluster C], negative alterations in cognitions and mood [cluster D], and alterations in arousal and reactivity [cluster E]), and the DAG for the cognitive network was re-estimated. This made the number of nodes more evenly distributed across PCL variables and cognitive variables, thus reducing the problem described above. However, as seen in Figure 6, even when PCL variables were collapsed into symptom clusters, they preceded the cognitive variables. There was just one exception; maladaptive beliefs about threat led to alterations in arousal and reactivity. Generally, however, it does not appear that the PTSD symptoms preceded the cognitive variables simply because there were more PCL variables. Rather, it may be that the pattern of effects observed is an accurate representation of the development of co-occurring PTSD symptoms and distorted beliefs.

PTSD symptoms are often present in the acute aftermath of a traumatic event and decrease over time for most individuals (i.e., acute stress disorder; Bryant, 2011; Cardeña & Carlson, 2011; Schell, Marshall, & Jaycox, 2004). A substantive goal of PTSD research has been to identify why, how, and for whom natural recovery from acute symptoms is stalled. In CPT, PTSD is described as a phenomenon of getting stuck in recovery. Thus, the cognitive model suggests that PTSD symptoms are present first, but the symptoms fail to remit due to avoidance and the subsequent development of maladaptive beliefs (Resick et al., 2017). Results from this study support the underpinnings of the cognitive model, as the DAG suggests that PTSD symptoms are more likely to precede the maladaptive posttraumatic beliefs. 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 as a pre-trauma risk factor for the development of PTSD symptoms (Kumpula et al., 2011).

### **Limitations and Future Directions**

Study findings should be interpreted in light of limitations. First, while the stability of the strength index was above the acceptable threshold ( $CS$ -coefficients  $> .25$ ) in both GGM networks, a  $CS$ -coefficient of  $.5$  is preferable (Epskamp et al., 2017). Similarly, in both networks, only approximately 50% of the edges had 95% CIs that did not overlap with zero, meaning they were significantly different from zero. Taken together, the stability of the estimated networks is less than ideal, and thus results should be interpreted with some caution. One reason the  $CS$ -coefficient may not have been high is due to the similarity in centrality across nodes. If strength (or expected influence, in this case) is similar across nodes, bootstrapping might lead to a different order of centrality due to sampling error rather than true instability (Fried, 2018).

Though DAG models are growing in popularity in psychological research (Bartels et al., 2019; Kuipers, Moffa, Kuipers, Freeman, & Bebbington, 2019; McNally, Heeren, et al., 2017; McNally, Mair et al., 2017), these findings should not be interpreted as inferring causality. While DAGs provide an important step toward generation of causal hypotheses, beyond what other methods are able to do, there are still limitations. As discussed by Moffa (2017), inferences of causality are only indisputable when all confounds have been measured. The networks estimated in this study do not comprise a full set of likely relevant etiological factors. Thus, there may be important mediators missing from the estimated networks. For example, a full account of the interaction between posttraumatic maladaptive beliefs and PTSD symptoms might include depressive symptoms, which were not measured in the current study, or other relevant psychological constructs, such as emotion dysregulation. Moreover, the network analysis literature acknowledges that there is likely heterogeneity in network structures within one population (Fried & Cramer, 2017). For example, the etiology of PTSD across different conceptualizations (i.e., network structure) may depend on both trauma type and time since trauma (Benfer et al., 2018; Kelley, Weathers, McDevitt-Murphy, Eakin, & Flood, 2009; Schell et al., 2004). Related to this point, in addition to requiring inclusion of all relevant variables in order to infer causality, DAG models are based on the assumption that the data that were collected were actually generated from a causal model (Jones et al., 2018). The data from the present data was 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). As such, caution is warranted in drawing causal inferences from the results of this study. In sum, it will be important to replicate study findings using longitudinal

methods, including measures taken shortly after the traumatic event, and also by limiting the sample to those who have experienced a specific type of trauma (e.g., motor vehicle accident, sexual assault).

Finally, while research supports the use of MTurk for data collection, especially compared to undergraduate samples (Chandler & Shapiro, 2016), MTurk samples may not accurately represent the general population. Specifically, the current sample had an underrepresentation of non-White individuals. While this was not a treatment seeking sample of trauma survivors, it is notable that 35.3% of the sample reported experiencing clinically significant PTSD symptoms (i.e.,  $\geq 33$ ; Bovin et al., 2016). While use of a community sample bolsters generalizability of results, it is important to insure that these findings replicate in clinical samples. Overall, future studies continuing this line of investigation would benefit from using a more diverse sample, with individuals presenting with a greater degree of PTSD symptoms severity.

## **Conclusion**

In spite of the above 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 reinforce PTSD symptoms. For 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 engagement in risky behaviors and lack of contact with values, among lack of contact with the present, trouble concentrating, and inaction, and between CF and strong negative beliefs. In sum, results from the present study suggest the possibility that the two conceptual models of PTSD examined in this study have different paths by which PTSD symptoms may arise and be maintained. These findings should be considered as initial evidence for formulating causal hypotheses about the etiology of PTSD under the cognitive and functional contextualist models.

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## Footnote

<sup>1</sup>A 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, 2018).

Table 1.

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

Variable	Internal consistency ( $\alpha$ )	M (SD)	1	2	3	4	5	6	7	8	9	10
1. MPFI-Avoid/Avd	.93	3.52 (1.22)	--	--	--	--	--	--	--	--	--	--
2. MPFI-Not Pres/NtP	.95	2.62 (1.33)	.44	--	--	--	--	--	--	--	--	--
3. MPFI-SelfCont/SIC	.94	2.70 (1.33)	.47	.72	--	--	--	--	--	--	--	--
4. MPFI-Fsn/Fusion	.95	2.89 (1.39)	.47	.70	.78	--	--	--	--	--	--	--
5. MPFI-NVI/No Values	.94	2.60 (1.34)	.41	.74	.78	.82	--	--	--	--	--	--
6. MPFI-Inact/Inaction	.96	2.68 (1.43)	.40	.71	.76	.84	.87	--	--	--	--	--
7. PMBS-Threat/Thr	.85	17.98 (7.41)	.35	.55	.58	.61	.61	.60	--	--	--	--
8. PMBS-SWJ	.76	14.03 (6.07)	.17	.43	.49	.54	.53	.57	.57	--	--	--
9. PMBS-RTO	.82	15.07 (6.12)	.004*	.17	.16	.20	.18	.20	.36	.42	--	--

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10. PCL-5	.97	25.87 (20.72)	.37	.59	.60	.63	.64	.60	.69	.50	.18	--
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*Note.* \*denotes correlation was not significant at  $p < .01$ ; all other correlations were significant at  $p < .01$ . Avoid/Avd A = Experiential Avoidance subscale; MPFI-Avoid/Avd = Lack of Present Moment Awareness subscale; SelfCont/SIC = Self-as-Context subscale; Fsn/Fusion = Cognitive Fusion subscale; NVI/No Values = Lack of Contact with Values subscale; Inact/Inaction = Inaction subscale; Thr/Threat = Threat of Harm subscale; SWJ = Self-Worth and Judgment subscale; RTO = Reliability and Trustworthiness of Others subscale; PCL-5 = PTSD Checklist for DSM-5 total score.

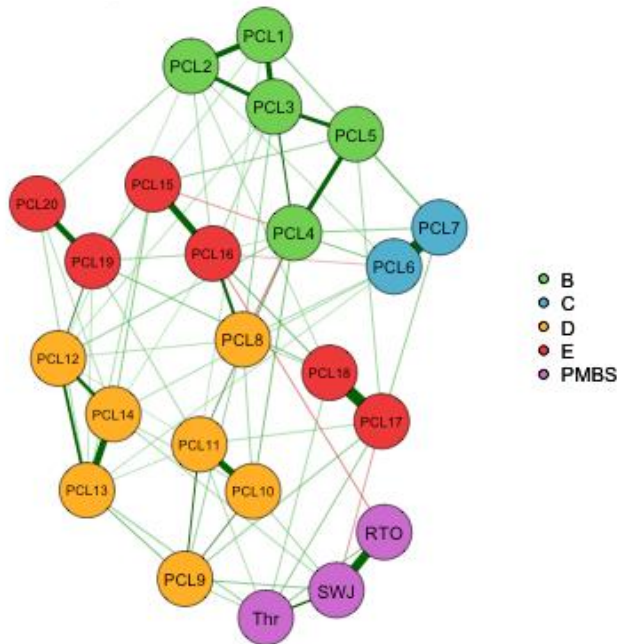
Table 2.

*List of abbreviated labels in networks, full variable name, and corresponding construct.*

Abbreviation in Network	Full Variable Name	Associated Construct
1	Unwanted Memories	<i>DSM-5 PTSD Cluster B</i>
2	Dreams	<i>DSM-5 PTSD Cluster B</i>
3	Flashbacks	<i>DSM-5 PTSD Cluster B</i>
4	Upset Reminders	<i>DSM-5 PTSD Cluster B</i>
5	Physical reminders	<i>DSM-5 PTSD Cluster B</i>
6	Avoid Thoughts	<i>DSM-5 PTSD Cluster C</i>
7	Avoid Reminders	<i>DSM-5 PTSD Cluster C</i>
8	Amnesia	<i>DSM-5 PTSD Cluster D</i>
9	Strong Negative Beliefs	<i>DSM-5 PTSD Cluster D</i>
10	Blame	<i>DSM-5 PTSD Cluster D</i>
11	Strong Negative Feelings	<i>DSM-5 PTSD Cluster D</i>
12	Anhedonia	<i>DSM-5 PTSD Cluster D</i>
13	Feel Distant	<i>DSM-5 PTSD Cluster D</i>
14	No Positive Emotions	<i>DSM-5 PTSD Cluster D</i>
15	Irritability	<i>DSM-5 PTSD Cluster E</i>
16	Risky Behavior	<i>DSM-5 PTSD Cluster E</i>
17	Hypervigilance	<i>DSM-5 PTSD Cluster E</i>
18	Startle	<i>DSM-5 PTSD Cluster E</i>
19	Trouble Concentrating	<i>DSM-5 PTSD Cluster E</i>

20	Sleep	<i>DSM-5</i> PTSD Cluster E
PMBS-Thr/Threat	Threat of Harm	Cognitive Model
PMBS-RTO	Reliability and Trustworthiness of Others	Cognitive Model
PMBS-SWJ	Self-worth and Judgment	Cognitive Model
MPFI-Inc/Inaction	Inaction	Functional Contextualist Model
MPFI-NVI/No Values	Lack of Contact with Values	Functional Contextualist Model
MPFI-Fsn/Fusion	Cognitive Fusion	Functional Contextualist Model
MPFI-SIC/SelfCont	Self-as-Content	Functional Contextualist Model
MPFI-NtP/Not Pres	Lack of Contact with Present Moment	Functional Contextualist Model
MPFI-Avd/Avoid	Experiential Avoidance	Functional Contextualist Model

Cognitive Graph



Functional Contextualist Graph

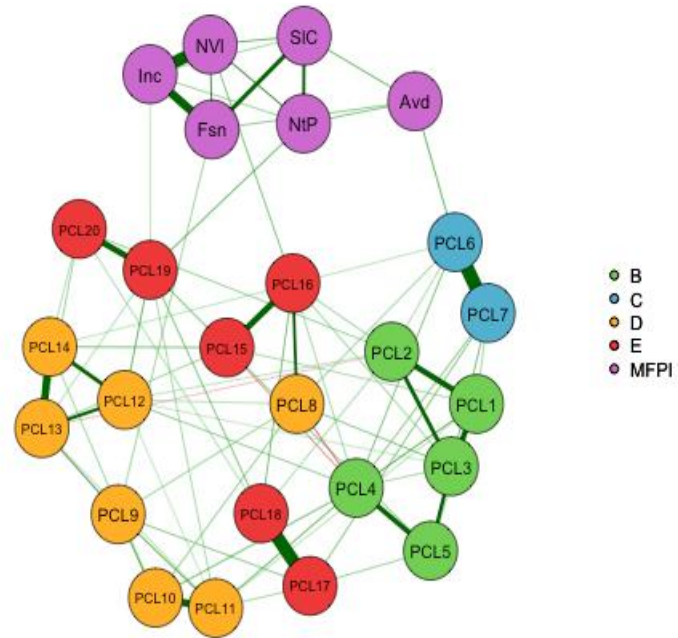
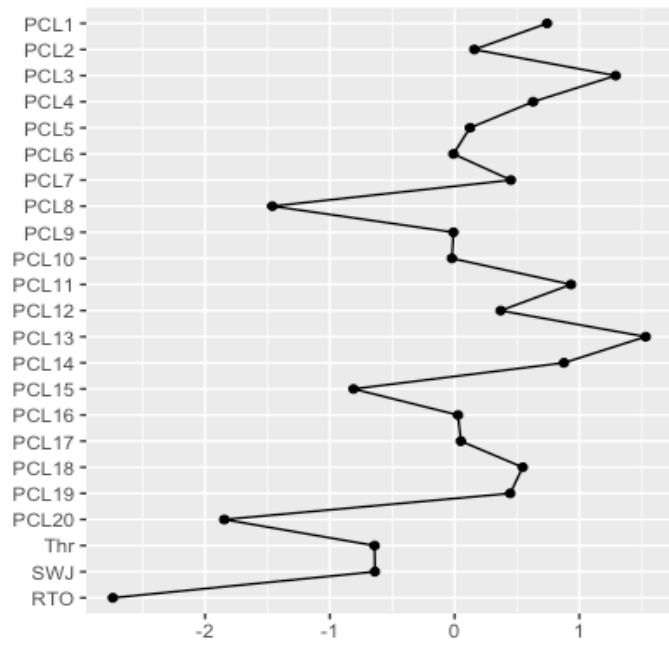


Figure 1. Cognitive GGM network (left) and functional contextualist GGM network (right). See Table 2 for label names.



Cognitive Graph



Functional Contextualist Graph

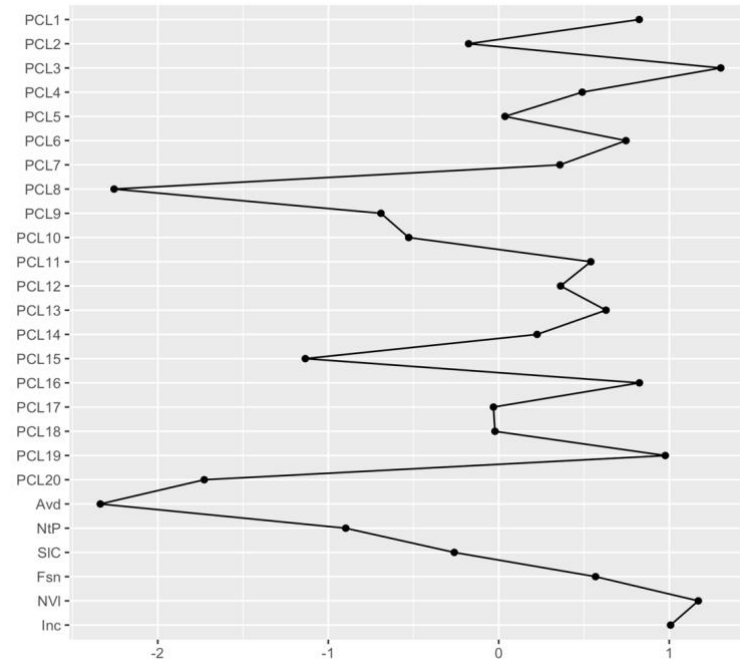
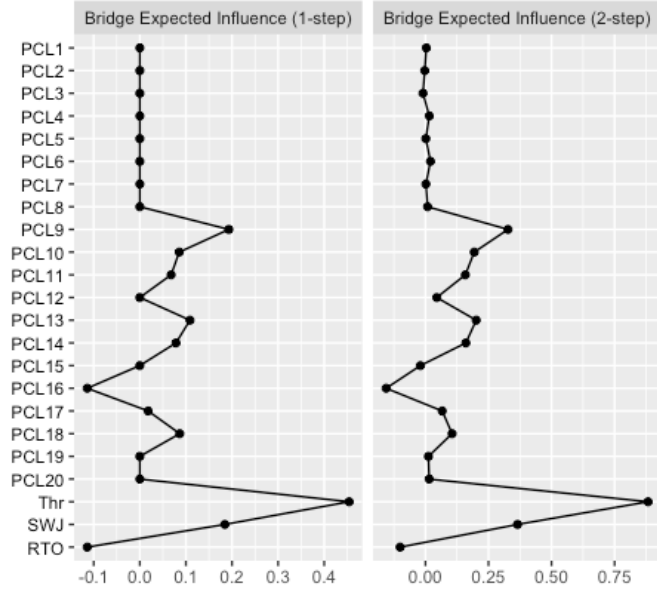


Figure 2. Expected node influence for cognitive GGM network (left) and functional contextualist GGM network (right). See Table 2 for label names.

### Cognitive Graph



### Functional Contextualist Graph

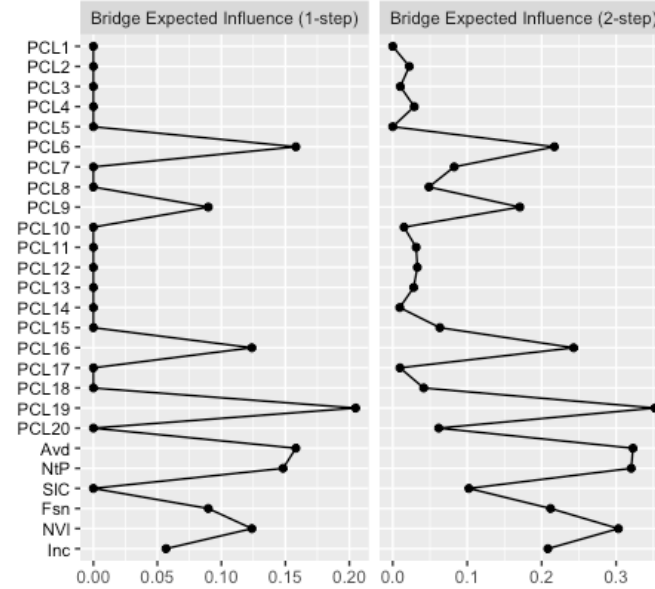
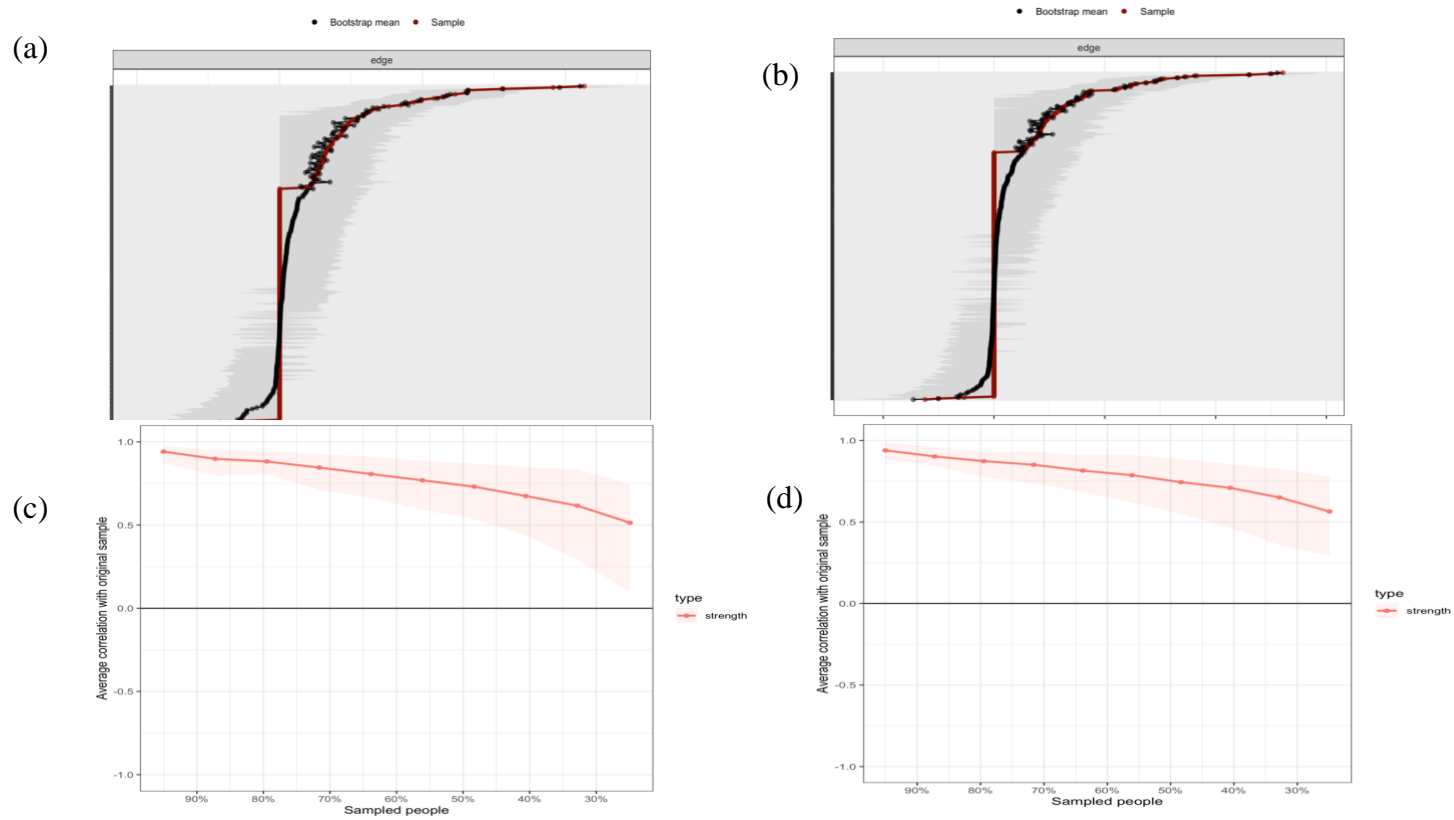
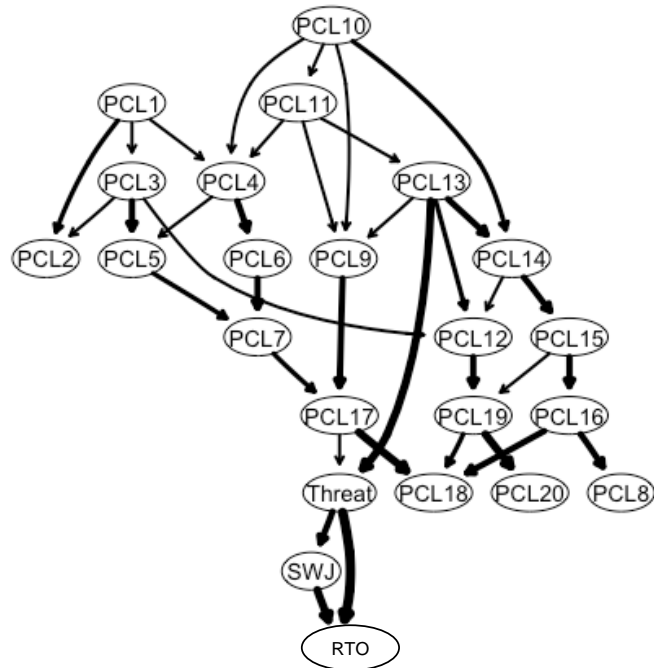


Figure 3. One- and two-step bridge expected influence for cognitive GGM network (left) and functional contextualist GGM network (right). See Table 2 for label names.



*Figure 4.* Bootstrapped confidence intervals for estimated edges in the cognitive GGM (4a) and functional contextualist GGM (4b). Correlation of node strength in case-dropped networks and original networks (4c: cognitive; 4d: functional contextualist). Shaded areas represent 95% confidence intervals.

### Cognitive Graph



### Functional Contextualist Graph

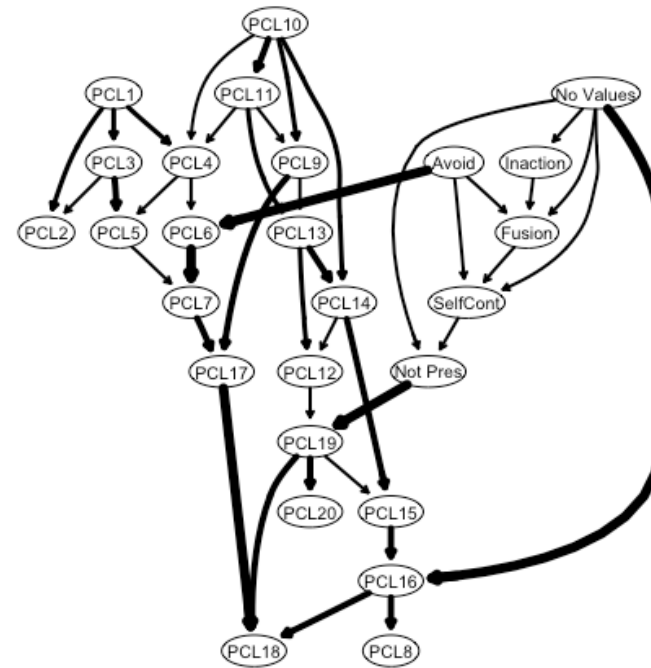
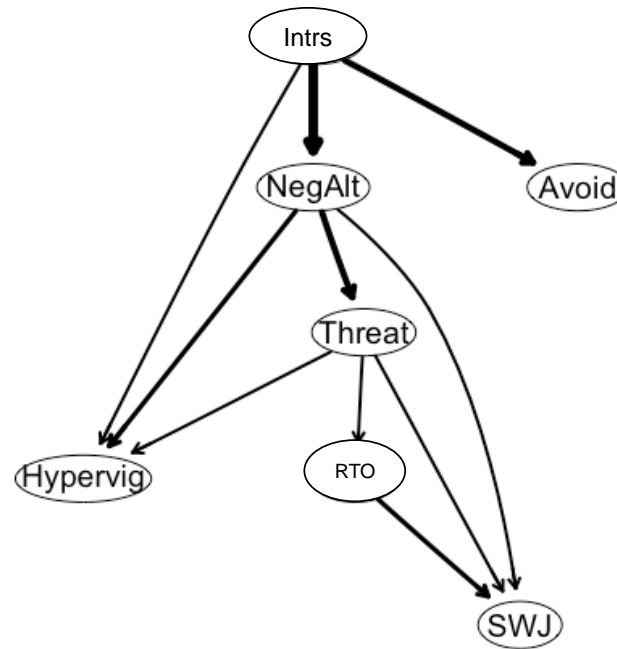


Figure 5. Directed acyclic graphs for the cognitive (left; without model specification) and functional contextualist (right; with model specification) networks. Thicker lines indicate greater probability of causality. See Table 2 for label names.

## Cognitive Graph



*Figure 6.* Directed acyclic graphs for the cognitive with PTSD symptoms collapsed into clusters. 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.