2018-08-22

Obsessive-Compulsive Disorder, Trauma, and Stress: A Network Approach

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OBSESSIVE-COMPULSIVE DISORDER, TRAUMA, AND STRESS: A NETWORK APPROACH

By

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A DISSERTATION

Submitted to the Faculty
of the University of Miami
in partial fulfillment of the requirements for
the degree of Doctor of Philosophy

Coral Gables, Florida

December 2018

UNIVERSITY OF MIAMI

A dissertation submitted in partial fulfillment of
the requirements for the degree of
Doctor of Philosophy

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CARBONELLA, JULIA YANG (Ph.D., Psychology)
Obsessive-Compulsive Disorder, Trauma, and Stress: A Network Approach. (December 2018)

Abstract of a dissertation at the University of Miami.

Dissertation supervised by Professor Kiara Timpano.
No. of pages in text. (125)

Experience of traumatic life events (TLEs) has consistently been identified as a risk factor that can trigger and exacerbate obsessive compulsive symptoms (OCS). At the clinical level of severity, OCD and post-traumatic stress disorder (PTSD) are also highly comorbid at rates greater than in the general population. However, much remains unclear about the differential interrelations between symptoms of OCD and PTSD, as well as the influence of a traumatic history on the connectedness of hallmark features of OCD. The overarching study applied network analysis – a recently developed tool that can help shed light on symptom structure and relations – to examine OCD, traumatic events, and PTSD from two separate but complementary perspectives. With a sample of individuals with lifetime OCD, Study 1 took first steps to examine the structure of OCD as measured by symptom dimensions in conjunction with obsessions and compulsions severity. Findings highlighted aggressive, sexual, religious obsessions and checking compulsions as a central component through which other OCD features are connected. It also provided qualitative evidence of a more densely connected network of OCD components in those with a trauma history, in support of TLEs as an important vulnerability factor for OCD. Study 2 expanded upon these findings with a separate sample of treatment-seeking OCD

individuals, investigating symptom-level relationships between the heterogeneous syndromes of OCD and PTSD. As clinical correlates, anxiety and depression were not found to explain any symptom overlap between OCD and PTSD, which were found to form more independent symptom constellations than expected. Interestingly, OCD nodes were found to form two clusters of a) control/resistance and b) time occupied, interference, and distress; meanwhile, re-experiencing symptoms were highlighted as the central PTSD symptom type. Overall, findings illustrate the utility of the network approach in studying psychopathology, and point towards mechanistic links between symptoms that may be important to target in clinical interventions for trauma and OCD.

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Chapter 1: General Introduction

Obsessive Compulsive Disorder (OCD) is an extremely impairing condition that affects approximately 2% of the population and has been identified as one of the top ten leading forms of disability worldwide (Benito & Storch, 2011). The distinct core features of OCD are comprised of recurrent, intrusive thoughts, as well as compulsive behaviors often repeated in a certain nature in an attempt to neutralize these obsessions (Mataix-Cols, Rosario-Campos, & Leckman, 2005). As a notoriously chronic and burdensome syndrome, OCD is not only associated with financial and psychosocial difficulties, but is often severely distressing to both the individual and their family (Adam, Meinlschmidt, Gloster, & Lieb, 2012; I. S. Fontenelle et al., 2010; Fullana et al., 2009; Lopez & Murray, 1998). Obsessive compulsive symptoms (OCS) are dimensionally distributed (Abramowitz et al., 2010; Olatunji, Williams, Haslam, Abramowitz, & Tolin, 2008), and up to 8.7% of the general population suffer from subclinical OCS, which in and of themselves can cause notable distress and impairment not only in terms of psychological wellbeing, but also in social and workplace interference (Adam et al., 2012; Angst et al., 2004).

The conceptualization of OCD presented in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5; American Psychiatric Association, 2013) focuses almost exclusively on obsessions or compulsions as a means to characterize this syndrome. Similarly, the cognitive-behavioral model of OCD highlights the functional relationship between obsessive beliefs, obsessions, and compulsions (Rachman, 1997; Salkovskis, 1985). It purports

that high levels of obsessive beliefs, such as over-estimation of threat, over-importance of thoughts, and inflated responsibility, lead one to misinterpret the value of naturally occurring and common intrusive thoughts (e.g., “my spouse is going to die in a car accident”) (Rachman, 1997). The experience of these unwanted recurrent thoughts, distorted by the interpretation of intrusive thoughts as dangerous, can cause a great deal of anxiety (Rachman, 1997). This distressing emotion can drive an individual to perform certain compulsive rituals, whether mental or behavioral, in an effort to alleviate the anxiety associated with these intrusive thoughts or to “cancel them out” (e.g., repeating a prayer four times) (Swinson, Antony, Rachman, & Richter, 2001; Taylor, Abramowitz, & McKay, 2007). Yet, by lowering anxiety, the compulsion registers as actually helping prevent the harmful intrusion, which over time reinforces the maladaptive obsessive beliefs and perpetuates the vicious cycle (Swinson et al., 2001; Taylor et al., 2007). In a nutshell, obsessions give rise to corresponding compulsions, which then effectively reinforce obsessions.

However, it is important to note that the thematic content of obsessions and compulsions can vary dramatically from patient to patient. This heterogeneity complicates the manner in which OCD is operationalized and measured, as well as our understanding of vulnerabilities and risk factors. A large body of research has focused on trying to explain the extreme diversity of OCD symptoms, while simultaneously building off the cognitive-behavioral understanding that obsessions and compulsions are functionally connected. A series of reports conducted factor analyses on the most commonly endorsed OCS (Abramowitz et

al., 2010; Mataix-Cols et al., 2005; McKay et al., 2004), and have consistently found support for four or five primary symptom dimensions, including: checking and responsibility for harm, contamination/washing, symmetry/ordering, hoarding[1], and unacceptable or repugnant obsessions (e.g., sexual, aggressive, or religious). Of note, each symptom dimension considers both obsessions and compulsions that are thematically connected, each theme reflecting a distinct functional relationship between the two. Research investigating specific OCS dimensions has revealed ties to varying levels of psychiatric comorbidity (Hasler et al., 2005; McKay et al., 2004), as well as varying levels of treatment efficacy (Abramowitz, Franklin, Schwartz, & Furr, 2003; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002; Rufer, Fricke, Moritz, Kloss, & Hand, 2006), both of which complicate our overall conceptualization of the etiological model of OCD. For instance, one study found checking compulsions and aggressive, sexual, and religious obsessions to bear unique associations with comorbid depression and anxiety disorders (Hasler et al., 2005). The symptom dimensions have also been associated with differential neural regions implicated in cognitive and emotional processing (Mataix-Cols et al., 2004). For example, activation of bilateral prefrontal regions and right caudate nucleus have been linked with washing/contamination symptoms, while activation of the putamen, thalamus, and dorsal cortical areas have been associated with the checking/harm dimension (Mataix-Cols et al., 2004). The research on symptom dimensions

[1] Hoarding symptoms have traditionally been categorized as a symptom subtype of OCD; however, recent evidence suggests that hoarding symptoms can either emerge as a result of hoarding disorder, a discrete condition in DSM-5, or reflect symptoms of OCD (Morein-Zamir et al., 2014; Pertusa et al., 2008; Rachman, Elliott, Shafran, & Radomsky, 2009).

therefore indicates the necessity for further investigation of the phenotypic profiles, their associated vulnerabilities, and their relationships to comorbid psychopathology.

Relationship between OCD, Stressful or Traumatic Life Events, and PTSD

Stressful life events (SLEs) are remarkably common, affecting about a third of the population within the past year alone (Wethington & Kessler, 1986). Importantly, even within this normative range, general life stress has been identified as a crucial vulnerability factor for OCS (Bogetto, Venturello, Albert, Maina, & Ravizza, 1999; Cromer, Schmidt, & Murphy, 2007; Real et al., 2011). OCS are notorious for their tendency to wax and wane over time (Steketee, Eisen, Dyck, Warshaw, & Rasmussen, 1999), which is likely exacerbated by the occurrence of psychosocial stressors (see Cromer et al., 2007; Lin et al., 2007). SLEs have been noted to cause more frequent intrusive thoughts (Brewin, Gregory, Lipton, & Burgess, 2010; Frewen, Schmittmann, Bringmann, & Borsboom, 2013). Compared to healthy controls, those with OCD report significantly more SLEs during the year prior to OCS onset (Gothelf, Aharonovsky, Horesh, Carty, & Apter, 2004; McKeon, Roa, & Mann, 1984). Yet, despite the high likelihood of experiencing an adverse event (Kessler, 1996), many gaps of knowledge remain about the downstream effects on OCS. Further research may help clarify the specific role of SLEs in the onset of obsessional thinking and symptoms.

More severe in nature than SLEs, traumatic events have also been linked with greater OCS. Evidence suggests a connection between OCS and childhood

trauma, of both emotional and physical natures (Lafleur et al., 2011; Lochner et al., 2002; Mathews, Kaur, & Stein, 2008). In adults, case studies have also documented onset of OCS following a severe traumatic event, such as combat exposure (de Silva & Marks, 1999, 2001; Pitman, 1993). For example, de Silva and Marks (1999) described the case of Mrs. T., a middle aged woman who had witnessed the fatal shooting of her brother. She experienced recurrent and vivid mental images of her brother collapsing, covered in blood. Over time, she developed the habit of focusing on a countering image of her brother, alive and healthy at a younger age. She engaged in this mental ritual compulsively, which reduced her anxiety in the short-term. However, after a while, Mrs. T. found that she began to apply this tactic more generally – she was compelled to neutralize any distressing or negative thought with a positive or idealistic image. She had also developed specific phrases that she would repeat silently to accompany the comforting image. Given her cognitive compulsions that had generalized beyond intrusive thoughts of the shooting, her daily functioning was severely impacted.

As it stands, this literature clearly indicates that trauma is linked with higher occurrence of OCS and greater severity of OCD; however, it is less clear how the experience of an extremely adverse event would relate to specific types of OCS. There are conflicting reports of which OCS dimensions may bear the strongest link to a history of traumatic events; for instance, one study suggesting contamination/cleaning symptoms (Real et al., 2011) and another indicating obsessions/checking and symmetry/ordering (Cromer et al., 2007). More

research is warranted to clarify whether experience of a traumatic event may yield different outcomes with regards to the trajectory of OCS.

Given the link between OCD and SLEs or traumatic events, this raises the question of how symptoms of post-traumatic stress disorder (PTSD) may bear a unique connection with those of OCD. It is rather striking that PTSD is a syndrome that is often comorbid with OCD, at rates much higher than expected in the general population - about 20% of OCD patients meet criteria for lifetime PTSD (Huppert et al., 2005; Ruscio, Stein, Chiu, & Kessler, 2010). In general, comorbidity of psychiatric disorders is consistently linked to a range of negative outcomes, including more severe daily impairment, greater need for treatment, and even higher suicidality (e.g., Brown & Barlow, 1992; Bruce et al., 2005; Nock, Hwang, Sampson, & Kessler, 2010). Comorbid PTSD is therefore associated with a more severe clinical OCD presentation, and can result in worse treatment outcomes (Gershuny, Baer, Jenike, Minichiello, & Wilhelm, 2002; Gershuny et al., 2008). Further complicating our understanding of the co-occurrence of OCD and PTSD, there is burgeoning evidence in support of a subtype of “post-traumatic OCD” that is distinct to those who have developed OCS after trauma; comparing those who developed OCD either before or after a traumatic event have been found to not only differ on age of onset and suicidal ideation, but also in more severe contamination/washing and miscellaneous OCS (L. F. Fontenelle, Cocchi, Harrison, Miguel, & Torres, 2011; L. F. Fontenelle et al., 2012).

Yet, despite its relative over-representation in the OCD population, much remains unclear about the interplay between PTSD symptoms and OCS. Given the markedly heterogeneous nature of both syndromes, further research is warranted at the symptom level to evaluate how specific symptoms of PTSD may differentially contribute towards OCS dimensions. For instance, one possible theoretical mechanism contributing to overlap between PTSD and OCD is that intrusive thoughts, initially associated with re-experiencing the trauma, eventually develop into full-fledged obsessions (de Silva & Marks, 1999). This connection may be facilitated by several cognitive vulnerability factors of OCD that may be activated or exacerbated by PTSD symptoms (de Silva & Marks, 1999), such as over-estimation of threat, elevated moral standards, and catastrophic interpretations of the importance of thoughts (Rachman, 1997). Another (not mutually exclusive) possibility is that behaviors originally performed as a consequence of suffering from PTSD (e.g., checking one's environment, repetitive washing, or neutralizing distressing mental images) eventually crystallize as compulsive rituals, and consequently reinforce associated intrusive thoughts to perpetuate the cycle of obsessions and compulsions (de Silva & Marks, 1999; Rhéaume, Freeston, Léger, & Ladouceur, 1998). These proposed pathways are in need of greater empirical support to clarify whether specific symptoms of PTSD may predispose one towards particular OCS dimensions (de Silva & Marks, 2001). Also, given that the majority of extant studies have broadly examined general diagnostic indicators or total sum-scores (Grabe et al., 2007; Huppert et al., 2005; Mathews et al., 2008; Nacasch, Fostick, & Zohar, 2011),

more research is warranted on the symptom-level to better understand the specificity of the connection between PTSD facets and OCS dimensions.

Overall, both OCS and the experience of trauma and/or SLE are remarkably heterogeneous conditions. There is clearly a unique connection between the two constructs, consistently demonstrated across general life stress, traumatic events, and PTSD. Additional investigations into how the diverse symptoms of OCD and PTSD map onto one another, as well to stressful or traumatic life events, will help shed light on their complex, dynamic connection and aid in improving treatment targets and outcomes (de Silva & Marks, 1999; Gershuny, Baer, Radomsky, Wilson, & Jenike, 2003; Rachman, 1991).

Latent Variable versus Network Analysis Approach

The present research aims to utilize network analysis as a novel perspective to explore the intricacies of how OCS and trauma are connected. By simultaneously examining the interconnections linking together a group of symptoms, networks can help complement more established methods for examining relationships between constructs, such as the latent variable approach. Traditionally, psychological disorders have been conceptualized as latent entities. For instance, MDD is typically modeled as a latent variable that, rather than being measured directly, gives rise to observable symptoms (e.g., depressed mood, or loss of interest in usual activities) (Borsboom, 2008; Borsboom, Mellenbergh, & van Heerden, 2003). However, this latent variable approach bears several critical flaws upon closer examination. For one, it assumes a typical medical disease model that may not be appropriate for mental

disorders. As Borsboom and Cramer (2013) describe with the following example, it is possible to identify a distinct medical condition that is separate from its symptoms; one can have a brain tumor without headaches, and one can have headaches without a brain tumor. However, if a patient has both headaches and a brain tumor, and the brain tumor is in fact the root cause of the headaches, removing the brain tumor (the underlying condition) would directly alleviate the headaches (the symptom). Yet, this medical model becomes problematic for psychological disorders. Applying similar logic, it would thus be possible to have MDD as an underlying latent condition without also having its defining symptoms of depressed mood or anhedonia, and vice versa, which is a conceptual nonstarter (Borsboom & Cramer, 2013). On the contrary, rather than disorders as latent diseases that cause downstream observable symptoms, network theorists propose that these symptoms themselves are actually what comprise psychological disorders. This perspective calls for greater focus on individual symptoms and how they interact with one another and with associated factors, rather than further attempts to fit psychological disorders into the medical model as latent conditions that underlie a shared set of symptoms.

Of particular interest to this investigation, the latent variable approach falls short when considering the question of comorbidity in psychopathology – a central conundrum that has challenged the field for decades (e.g., Angold, Costello, & Erkanli, 1999; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Lai, Cleary, Sitharthan, & Hunt, 2015; Mineka, Watson, & Clark, 1998). With a latent variable measurement model, two disorders themselves covary, each

underlying a certain set of symptoms (Borsboom et al., 2003). However, this is problematic in violating the assumption of local independence (i.e., that symptoms of a latent disorder are independent from one another) (Borsboom, 2008). For purposes of latent model analysis, the covariance of the observable symptoms is attributed to the role of the covarying latent variables as the "common cause" (Cramer, Waldorp, van der Maas, & Borsboom, 2010; Robinaugh, LeBlanc, Vuletich, & McNally, 2014). Akin to an example provided by Cramer, Borsboom, Aggen, and Kendler (2012), an underlying episode of MDD is a singular "common cause" that gives rise to depressive symptoms, and traumatic events impact depressive symptoms only indirectly through this latent syndrome of MDD (see Figure 1.1). Yet, our existing knowledge of symptoms' interconnectedness contradicts this approach. For instance, if you have sleep disturbances, you are more likely to be fatigued. Both are DSM symptoms of MDD, yet clearly interact directly and causally with one another; it is not the case that MDD separately gives rise to both sleep disturbances and fatigue, as the "common cause" latent model would assert (Borsboom & Cramer, 2013; Cramer et al., 2010).

In contrast with the latent perspective, the network analysis approach provides an alternative framework from which to examine dynamic models of psychopathology, by examining relationships between symptoms themselves, rather than delineating distinct latent disorders. It takes the stance that symptoms cannot be separated from – and do not merely reflect the presence of – an underlying disorder, but are what actually constitute the disorder (McNally et al.,

2015). This redirects the spotlight towards understanding the specific symptoms of a syndrome and how they are directly related to one another (or with those of a comorbid disorder) (Borsboom & Cramer, 2013; Robinaugh et al., 2014). Figure 1.2 shows this alternative network approach, in line with Cramer et al. (2012)'s theoretical depiction of comorbidity. In contrast with the latent variable approach presented in Figure 1.1, traumatic events can impact these depressive symptoms directly. Importantly, as Figure 1.2 emphasizes, depressive symptoms are thought to cluster together not due to a common underlying cause of an episode of MDD, but because of the direct causal relationships between them (Borsboom & Cramer, 2013; Robinaugh et al., 2014). Thus, not only do network analyses adequately represent disorders as the complex systems they are, but modeling networks also nullifies the axiom of local independence by embracing the observed casual relations amongst symptoms (Borsboom & Cramer, 2013; Cramer et al., 2010; McNally et al., 2015; Robinaugh et al., 2014).

Implications for using a network approach have only recently been explored with regards to psychopathology, but as the "new game in town" (Borsboom & Cramer, 2013, p. 93), it bears potential for numerous benefits. Network analyses are helpful in shedding light on comorbidity, demonstrating how symptoms may be likely to co-occur with or trigger other symptoms regardless of whether the diagnostic threshold has been met (Fried, 2015). Initial networks of DSM symptoms have evidenced a "small world structure" where one can "jump" from one symptom to another in several steps, which provides bountiful fodder for a closer exploration of comorbidity and its mechanisms

(Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011; Goekoop & Goekoop, 2014).

The capability to examine node centrality is a key benefit of network techniques; the more central a node is, the more direct connections it has with other nodes in the network, and thus bears greater influence relative to these other symptoms (Borsboom & Cramer, 2013). As an apt analogy, Fried (2015) liken a highly central symptom to a celebrity's greater influence on a social media network; if one has more "followers" and connections, any information shared is more likely to quickly spread throughout the social network. Central nodes are more likely to spark the onset of other symptoms in the network and are thus more "dangerous" factors that bear the greatest level of risk (Borsboom & Cramer, 2013, p. 114). Nodes with high strength centrality are considered "ripe as targets for clinical interventions" (McNally, p. 3). The main indicator of centrality is *node strength*, defined as the sum of the weights of each edge connected to the node. This is a stable and commonly-used metric in network analyses (Fried, 2015; Opsahl, Agneessens, & Skvoretz, 2010); it provides a quantifiable value of the total "impact factor" a specific node has on all other nodes in the network. In the following studies, node strength will be the primary outcome of centrality.

Similarly, edges between nodes in each network – essentially, how connected they are to other nodes – can be quantified, which can help identify links between symptoms that are more likely to co-occur (Fried, 2015; Robinaugh et al., 2014). For example, network methods have recently been applied to help

conceptualize several psychopathological symptom models, such as PTSD (McNally et al., 2015), depression (Cramer et al., 2012; Fried, 2015; van Borkulo et al., 2015), and complicated grief (Robinaugh et al., 2014). Many of the connections between symptoms have been illuminating; for instance, McNally et al. (2015) inferred a strong causal relation between anger and concentration problems in PTSD individuals, an link overlooked by extant research that warrants further investigation and clinical attention.

Investigating the centrality of symptoms and edge weights between them is a novel approach that can complement our knowledge of the etiology and maintenance of syndromes. For instance, analyses of symptom severity have helped advance our knowledge of disorders, but do not necessarily equate to how fundamental the symptom is within the full context of a given network (Fried, 2015; Opsahl et al., 2010). These insights may yield important information for targeted intervention as well as prevention. Moreover, it has recently become possible to compare the overall connectivity of two networks, which can help reveal different symptom profiles such as that of individuals who experience symptom remission, versus those who suffer from a chronic course of illness (van Borkulo et al., 2015). Better understanding of these group differences can further our understanding of how psychopathology can wax and wane, as well as how to best treat symptoms accordingly.

Network Analysis and OCD

OCD has long been considered a latent entity from which its core features of obsessions and compulsions arise. From this perspective, an individual “has”

the underlying disease of OCD, which consequently causes these observable symptoms as indicators of OCD's presence (McNally et al., 2015). In contrast, the network approach conceptualizes OCD not as a latent variable, but as a dynamic system comprised of interrelated symptoms. These symptoms are not assumed to covary due to an overarching mental disorder as a shared cause, but as a result of their interrelated effects on each other (McNally et al., 2015). An episode of OCD (or any other psychological disorder) is seen to occur when enough symptom nodes "turn on, transmitting activation to connected nodes, and settling into a pathological equilibrium" (McNally et al., 2015, p. 839). Network analyses aim to identify these potential causal relations, in an effort to shed light on how specific factors may be more likely to co-occur with neighboring nodes and reinforce a cascade of related symptoms (McNally et al., 2015).

Currently, not much is known about the putative network structure of OCS. An initial study has examined certain OC dimensions strictly with regards to autism in a child sample, and found compulsions to be influential with regards to repetitive behaviors in autism (Ruzzano, Borsboom, & Geurts, 2015). There is also recent evidence that specific OCS, such as distress associated with obsessions, may be more central as bridge symptoms helping to explain the comorbidity between OCD and depression (McNally, Mair, Mugno, & Riemann, 2017). Yet, the structure of the overarching network of OCD's hallmark features remains unclear, particularly when considering multiple indicators such as severity and symptom dimensions. The cognitive behavioral model of OCD provides a broad theoretical framework for how obsessions and compulsions

generally interact (Rachman, 1997), but utilization of network analyses will help narrow down specific OCS factors that may be more central in perpetuating the cycle of symptoms.

The network approach may additionally be beneficial in helping to clarify the nature of symptom overlap between OCD and PTSD that may underlie their unexpectedly high rates of comorbidity. Networks of these syndromes at the symptom level can examine these factors simultaneously to determine which specific symptoms may be of greater relative importance inconsidering the comorbidity of these two disorders. Rather than adopting the latent variable perspective of OCD and PTSD as two separate entities that happen to frequently co-occur at high rates, a network approach helps explain their comorbidity as a direct result of having interrelated symptoms (Cramer et al., 2010; Robinaugh et al., 2014). One example of this type of approach is a recent article by Robinaugh et al. (2014), which used the network analysis approach to examine the overlap of depressive symptoms and complicated grief due to bereavement. The authors discovered that specific symptoms – including loneliness and emotional numbness – served to bridge the two networks, potentially contributing to the high rates of comorbid depression and complicated grief. In this view, one does not just happen to suffer from both depression and complicated grief, but because overlapping symptoms are functionally interwoven. A similar process was found in Cramer et al. (2010)'s examination of MDD and generalized anxiety disorder (GAD); certain symptoms such as fatigue and sleep disturbances were found to be highly central, and may play a large role in developing other

symptoms of both MDD and GAD. An examination of OCD dimensions and PTSD symptoms may similarly point to specific factors that may help explain comorbidity.

With its ability to consider multiple symptoms simultaneously, networks can highlight specific factors as central or peripheral in within the nodes included in the network, hence expanding our understanding of “hot button” symptoms and heterogeneous symptom profiles that characterize OCD. As such, analyses can help to clarify uncertainties raised by the extant body of literature, such as those regarding whether a history of trauma can affect the connection between OCD symptoms, differential relations between OCD dimensions, and the role of specific clinical correlates. Importantly, networks can also shed light on putative downstream connections for how particular OCD symptoms may influence not only one another but also PTSD symptoms. Altogether, network analysis is an intriguing approach that may provide a more fine-grained insight in comparison to previous investigations. Findings can not only help us better understand the structure of individual OCS, but can also illuminate complex, dynamic interactions between symptoms of OCD and trauma, which thus far have been difficult to tease apart. The overall program of research aims to illustrate the map of associations between these realms and highlight symptoms that are most central to the network, as a novel empirical contribution towards entangling sources of comorbidity. As the first investigation to-date using a network approach to investigate these symptoms together, findings will aim to identify

central “hotspots” and functional relationships between symptoms of OCD and a traumatic background.

Overview of Studies

Within this overarching investigation, we will use network analyses to investigate the comorbidity of OCD and the overarching construct of a traumatic history, from two separate, but related perspectives across two studies. Study 1 will take a first look with a large sample of those with lifetime clinical OCD to examine the structure of OCD as measured by the hallmark features of obsessions and compulsions severity and symptom dimensions. This study will also determine whether OCD networks differ between those with and without a history of trauma, to help shed light on whether traumatic events may yield more densely and differentially connected networks. Study 2 will expand upon these findings on a separate sample of treatment-seeking clinical OCD individuals, to clarify symptom-level relationships that may help explain the heterogeneous nature of OCD and PTSD. Networks will reveal whether particular PTSD symptom clusters may differentially relate to specific indicators of OCD severity, as well as whether clinical correlates of anxiety and depression may play a role in symptom overlap. Taken together, the findings of these studies will help identify specific factors that are central in the complex relation between OCD and trauma, as well as potential mechanisms to explore in future research.

Chapter 2: Study 1 – OCD Network and Trauma History

Background

OCD is an impairing condition that is comprised of unwanted, repetitive obsessions, as well as corresponding compulsive rituals (Mataix-Cols et al., 2005). The cognitive-behavioral model of OCD provides a broad theoretical framework for how obsessions and compulsions are functionally related. High levels of maladaptive obsessive beliefs (e.g., over-estimation of threat and inflated responsibility) lead to misinterpretation of the value of intrusive thoughts (Rachman, 1997), which in turn, triggers distress in response to intrusive thoughts. In order to neutralize the anxiety and distress, an individual with OCD will then engage in compulsive mental or behavioral rituals (Swinson et al., 2001; Taylor et al., 2007). Yet, by effectively reducing anxiety in the moment, the compulsion over time reinforces the dysfunctional obsessive beliefs and perpetuates a vicious cycle (Swinson et al., 2001; Taylor et al., 2007).

Despite this clear theoretical conceptualization, there remains much to be explored with regards to the distinct components of OCD's nomological net. Aside from overarching symptom severity, OCD is notoriously heterogeneous in symptom presentation, which complicates our understanding of how specific symptoms play a role in its onset and maintenance. It is difficult to conceptualize OCD as a cohesive syndrome without a more thorough understanding of how these symptom dimensions may interact not only with one another but in the context of total obsessions and compulsions severity as well. It is also important

to gain further insight into how these dimensions and severity are differentially connected to other syndromes comorbid with OCD.

Thus far, relying upon common statistical methods of regression has proved challenging in considering all of these factors in conjunction, particularly at a clinical level of severity. Multiple regression not only focuses on one outcome variable at a time, but also precludes controlling for all other variables within the scope of each study. The network approach is a novel statistical method that may be helpful in examining these diverse factors simultaneously to highlight specific aspects of OCD and corresponding symptom links that may play an important role in connecting other symptoms and maintaining this debilitating condition. The traditional latent model presents OCD as an underlying entity that gives rise to obsessions and compulsions, and manifests in varying observable symptoms. Conversely, the network approach proposes that specific symptoms themselves are actually what comprise psychological disorders. This perspective emphasizes the connectedness between individual symptoms and associated factors, thus expanding our understanding of “hot button” symptoms and heterogeneous presentations. Examining a range of symptoms simultaneously in a network may shed light on specific components that could play a central role in perpetuating OCD and “triggering” related features, thus maintaining this chronically impairing disorder.

Burgeoning evidence from network analyses of other psychiatric disorders have been illuminating: for instance, highlighting loneliness and emotional numbness as critical features contributing to the overlap between complicated

grief and depression (Robinaugh et al., 2014), or the importance of obsessional distress in activating depression in those with OCD (McNally, Mair, et al., 2017). With respect to our own investigation, we hope to highlight specific OCD features that may play a substantial role in activating other clinically relevant symptoms. Network examinations of both OCD severity as well as symptom type will provide a more fine-grained insight into the plausible structure of OCD. A network approach can also allow one to explore the potential impact of specific risk factors (e.g., having a history of trauma). Examining the causal structure of OCD features will help fill in the gaps of how specific characteristics bear high risk in maintaining related symptoms and comorbid symptoms when “turned on.” Thus, a pilot network in a population of those with lifetime OCD can highlight how specific symptoms may interact when considered as a whole, thus advancing our functional analysis of OCD as well as its relation with co-occurring syndromes.

As aforementioned, OCD is remarkably diverse in its symptom dimensions, which can complicate our conceptualization of OCD alongside symptom severity. Commonly identified OCD dimensions include responsibility for harm, contamination, symmetry/order, and unacceptable or repugnant thoughts (e.g., sexual, aggressive, or religious; Abramowitz et al., 2010; Mataix-Cols et al., 2005; McKay et al., 2004). Of note, experiencing OCD symptoms in one dimension is not mutually exclusive from experiencing symptoms in another (i.e., one could be either high/low in contamination/cleaning and high/low in symmetry/ordering). Given that OCD symptom dimensions have previously evidenced differential links with varying psychiatric comorbidity (Hasler et al.,

2005; McKay et al., 2004), the occurrence of traumatic life events (Cromer et al., 2007), and treatment efficacy (Abramowitz et al., 2003; Mataix-Cols et al., 2002; Rufer et al., 2006), it would be illuminating to consider symptom dimensions when investigating the overarching network of OCD.

An additional consideration relevant for network analysis is that OCD is also remarkably comorbid with other conditions, with 90% of those with lifetime OCD meeting criteria for another lifetime disorder (Ruscio et al., 2010). Ruscio et al. (2010)'s nationally-representative study found anxiety (76%) and mood (63%) disorders to be most common; 39% reported a lifetime substance use disorder as well. Comorbidity in association with OCD is linked with worse treatment outcomes (e.g., Gershuny et al., 2002; Gershuny et al., 2008; Overbeek, Schruers, Vermetten, & Griez, 2002; Pallanti, Grassi, Cantisani, Sarrecchia, & Pellegrini, 2011), and is generally accompanied by greater severity of symptoms and impairment (e.g., Brown & Barlow, 1992; Bruce et al., 2005; Nock et al., 2010). Thus, comorbid disorders are clinically relevant features to consider in the exploration of OCD, particularly in the application of a network analysis that may reveal differential connections to specific OC symptoms. Further research is warranted to evaluate whether and how specific OCD features may act as a bridging symptom with comorbid syndromes.

A growing body of literature has implicated traumatic life events as a key risk factor in the genesis and manifestation of OCD. While not all individuals with OCD report experiencing a traumatic event, a staggering 54% of patients do endorse a positive history (Cromer et al., 2007). Traumatic events experienced in

childhood, both emotional and physical types, have been linked with greater OCD symptoms (Briggs & Price, 2009; Lafleur et al., 2011; Lochner et al., 2002; Mathews et al., 2008). Trauma experienced in adulthood (de Silva & Marks, 1999, 2001; Nacasch et al., 2011; Sasson et al., 2005) have been linked to greater subsequent OCD severity. It is thus important to consider how having experienced trauma may yield differences in how specific OCD components interact with one another, as well as with comorbid features. In other words, trauma may be a vulnerability factor for developing a more densely connected network of OCD nodes, such that individuals who endorse having experienced trauma may be more predisposed to make “jumps” between OCD symptoms and comorbid features.

Case studies have indicated that after experiencing a traumatic event, many individuals have a tendency to develop both obsessive and compulsive tendencies in general (de Silva & Marks, 1999, 2001; Pitman, 1993). It may be the case that anxious responding to trauma increases the occurrence of unwanted intrusive thoughts, which “are after all the raw material for full obsessions” (Rachman, 1997, p. 797). Repetitive rituals following trauma may easily generalize into full-blown compulsions; meanwhile, faulty misattributions (e.g., heightened guilt after a trauma) could develop into obsessive beliefs (de Silva & Marks, 1999, 2001; Pitman, 1993). In this manner, if one specific symptom onsets, having a traumatic history may put individuals at higher risk of developing other OCD symptoms across the board. In particular, evidence is also mixed with regards to whether specific symptom dimensions may be more

strongly linked with a history of trauma; one study points to contamination/cleaning symptoms (Real et al., 2011) while another indicates obsessions/checking and symmetry/ordering (Cromer et al., 2007). Comparing the full network of OCD symptoms across those with a history of trauma and those without a history of trauma may help clarify whether a traumatic background yields a different presentation of OCD symptoms and related features. Overall, if networks are found to differ between those who have and have not suffered a traumatic life event, trauma may be implicated as a critical factor that has far-reaching consequences for how easily OCD symptoms – and comorbid disorders – are activated in response to one another.

Aim 1: A First Look at the Network Structure of OCD.

Aim 1 was to examine the putative causal structure of OCD as measured by its hallmark features of obsessions and compulsions, operationalized using the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) subscales (see Methods below). This is the first study to our knowledge to do so in conjunction with factor scores derived from the primary OCD symptom dimensions, including: repugnant obsessions/checking, symmetry/ordering, contamination/cleaning, and hoarding[2] (Hasler et al., 2005). It is important to note that *factor scores* are a measure of how many symptoms are reported by an individual within each symptom dimension. In contrast, *overall severity* assesses the degree to which obsessions

[2] Of note, the present database was collected prior to Hoarding Disorder (HD) being classified as a disorder separate from OCD in the DSM-5. As such, individuals were not assessed or screened out for meeting clinical criteria for Hoarding Disorder, and as a result concurrent HD and OCD cannot be ruled out. Thus, hoarding as a derived factor was retained in our network analyses, as we could not distinguish between “OCD-type HD” or HD as a distinct disorder.

and compulsions are debilitating to the patient, as a general measure of distress, interference, frequency, control, and resistance – rather than symptom count within dimensions. Given the complex nature of OCD, both factor scores and symptom severity across symptom dimensions will be considered simultaneously, yet as separate entities. Both conceptualizations are important to consider in an effort to capture and characterize the remarkable heterogeneity of this disorder; for instance, greater severity is associated with greater comorbidity and quality of life (Brown & Barlow, 1992; Bruce et al., 2005; I. S. Fontenelle et al., 2010; Nock et al., 2010), while symptom dimensions bear differential links to risk factors and treatment outcomes (Abramowitz et al., 2003; Cromer et al., 2007; Hasler et al., 2005; Mataix-Cols et al., 2002; McKay et al., 2004; Rufer et al., 2006). It may be the case that severity of symptoms, obsessions in particular, plays a more central role in connecting the OCD network and comorbid features. Alternatively, a particular symptom dimension may be more crucial in linking together the overall OCD network. That finding would support the notion that a particular OCD subtype may yield to a factor profile accompanied by greater psychiatric comorbidity (Hasler et al., 2005).

Given the important role of comorbidity in our conceptualization of OCD, the network additionally included three indicators of comorbidity: number of comorbid anxiety disorders, number of comorbid mood disorders, and number of comorbid alcohol/substance disorders. As an exploratory variable, we also included the number of comorbid OC-spectrum disorders, as it is largely unclear how they may differentially relate to the specific OCD components of severity and

symptom dimensions. Finally, age of OCD onset was included in the network as a clinically relevant node, as evidence suggests it may be differentially linked with symptom severity, symptom type, and comorbid conditions (Diniz et al., 2004; Millet et al., 2004).

An estimated regularized network was constructed to determine the centrality of each OCD node, respectively. Edges reflect the strength of the association of each pair of nodes, while controlling for all of the other nodes in the network (Borsboom & Cramer, 2013). These simultaneous partial correlations improve the interpretability of findings, such that displayed edges indicate potential causal relations and are less likely to be spurious (Robinaugh et al., 2014). Within this network, the primary outcome was *node strength centrality* (the sum of the weights of each edge connected to the node), which quantifies the impact a specific node has on the others in the network (Fried, 2015; Opsahl et al., 2010). We also computed two other measures of node centrality, including *betweenness centrality* (reflecting how often a particular node lies on the shortest path between other pairs of nodes) as well as *closeness centrality* (indicating the average distance between a specific node and all the other nodes in the network) (Epskamp, Borsboom, & Fried, 2017).

Hypothesis 1.1. We predicted that severity of obsessions (Y-BOCS Obsessions subscale) will be the most central node in the network. Given the cognitive-behavioral model of OCD, one can reason that severity of obsessions may act as the primary driving mechanism from which other OCD and relevant features stem. Per the cognitive theory of obsessions (Rachman, 1997, 1998),

once an obsession arises – reinforced by misinterpretations about its significance – it consequently leads to greater distress, fear, and neutralizing behaviors. It may be the case that greater obsessions severity is a key feature with the strongest interconnections in the overall network (i.e., more so than Y-BOCS Compulsions, or the OCD dimensions as represented by the four factor scores). As greater obsessions provoke not only greater emotional distress but also interference across social, occupational, and daily functioning domains, it may more easily relate to comorbid disorders as well.

Hypothesis 1.2a. We predicted that with regards to the four OCD factors, repugnant obsessions/checking compulsions would bear the strongest negative edge with age of OCD onset. Though research is scarce in this area, this finding would be in line with previous studies indicating that those suffering from early age of OCD onset more commonly endorse obsessions/checking and repeating/counting symptoms (Hasler et al., 2005; Millet et al., 2004; Minichiello, Baer, Jenike, & Holland, 1990).

Hypothesis 1.2b. Out of the four OCD factors, repugnant obsessions and checking compulsions would bear the strongest edge to comorbid anxiety disorders, as well as to comorbid mood disorders. This would reflect previous research indicating that aggressive/sexual/religious/somatic obsessions and checking compulsions are strongly linked with anxiety and depression (Hasler et al., 2005).

Aim 2: Does a History of Trauma Influence the Network of OCD?

Our second aim was to explore whether network connectivity of OCD may differ between individuals who have experienced a traumatic life event versus those who do not have a trauma history. Findings will help shed light on whether traumatic events may yield more densely and differentially connected OCD networks, thus providing a foundation for further exploration of whether traumatic events may have important downstream consequences for increased risk for greater OCD. For those who have suffered a traumatic event, it may be the case that the onset of each OCD symptom may more easily lead to a chain of associated symptoms, which may contribute to worse treatment outcomes in those with a traumatic history (Gershuny et al., 2002; Gershuny et al., 2008). Findings from this large clinical sample are not only generalizable, but may point to the importance of early identification and intervention of OCD in those with a history of trauma.

The OCD network for participants who endorsed experiencing at least one traumatic event was compared to the network of those who denied a history of trauma. The same OCD nodes described above in Aim 1 will be replicated in this network comparison. The *global strength* of each network – a measure of how densely connected the nodes in each network are – was compared between groups (see van Borkulo et al., 2015).

Hypothesis 2.1. We predicted that the global network strength between nodes in the OCD network will be greater in the Trauma History group, compared to the No Trauma History group. Theories and case studies purport that

experience of trauma can increase the tendency to experience greater obsessions and compulsions, as well as obsessive beliefs (de Silva & Marks, 1999, 2001; Pitman, 1993; Rachman, 1997). It may be the case that in individuals with a trauma history, once one OCD node is activated, they may be at greater risk for a more densely connected network of other OCD symptoms and clinically relevant features.

Methods

Participants

The sample consisted of 265 individuals who were consecutively admitted to the Adult OCD Research Program at the National Institute of Mental Health, as part of a larger investigation on the genetics of OCD. These participants were recruited via local advertisements, websites, and referrals from physicians and psychologists, as part of a larger investigation. Inclusion criteria included being age 18 or older, and having a primary diagnosis of OCD per the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 2001). Participants were excluded based on endorsing current schizophrenia or psychosis, severe mental incapacitation, or secondary OCD resulting exclusively from depression.

The sample included 166 (62.9% of the sample) females, and was 95% Caucasian, with 1.6% Hispanic, 1.1% Asian, 1.1% African-American, and 1.1% other. The mean age was 41 (*SD* = 15.02), with average age of OCD onset being 14.18 years old (*SD* = 8.67; range = 3-55 years). The Yale-Brown Obsessive Compulsive Scale Interview (Y-BOCS; Goodman et al., 1989) was used to

assess symptom severity; the average Y-BOCS score was 21.63 (*SD* = 9.34). The mean Y-BOCS obsessions score was 10.8 (*SD* = 5.3), while the mean Y-BOCS compulsions score was 10.5 (*SD* = 4.8). 52% of the sample was currently or previously married, and 67% endorsed having a college degree or higher. Similar to epidemiological studies, 90% of participants met criteria for one or more lifetime Axis I psychological disorders besides OCD.

Measures

Structured Clinical Interview for DSM-IV (SCID; First et al., 2001). The SCID is a semi-structured interview assessing current and lifetime Axis I disorders. Participants were administered the SCID by trained clinical interviewers, and data were blind-diagnosed by two independent clinicians in order to ensure reliability. The diagnostic ability of this group in previous studies has been found to demonstrate excellent reliability (LaSalle et al., 2004). The SCID was used to derive the comorbidity variables, reflected by a count of the number of disorders each participant met clinical criteria for. Mood disorders included depression, dysphoria, bipolar I, and bipolar II; anxiety disorders included panic disorder, agoraphobia, social anxiety disorder, specific phobia, generalized anxiety disorder, and post-traumatic stress disorder; substance use disorders included abuse or dependence of substances and alcohol; and OC-spectrum disorders included trichotillomania, skin-picking, tic disorder, Tourette's disorder, body dysmorphic disorder, binge eating disorder, anorexia nervosa, bulimia nervosa, eating disorder not otherwise specified, somatoform disorder, and hypochondriasis.

Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989). The Y-BOCS is a gold-standard measure of OCD severity, and has demonstrated good reliability and validity (Goodman et al., 1989). The Y-BOCS Symptom Checklist was used to assess the presence or absence of 72 different types of obsessions and compulsions. Factor scores for the different symptom dimensions of OCD were then derived from the checklist's 13 Y-BOCS categories, using principal component analysis [see Hasler et al. (2005) for details of factor analysis]. Four factor scores were generated for each participant and were standardized such that the mean was 0 and standard deviation was 1. Each score reflects the strength of the subject's symptom profile with each of the four factors: obsessions/checking, symmetry/ordering, contamination/cleaning, and hoarding, and have been utilized in previous OCD research as well (e.g., Cromer et al., 2007; Mataix-Cols et al., 2005). As aforementioned, factor scores reflect how many symptoms within each dimension are endorsed.

Following the completion of the symptom checklist, participants responded to a series of severity questions. Across all endorsed obsessions and compulsions, respectively, participants provided scores on the following five items: *duration* of symptoms, *distress* experienced as a result of the symptoms, *interference* caused by symptoms, level of *resistance* in response to symptoms, and level of *control* over symptoms. Each item was rated on a scale from 0 (least severe) to 4 (most severe). Responses to these five items are summed to yield the obsessions and compulsions severity subscales, as well as a total score.

These two subscale scores of obsessions and compulsions severity are collapsed across symptom dimensions.

Trauma History measure (Cromer et al., 2007). TLEs were assessed in a diagnostic interview – specifically, using the information provided by participants in the "Traumatic Events List" in the PTSD module of the SCID. Participants are asked an open-ended question with regards to having experienced any extremely upsetting, stressful, or traumatic life experiences, with several examples provided. The clinician documented all reported events prior to conducting the PTSD module, such that all significant TLEs were accounted for regardless of whether the individual met full criteria for PTSD. In this study, a trauma history was considered having a lifetime presence of at least one TLE (endorsed by 54% of the sample).

Data Analytic Approach

Estimated Regularized Network. The R package *qgraph* (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012) was used to compute the networks for Study 1. The nodes in the network represented the following, as numbered: (1) OCD severity for obsessions (ObsSever) and (2) compulsions (CompSever); OCD factor type: (3) aggressive, sexual, religious obsessions, and checking compulsions (Obs/Check), (4) symmetry obsessions, counting/ordering compulsions (Symm/Ord), (5) contamination obsessions and cleaning compulsions (Cont/Clean), (5) hoarding obsessions and compulsions (Hoard); as well as several clinically relevant comorbid factors: (7) total number of anxiety disorders (AnxDx), (8) total number of mood disorders (MoodDx), (9) total

number of substance/alcohol disorders (SubsDx), (10) total number of OC-spectrum disorders (11), and (12) age of OCD onset (AgeOnset).

Each edge depicted in the network represents the strength of association between each pair of symptoms, after statistically controlling for all the other (ten) factors in the network; essentially, an adjusted partial correlation estimated with the “least absolute shrinkage and selection operator” (LASSO) technique. LASSO is used to help circumvent the large number of parameters that would have to be estimated in a comprehensive model; by minimizing the edge estimates that are negligible, it returns a sparse (i.e., conservative) network. This estimation technique increases interpretability of the model, such that the smallest number of edges that explain the data’s covariance are ultimately included (Epskamp et al., 2017). A tuning parameter is selected to adjust the sensitivity of the estimation; this parameter is calculated using the Extended Bayesian Information Criterion (EBIC), incorporated into the *qgraph* package (Epskamp et al., 2017). Node centrality is quantified (again using *qgraph*) by examining the strength of a node, which is calculated by summing the weights (in other words, the correlation magnitudes) of each edge connected to the node. A higher node strength value indicates greater centrality, such that a node is more directly connected to other nodes in the network.

Bootstrapping Analyses. Relatively few studies have progressed beyond estimating networks to examine their stability (Santos, Fried, Asafu-Adjei, & Ruiz, 2017). In line with procedures established by Epskamp et al. (2017) and utilized in the most up-to-date network studies (McNally, Mair, et al., 2017; Santos et al.,

2017), we conducted bootstrapping analyses with the R package *bootnet*. Results from 1000 bootstrapped networks provide an estimation of how stable centrality indicators are (i.e., by providing a coefficient between 0 and 1, with larger values reflecting higher stability). Moreover, bootstrapping results also provide confidence intervals (CIs) for the strength of each retained edge in the network, indicating accuracy and significance (i.e., whether the CI encompasses zero).

Network Comparison Test. Overall network connectivity between Trauma Hx and No Trauma Hx groups was compared utilizing the Network Comparison Test in the R package *NCT* (see van Borkulo et al., 2015) to determine the extent of the different network structures and strengths between the two subsamples. The NCT is a two-tailed permutation test that repeatedly calculates the difference between two groups for individuals who are randomly regrouped. The findings are used to create a distribution under the null hypothesis, which assumes the two groups are equal; the observed difference between the actual observations is then compared at a significance level of .05.

Missing Data & Power Analysis

265 individuals were included in the full dataset; however, some were missing data for one or more of the above measures. For Aim 1, 241 participants had full data for the variables in question. In line with previous studies, we estimated a network model with the full dataset (N = 265), using pairwise analyses for all available information. All nodes were analyzed as continuous (rather than ordinal) variables. For Aim 2, the network comparison test cannot

handle missing data (Santos et al., 2017; van Borkulo et al., 2015). Thus, we only included the subsamples without missing data, which resulted in *n* = 113 for the Trauma Hx group (reduced from 142), and *n* = 103 for the No Trauma Hx group (reduced from 123).

With regards to network analyses, studies are considered adequately powered if there are at least 5 participants per node in the network (Wigman, de Vos, Wichers, van Os, & Bartels-Velthuis, 2016). For our primary aim – to examine the basic network of OCD and comorbid features – there are 11 nodes to be considered in the network; thus, a sample size of N ≥ 55 would be sufficient. Our actual sample size, N = 265, is thus appropriate for conducting our primary analyses. To our knowledge, there are no established guidelines for ensuring adequate power of a network comparison test analysis; we thus proceeded with our sample size adjusted for missing data (*n* = 113 and *n* = 103, for Trauma Hx and No Trauma Hx groups respectively).

Results

Descriptive Data

Table 2.1 shows the mean, standard deviation, and ranges for the variables of interest, while Table 2.2 shows their zero-order correlations. As reported in a previously published report, 54% of the sample endorsed experiencing at least one TLE (Cromer et al., 2007). A total of 238 events were described, the most frequent being: accident involvement (38 events), witnessed nonviolent death (24 events), witnessed crime (22 events), sexual abuse (22 events), and illness of self or loved one (19 events).

Aim 1: A First Look at the Network Structure of OCD

Figure 2.1 displays the estimated network with all 11 nodes included. As number of comorbid OC-spectrum disorders (OCSDx) was not connected with any other nodes, we re-conducted analyses to estimate a network excluding the OCSDx node, as displayed in Figure 2.2. Meanwhile, Figure 2.3 shows the corresponding centrality plot for this adjusted network, including node betweenness, closeness, and strength. Out of the three, node strength yielded the most stable estimation of centrality and was identified as being sufficiently stable (see Figure 2.4; stability coefficient = .44; suggested guidelines are >.25 and preferably >.50), per Epskamp et al. (2017). In other words, results showed that node strength centrality would remain stable even after dropping over 50% of the sample. Furthermore, node strength was highly correlated with both closeness (r = .92) and betweenness (r = .81). This supports the use of node strength as the primary indicator of centrality for this network; as described by McNally (2016), strength is an important indicator for interpreting psychopathology networks, as it highlights specific symptoms that may be critical in activating other symptoms.

The three most central symptoms in our network were: repugnant obsessions and checking compulsions (Obs/Check; strength coefficient of 1.26), severity of obsessions (ObsSever; .85), and symmetry obsessions and ordering compulsions (Symm/Order; .81). In contrast to our original Hypothesis 1.1, repugnant obsessions and checking compulsions (Obs/Check) was the node that yielded the greatest centrality as reflected by all three main indices: node

strength (1.18), closeness (.015), and betweenness (27) (see Figure 2.3). Difference tests showed that repugnant obsessions and checking compulsions was significantly more central than severity of obsessions and symmetry and ordering, but that the latter two did not significantly differ from one another in terms of strength centrality. Stability analyses supported this finding, as repugnant obsessions and checking compulsions yielded the greatest node strength in the bootstrapped network as well. As Figure 2.5 shows, the strength of repugnant obsessions and checking compulsions was significantly greater than any other node. In this bootstrapped network, severity of obsessions yielded the second most central node with regards to strength, yet did not significantly differ from several other nodes (i.e., anxiety disorders, severity of compulsions, contamination and cleaning, and symmetry and ordering).

In the estimated network of our sample data, the strongest edge was between obsessions severity (ObsSever) and compulsions severity (CompSever) with an edge weight of .47. Bootstrapped stability analyses demonstrated that this edge not only significantly differed from zero, indicating a reliable interconnection (Figure 2.6 and Table 2.3), but was also significantly greater than all other edges in the network (Figure 2.7). The next two strongest edges in the bootstrapped network were between repugnant obsessions and checking compulsions (Obs/Check) and obsessions severity (ObsSever; .25) and between comorbid mood disorders (MoodDx) and comorbid anxiety disorders (AnxDx; .24), which were both significant, but did not significantly differ between one another (Figure 2.6 and Table 2.3).

Amongst the four OCD factors, repugnant obsessions and checking compulsions (Obs/Check) demonstrated the strongest estimated edge weight to age of OCD Onset (OCDOnset; -.14); in comparison to symmetry/ordering (Symm/Ord; -.04), contamination/cleaning (Cont/Clean; -.07), and hoarding (Hoard; .00). This is in line with our hypothesis 1.2a, building on previous research, suggesting that greater obsessions/checking may be more strongly connected with an early age of OCD onset. The bootstrapping analyses confirmed that the only significant edge linking OCDOnset into the network was that to Obs/Check, while the relations to the other factors may not be reliable (Figure 2.6 and Table 2.3).

The edges linking each of the four factor scores to each of the four measures of comorbidity (anxiety, mood, substance, and OC-spectrum disorders) were also compared, to determine their relative weights. In nearly full support of our hypothesis 1.2b, repugnant obsessions and checking compulsions (Obs/Check) demonstrated the strongest edges to comorbid anxiety disorders (AnxDx; .20) as well as to mood disorders (MoodDx; .098). In addition, Obs/Check was the only OCD factor to bear any positive or negative estimated edge in the estimated model to substance disorders (SubsDx; .10). Bootstrapped analyses showed the edges to remain significant between repugnant obsessions and checking compulsions and anxiety disorders (Figure 2.6 and Table 3). However, the confidence interval of the estimated edge weights between repugnant obsessions and checking compulsions and mood versus substance disorders, respectively, did span zero on the lower end – 95% CI [.00, .31] and

[.00, .18], respectively – suggesting a potentially less reliable connection between repugnant obsessions and checking compulsions and other forms of psychiatric comorbidity (Figure 2.6 and Table 2.3).

Aim 2: Does a History of Trauma Influence the Network of OCD?

Hypothesis 2.1. The sample was divided into two groups, based on the TLE measure: having a lifetime presence of one or more TLEs, versus none. The adjusted OCD network from Aim 1 was constructed in *qgraph* for each group. Figure 2.8 displays the estimated network constructed for both those with a trauma history (Trauma Hx, n = 143) and with no trauma history (No Trauma Hx, n = 122), using pairwise deletion with all available data. However, as the NCT cannot handle missing data, we retained only participants with complete data for all 10 variables, which reduced sample sizes (and thus power) to n = 113 and n = 103 for Trauma Hx and No Trauma Hx groups, respectively. The adjusted networks with listwise deletion are shown in Figure 2.9, while Figure 2.10 shows their corresponding centrality plots.

Considering the adjusted networks, in the Trauma Hx group, node strength was again highly correlated with both closeness (r = .96) and betweenness (r = .84); stability correlations were invalid for the No Trauma Hx group given the lack of connectedness in the network[3]. Although a rather large portion of each sample was reduced when excluding missing data, we did

[3] Previous research with larger samples than ours have declined to perform bootstrapping for subgroups divided for NCT comparison analyses, citing a lack of sufficient power to draw conclusions (Santos et al., 2017). As an exploratory step, we did test the stability of each subgroup's estimated networks by performing bootstrapping analyses (as conducted with the overall network in Aim 1); unsurprisingly, findings did suggest instability in both models, most certainly due to a lack of power. Thus, interpretations of these secondary analyses should be perceived as merely suggestive of a foundation for further exploration of these symptom relations.

proceed to conduct the NCT as an exploration of group differences. Unsurprisingly, the NCT did not find significant differences between the two groups with regards to network structure (test statistic M = .26, P = .49) or global strength (test statistic S = 1.93, P = .11). The edge invariance test revealed that the edge between obsessions severity and compulsions severity – the strongest edge retained in the adjusted network for each group – did not significantly differ between the Trauma vs. No Trauma Hx groups (E = .057, P = .69), indicating that the link between these two nodes may be similar in both groups and could potentially account for the non-significant difference in global network strength.

Despite the lack of significant group differences per the NCT, the estimated networks were striking in their qualitative differences (see Figure 2.8). The network for the Trauma Hx group appeared very visually similar to that in Aim 1 with the full sample. Obs/Check remained as seemingly the most central node in this network, bearing direct edge weights with all nodes except CompSever, just as in the initial network with all participants included. Yet, in stark contrast, only six edges were retained in the No Trauma Hx network. Obs/Check remained the most central node in the group without a trauma history, remaining connected with AnxDx, Symm/Ord, and ObsSever. However, the edges between Obs/Check and OCDOnset, Cont/Clean, SubsDx, MoodDx, and Hoard, respectively, were missing in the No Trauma Hx network. The network structure of the No Trauma Hx group was very sparse compared to that of the Trauma Hx group as well as the combined sample.

Discussion

Altogether, Study 1 was a first step towards examining the putative connection between OCD severity, dimensions, and comorbid disorders. It contributes to the burgeoning recent literature of using the network approach to study psychopathology, as an additional “proof of concept” of the perspective networks can provide to complement the traditional categorical lens from which disorders are viewed (McNally, 2016). Importantly, this study highlighted the importance of aggressive, sexual, religious obsessions and checking compulsions (Obs/Check) as a critical component through which other OCD features are connected, as it proved to be the most central node across all three primary indices of strength, closeness, and betweenness. Though there was certainly a strong estimated edge weight linking this Obs/Check and severity of obsessions (ObsSever), our results indicate that the former node may more readily perpetuate or coincide with other OCD aspects, more so than factor scores of other symptom dimensions. Experiencing a greater variety of repugnant, ego-dystonic obsessions may promote or exacerbate obsessive symptoms of other dimensions, and may also be an important bridge to consider in the comorbidity that so often accompanies OCD. Individuals suffering from OCD may endorse symptoms across several dimensions, but it may be the case that those with more numerous repugnant intrusive thoughts tend to develop other psychiatric symptoms.

Out of the OCD factors, Obs/Check also evidenced the strongest estimated edge weight with age of OCD onset (OCDOnset). Bootstrapping

verified this to be the only significant edge tying in OCD Onset to the rest of the network. This is in support of tentative previous evidence that distasteful obsessions and checking is linked with an early (versus late) onset of OCD. It may be the case that these repugnant obsessions and checking rituals may be amongst the first to emerge in those who develop OCD, which eventually evolve over time or facilitate the development of other types of OCD symptoms. Further research could investigate whether greater repugnant obsessions/checking is consistently present in those with early-onset OCD, and/or whether more frequent/severe obsessions and checking symptoms may worsen over a longer duration of OCD. Of note, Millet et al. (2004) found that superstitious and magical thoughts were more common in individuals with early onset OCD (under age 15); future research could explore whether this type of magical thinking, if taken to an extreme level in childhood, may give rise to more unwanted obsessive intrusions and checking compulsions and consequently a potentially more complex course of OCD.

With regards to comorbid disorders, the strongest estimated edge weight was between number of anxiety disorders (AnxDx) and Obs/Check. This raises the possibility that having repugnant, unwanted intrusions and frequent checking urges may be the core factor that causes the most extreme distress and worry. Further research could help confirm directionality hypotheses of whether repugnant obsessions and mental compulsions may be a key stepping stone towards developing other anxiety disorders. Another (not mutually exclusive) alternative explanation to be explored is that those with other anxiety disorders,

such as generalized or social anxiety, may more readily develop the intrusive aggressive/sexual/religious obsessions that characterize Obs/Check, which could then act as a gateway to other OCD dimensions.

Meanwhile, number of mood disorders (MoodDx) was linked with both Obs/Check and Symm/Order, but bore the strongest edge weight with AnxDx. This may suggest that comorbid mood disorders, most notably major depression, may be more likely to go hand-in-hand with anxiety disorders in general, more so than any specific component or severity indicator of OCD in particular. A more pervasive trait, such as repetitive negative thinking, anxiety sensitivity, and/or distress tolerance, may play a larger role in the link between depression and OCD, which would be promising for future studies to investigate. Interestingly, number of OC-spectrum disorders (OCSDx) was not connected at all within our network, which is surprising given their classification, particularly within our sample of lifetime OCD. Given that our estimated network displays the connections that exist above and beyond all other correlations between node pairs, it may be the case that OC-spectrum disorders co-occur at relatively equal rates in those with lifetime OCD. Comorbidity with OC-spectrum disorders may be driven by mechanisms independent of our examined network, or may not be strongly linked with OCD symptom type or with other mood/anxiety syndromes.

Differential connections for the four OCD factors were striking in our network. As aforementioned, Obs/Check was not only the most central node that other symptoms were connected through, but was also the only factor linked to anxiety disorders and substance use disorders (and mood disorders as well,

though not established as significant in the bootstrapped network). Another finding of interest was that contamination/cleaning symptoms (Cont/Clean) was the only OCD factor with a significant edge with severity of compulsions (CompSever). This may be explained in part by the overt, externalizing nature of cleaning rituals, which may more readily cause impairment and distress on the compulsions subscale. In contrast, mental neutralizing rituals or reassurance-seeking associated with checking is more internal (hence the misnomer of a "pure obsessions" OCD subtype; see Williams et al., 2011). Similarly, ordering urges as a reaction to "not just right experiences" associated with symmetry symptoms may not manifest in as severe behavioral compulsions compared to cleaning rituals (Timpano, Carbonella, Zuckerman, & Çek, 2016). Finally, the OCD factor representing hoarding was linked only to Obs/Check and Symm/Ord in our estimated network – both connections which were not found to be significant in the bootstrapped network. This may indicate that hoarding symptoms may tend to arise as an independent dimension devoid from the others, with more distinct risk factors and etiology. It is also important to note that Hoarding Disorder has been classified as a separate syndrome in the DSM-5 (American Psychiatric Association, 2013), distinct from OCD-motivated hoarding symptoms. This distinction had not yet been made at the time this study's data was collected, which may have muddled the relationship between genuine OCD-type hoarding and the other symptom factors and severity.

With regards to Aim 2, the qualitative differences between the networks for individuals with versus without a trauma history were apparent. The network for

the Trauma Hx group appeared very similar to the one presented in Aim 1 with the full sample size. However, the No Trauma Hx network was strikingly sparse, in comparison. Though Obs/Check remained as a central node, it lacked estimated edges to most of the connected nodes present in the Trauma Hx network. Likely due to a lack of power from having to delete many cases of missing data, the NCT did not statistically confirm the visually drastic disparity between groups, indicating that the two samples' network structures and global strength did not significantly differ. Interpretations of group differences should be approached with caution, but provide interesting venues for further research to investigate. One possible explanation for foreseeable group differences (given greater power) is that those with a background of trauma may be more vulnerable to developing characteristics of OCD – such as the central repugnant obsessive tendencies of Obs/Check – which would easily ferment the rise of associated symptoms into full-blown OCD. In other words, in individuals with a trauma history, one activated node may more readily devolve into a slippery slope of OCD symptoms and associated anxiety and mood difficulties. This interpretation would be in support of traumatic life events as an important vulnerability factor for OCD (e.g., Cromer et al., 2007; Real et al., 2011). In particular, despite evidencing a strong edge to Obs/Check in both the overall and Trauma Hx networks, age of OCD Onset was not connected in the network for those with No Trauma Hx. Further studies may help clarify the impact of traumatic events experienced early in life (e.g., childhood abuse) on the presentation of early vs. late onset OCD.

Additionally, the strong link between obsessions and compulsions severity in the No Trauma Hx group may be interpreted in support of the cognitive-behavioral model of OCD. This is in line with compulsions serving to alleviate distress associated with obsessions, yet ultimately reinforcing them, such that the severity of obsessions and compulsions are tightly intertwined (Swinson et al., 2001; Taylor et al., 2007). This network for the No Trauma Hx group is in line with the DSM-5 characterization of OCD, such that obsessions drive compulsions, and vice versa. This raises the possibility that those who have not experienced trauma may be less likely to develop the interconnected web of other OCD features and comorbid disorders. As such, controlled treatment studies may clarify whether those without a trauma background may have a less complex presentation and greater therapeutic gains compared to those with a trauma history.

Of note, our network represented the connection between nodes across a group of individuals, and could take on idiosyncratic patterns when examining a specific patient. Given known differences in OCD between males and females, it may also be the case that their respective networks yield different structures and global strength. In contrast to other anxiety disorders, OCD has been found to present at roughly equal rates in males and females (Rasmussen & Eisen, 1992). However, being male has been found to predict not only a more chronic course of OCD but also an earlier age of onset (Bogetto et al., 1999), which could potentially make those corresponding nodes more central in a sample of men. Moreover, a history of substance disorders and social phobia is more prevalent

among male OCD patients (Bogetto et al., 1999; Noshirvani, Kasvikis, Marks, Tsakiris, & Monteiro, 1991), while females are more likely to present with depression and eating disorders (Bogetto et al., 1999; Castle, Deale, & Marks, 1995; Lensi et al., 1996; Noshirvani et al., 1991) – which could impact the centrality of these comorbid disorders in our network when comparing genders. Of particular relevance to our network is the finding that females are more likely to endorse a stressful event shortly before OCD onset (Bogetto et al., 1999); perhaps, in a larger study comparing exclusively women who have versus have not experienced trauma, the NCT would demonstrate more conclusive differences than our Aim 2 which pooled genders together.

Findings should be considered in light of several weaknesses of the study, including the retrospective, cross-sectional data collection. The sample size prevented us from exploring additional sub-group analyses in Aim 2 that would be underpowered, such as whether trauma occurred prior to or after OCD onset, whether trauma was experienced in adulthood or childhood, and the nature of trauma (e.g., of a sexual nature or not). It should also be noted that the indicators of comorbidity were a frequency count of other disorders meeting the clinical threshold rather than severity; similarly, OCD factors were derived from symptom type categorization from the Y-BOCS Checklist rather than severity of symptoms. This may have obscured edges that may have emerged with more fine-grained measures of comorbidity and OCD dimensions. For instance, we may have gleaned connections to OC-spectrum disorders if more detailed measures of trichotillomania and skin-picking were included to provide greater variability of

symptoms. Trauma history was also measured in a subjective way based on responses on the SCID, which precluded a closer look at severity of traumatic events.

Further studies in this realm may also explore alternative network approaches, such as using Bayesian algorithms that may shed more light on directionality and causality between pairs of nodes (e.g., McNally, 2016; McNally, Mair, et al., 2017). A major strength of the study – examining a large sample of individuals with lifetime clinical OCD – also introduces uncertainty of whether findings may also extend to those with subclinical OCD symptoms. It is also important to take a cautious approach in interpreting our findings on a more individual level; our networks represent group data as a composite, while individual networks may differ both from one another and from the group network. Finally, as is true for all network analyses, the selection of nodes is somewhat subjective; our networks reflect a constellation of indicators and nodes that are of interest to the overarching research question, but alternative conceptualizations may be equally valid.

Ultimately, this study was novel in its application of network analysis to examine the interrelations between OCD severity, symptom type, onset, and relevant comorbid aspects. Altogether, findings highlighted the central role of aggressive/sexual/religious obsessions and checking compulsions as a crucial “hotspot” from which other symptoms may stem. The network approach allowed us to glean in a clinical sample that it may specifically be frequent repugnant obsessions that are important to target in early interventions, rather than overall

severity of obsessions. The NCT also provided initial qualitative evidence in support of the importance of a traumatic background in facilitating the interconnection between OCD features, which further studies with larger samples would be wise to explore.

Chapter 3: Study 2 – Network of OCD and PTSD Symptoms

Background

Obsessive Compulsive Disorder (OCD) is an extremely impairing condition that affects approximately 2% of the population and has been identified as one of the top ten leading forms of disability worldwide (Benito & Storch, 2011). OCD is characterized by recurrent intrusive thoughts and corresponding compulsions, both of which can cause distress and impairment across a range of social, financial, and work-related domains (Adam et al., 2012; Lopez & Murray, 1998; Mataix-Cols et al., 2005). The cognitive-behavioral model of OCD highlights the functional relationship between obsessive beliefs, obsessions, and compulsions (Rachman, 1997; Salkovskis, 1985). It purports that high levels of obsessive beliefs (such as over-estimation of threat, over-importance of thoughts, and inflated responsibility) lead one to misinterpret the value of intrusive thoughts and thus can cause a great deal of anxiety (Rachman, 1997). This distress compels the individual to perform mental or behavioral rituals in an effort to alleviate the anxiety associated with obsessions (Swinson et al., 2001; Taylor et al., 2007). Yet, by temporarily lowering anxiety, the compulsion is attributed as preventing the harmful intrusive thought, thus perpetuating a vicious cycle (Swinson et al., 2001; Taylor et al., 2007).

OCD is also notorious for its extremely high comorbidity with other psychiatric disorders, with 90% of lifetime OCD patients meeting criteria for another lifetime disorder (LaSalle et al., 2004; Ruscio et al., 2010). Approximately 76% of these individuals experience an anxiety disorder, followed closely by 63%

suffering from a mood disorder (Ruscio et al., 2010). Comorbidity in association with OCD is associated with a range of negative factors, including more severe daily impairment, greater need for treatment, and even higher suicidality (e.g., Brown & Barlow, 1992; Bruce et al., 2005; Nock et al., 2010). Of note, post-traumatic stress disorder (PTSD) and OCD are highly comorbid, at rates much higher than expected in the general population – about 20% of OCD patients meet criteria for lifetime PTSD (Huppert et al., 2005; Ruscio et al., 2010), over twice the prevalence rate in the general population (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Comorbid PTSD is linked with a more severe clinical OCD presentation, and can result in worse treatment outcomes (Gershuny et al., 2002; Gershuny et al., 2008).

Despite the high levels of comorbidity, much remains unclear about how the specific symptoms of OCD and PTSD may be differentially connected with one another, let alone within patients with clinical levels of OCD. As both syndromes are particularly heterogeneous in symptom presentation, further research is warranted to evaluate how specific aspects of PTSD may relate to OCD's hallmark components of obsessions and compulsions. This endeavor is especially interesting in light of common themes between the two disorders (e.g., repetitive intrusive thoughts).

The network approach is a perspective recently applied to psychopathology that may help highlight both theoretical and clinically relevant links between symptoms, as a complement to the traditional latent model approach (Borsboom & Cramer, 2013). Rather than conceptualizing disorders as

underlying entities that give rise to symptoms, network analyses directly examine symptom interactions from the perspective that constellations of symptoms themselves are actually what constitute psychopathological syndromes (McNally, 2016; McNally et al., 2015). The network approach is helpful in exploring symptom overlap between OCD and PTSD by examining collective symptoms simultaneously, highlighting individual ones that may be driving this interesting comorbidity – not only with one another but also with common clinical correlates like depression and anxiety. Burgeoning evidence from network analyses of other comorbid disorders have been illuminating: for instance, loneliness and emotional numbness were identified as critical features in the overlap between complicated grief and depression (Robinaugh et al., 2014). Similarly, distress associated with obsessions was found to be important in activating depression in those with OCD (McNally, Mair, et al., 2017). Likewise, we hope to uncover specific OCD and PTSD symptoms that may cluster together, as well as central features that may play a substantial role in connecting more peripheral symptoms, which can uncover a theoretical foundation for further studies of directionality.

On their own, PTSD symptoms have generally demonstrated differential links to a range of psychological factors, providing reasonable belief that the same may hold in relation to OCD symptoms. Confirmatory factor analyses – in line with the DSM-5's characterization of PTSD (American Psychiatric Association, 2013) – have found a four-factor model to be the best fit for PTSD: *re-experiencing* and intrusive recollections of the traumatic event, *avoidance* of

relevant stimuli, *emotional numbing*, and *hyperarousal* (Asmundson et al., 2000). In a sample of war veterans, the numbing PTSD symptom cluster was found to be the only one directly related to suicidal ideation, while re-experiencing was the only symptom cluster directly linked with aggressive behaviors (Hellmuth, Stappenbeck, Hoerster, & Jakupcak, 2012). Dimensions of anxiety sensitivity (AS) have been found to bear specific links with certain PTSD clusters as well (Asmundson & Stapleton, 2008; Collimore, McCabe, Carleton, & Asmundson, 2008), which may reciprocally predict later AS severity (Marshall, Miles, & Stewart, 2010). PTSD clusters have also been linked with different patterns of substance use, suggesting that women with greater symptoms of hyperarousal (as opposed to other PTSD dimensions) tend to use drugs to treat their symptoms, which in turn exacerbates arousal symptoms (Sullivan & Holt, 2008). Of note, one recent paper has illustrated the utility of network analyses in examining the putative structure of PTSD symptoms in a sample of survivors of a natural disaster. McNally et al. (2015) found hypervigilance and future foreshortening to be central symptoms tying the network together, and also highlighted the importance of connections between anger/irritability, sleep, and concentration that warrant further empirical and clinical attention.

With regards to comorbidity with OCD, it is still unclear if and how these aforementioned PTSD symptoms may differentially cohere or cluster with specific features of obsessions and compulsions. Network analyses may help reveal functional relationships between symptoms that may also underlie the heterogeneous presentation of OCD, especially in a clinical population. In

particular, re-experiencing symptoms of PTSD warrant further exploration in relation to OCD. The nature of repeated intrusive thoughts is similar to the unwanted obsessions about harm to oneself or others, and could be central in helping explain the comorbidity between the two communities of symptoms. One possibility is that a general tendency to make catastrophic misinterpretations about intrusive thoughts relating to trauma may generalize to full-blown obsessions (de Silva & Marks, 1999, 2001). This degree of symptom overlap between obsessing and PTSD intrusions was highlighted in a study employing expert psychologists, who rated three items from the Obsessive Compulsive Inventory-Revised (a well-established measure of OCS) as definitively overlapping with PTSD (Huppert et al., 2005). All three items were from the obsessing subscale: “unpleasant thoughts come into my mind against my will and I cannot get rid of them,” “I find it difficult to control my own thoughts,” and “I am upset by unpleasant thoughts that come into my mind against my will” (Huppert et al., 2005). Empirical evidence of a strong connection between re-experiencing and obsessions about harm in a large clinical sample, above and beyond that of other symptoms, may highlight these factors as critical in helping maintain the network of OCD and PTSD symptoms.

OCD symptoms are often challenging to operationalize in empirical studies, which holds true for network analyses as well. Per the cognitive-behavioral model of OCD, compulsions mainly function to neutralize or decrease anxiety or discomfort associated with obsessions (Swinson et al., 2001; Taylor et al., 2007). However, it may be more helpful to examine item-level components of

particular components of obsessions versus compulsions, as we will do by incorporating item scores on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), a gold-standard measure of OCD severity. The Y-BOCS consists of scores of time occupied, interference, level of distress, degree of resistance, and degree of control, for both obsessions and compulsions. This more fine-grained approach may help elucidate whether certain OCD components may be more important to consider in relation to particular PTSD symptoms, which largely remains unclear. Furthermore, tentative evidence of PTSD symptoms and OCD symptom *dimensions* (rather than *severity*) have been mixed, with some studies finding traumatic life events to predict obsessions/checking and symmetry/ordering (Cromer et al., 2007; Grisham et al., 2011), and others supporting greater contamination/washing symptoms in association with co-morbid PTSD (L. F. Fontenelle et al., 2011; L. F. Fontenelle et al., 2012). This raises the possibility that specific components of OCD severity (e.g., time occupied, interference, distress, resistance, or control, as mentioned above) may be more functional in explaining comorbidity between OCD and PTSD, as symptoms that can be collapsed across symptom dimensions or types. For instance, case studies have suggested that following a trauma, victims tend to experience disgust regarding physical reminders of the incident (e.g., feeling tainted when recalling one's own wounds, or a desire to wash oneself upon reminders of a sexual assault) (de Silva & Marks, 1999, 2001; Sasson et al., 2005). It may be the case that greater re-experiencing symptoms of trauma – rather than directly predicting contamination/washing – may actually underlie

greater *distress* associated with obsessions in general. In the context of the cognitive-behavioral OCD model, this connection may consequently increase contamination/washing compulsions in order to help negate feelings of disgust. As such, it may be important to consider these trans-dimensional OCD symptom *severity* indicators, in order to better understand how PTSD symptoms may functionally relate to OCD. As the first study to our knowledge to examine a network of hallmark features of OCD severity and PTSD symptoms in an OCD population, we will thus exclusively focus on severity as a potentially important clue to understanding these underlying mechanisms. Future studies may elect to investigate symptom dimensions in conjunction with (or separately from) severity.

In summary, Study 2 will examine OCD and PTSD symptom-level relationships in a clinical OCD sample; results may help explain the nature of symptom overlap between these two heterogeneous disorders. This study will also take a closer look at specific features of obsessions and compulsions, and network edges may help point towards potential causal mechanisms of comorbidity warranting future exploration (McNally, Mair, et al., 2017; Ruscio et al., 2010). Network examinations of severity at the symptom-level will provide a more fine-grained insight into these two disorders in comparison to previous investigations, which have typically relied more broad indicators, such as the presence/absence of a traumatic event, diagnostic status of PTSD and/or OCD, or total sum-scores of symptom severity (Grabe et al., 2007; Huppert et al., 2005; Mathews et al., 2008; Nacasch et al., 2011). Networks will help present an initial lay of the land for how these syndromes functionally connect and map onto each

other, furthering our knowledge of factors that may bear the greatest risk in activating other symptoms in the network. Of notable strength is our large sample of individuals with clinical OCD and examination of specific components of obsessions versus compulsions, which yield greater symptom variability and clinical utility. Findings will benefit not only our conceptualization of OCD and PTSD as highly comorbid disorders, but also the refinement of early prevention and intervention efforts for susceptible individuals, providing insight for tailoring treatments according to individuals' symptom presentation. For instance, for those with recurrent thoughts and nightmares of an experienced trauma, cognitive interventions and mindfulness techniques could help break the link between re-experiencing and intrusive thoughts. In a similar vein, if distress associated with obsessions is highlighted as an important connection to PTSD symptoms, emotional tolerance training may be an important component to include in therapy.

Aim 1: A First Look at the Network Structure of OCD and PTSD Symptoms.

Our first aim will be to examine the putative causal structure of the hallmark features of obsessions and compulsions, in conjunction with PTSD symptoms. An estimated regularized network will be constructed to determine the centrality of each respective node. Edges reflect the strength of the association of each pair of nodes, while controlling for all of the other nodes in the network (Borsboom & Cramer, 2013). These simultaneous partial correlations improve the interpretability of findings, such that displayed edges indicate potential causal relations and are less likely to be spurious (Robinaugh et al., 2014). Within this

network, the primary outcome will be *node strength centrality* (the sum of the weights of each edge connected to the node), which quantifies the impact a specific node has on the others in the network (Fried, 2015; Opsahl et al., 2010).

OCD obsessions and compulsions severity will be operationalized using the ten item scores on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), including the following: (1) time occupied by obsessions (ObsTime); (2) interference caused by obsessions (ObsInt); (3) distress caused by obsessions (ObsDis); (4) difficulty resisting obsessions (ObsRes); (5) difficulty controlling obsessions (ObsCont); (6) time occupied by compulsions (CompTime); (7) interference caused by compulsions (CompInt); (8) distress caused by compulsions (CompDis); (9) difficulty resisting compulsions (CompRes); and (10) difficulty controlling compulsions (CompCont). These exact symptoms have been recently investigated in a network analysis of OCD and depression symptoms (McNally, Mair, et al., 2017).

In line with a separate study of PTSD symptom networks, we will also incorporate 17 symptoms of PTSD, based on the PTSD Checklist-Civilian Version (PCL-C) (McNally et al., 2015). Of note, when using the PCL-C version, individuals are asked about their recent responses to stressful life events (SLE), rather than a truly traumatic event warranting a clinical PTSD diagnosis. The re-experiencing symptom cluster includes: (a) intrusive memories, thoughts, or images of the SLE (Intrusion); (b) traumatic dreams (Dreams); (c) flashbacks (Flash); (d) feeling upset in response to reminders of SLE (Upset); and (e) physiological reactivity to reminders of the SLE (PhysioR). The avoidant cluster

includes: (f) avoidance of thoughts or feelings about the SLE (AvoidTh) and (g) avoidance of activities or situations reminiscent of the SLE (AvoidAct). The numbing cluster consists of: (h) having trouble remembering parts of the SLE (Amnesia); (i) loss of interest in previously enjoyed activities (LossInt); (j) feeling distant or cut off from people (Distant); (k) feeling emotionally numb (Numb); and (l) feeling that your future will be cut short (Future). Finally, the hyperarousal symptom cluster includes: (m) difficulty falling or staying asleep (Sleep); (n) feeling irritable or having angry outbursts (Anger); (o) difficulty concentrating (Concen); (p) hypervigilant, watchful or super alert (Hyper); and (q) feeling easily startled or jumpy (Startle).

Hypothesis 1.1. We hypothesize that out of the PTSD symptoms, intrusive memories/thoughts/images will yield the highest node strength centrality. As earlier described, having repeated intrusive thoughts is similar to the unwanted obsessions about harm to oneself or others, and could be centrally related to both communities of symptoms.

Hypothesis 1.2. We predict that distress caused by obsessions will be the most central OCD node. Similarly, greater distress associated with obsessions may help explain the comorbidity between OCD and PTSD, such that it may be linked with more frequent and/or severe unwanted intrusive thoughts, feelings, and recollections about a stressful life event.

Hypothesis 1.3. We hypothesize that out of the possible links between OCD and PTSD symptom pairs, distress caused by obsessions and intrusive memories/thoughts/images will bear the strongest connection. This would reflect

the theorized symptom overlap between the two syndromes. In other words, distress caused by obsessions would be a bridge symptom linking OCD and PTSD, and would be at the core of the OCD symptom cycle. This would also be in line with the cognitive-behavioral model of OCD, such that greater distress about unwanted, repetitive thoughts, in turn leads to more severe compulsions that function to neutralize discomfort (Rachman, 1997, 1998).

Aim 2: Incorporating Clinical Correlates into the OCD and PTSD Network.

Given the high comorbidity of both OCD and PTSD with depression and anxiety (Ruscio et al., 2010), our final network will also include depression levels as measured by the Beck Depression Inventory-II (Depress) and anxiety levels as measured by the Beck Anxiety Inventory (Anxiety). Each will be included as a node in the network developed in Aim 1.

Hypothesis 2.1. Anxiety and/or depression may help bridge the clusters of OCD and PTSD symptoms, such that the network including these two nodes will feature one or both as central symptoms that bear a strong edge to one or more pairs of OCD and PTSD symptoms.

Methods

Participants

The total sample will consist of 1028 consecutive OCD patients from the Brazilian Research Consortium on Obsessive-Compulsive Spectrum Disorders (CTOC). A comprehensive description of the CTOC methods has been outlined by Miguel et al. (2008), including details of the clinician training procedure and inter-site demographics. Participants were recruited from seven different

universities in Brazil and were interviewed between 2003 and 2008. Inclusionary criteria included a primary OCD diagnosis per the SCID-IV. Exclusionary criteria included psychotic disorders and significant mental impairment. Inter-rater reliability was 96%. The mean age was 34.7 (SE = 0.51), with average age of OCS onset around 13 years; 84.6% were Caucasian and 56.3% were female. The mean Y-BOCS score was 24.9 (SE = 0.35). The Symmetry OCS dimension was the most common, with 87.6% reporting Symmetry symptoms; Contamination was the next most common (72.4%), followed by Obsessions about Harm (65.6%), Sexual/Religious (50.5%), and Hoarding (50.0%). 69.7% of the sample met criteria for MDD, and 15.6% met criteria for PTSD.

Measures

Structured Clinical Interview for DSM-IV (SCID; First et al., 2001). The SCID is a semi-structured interview assessing current and lifetime Axis I disorders. Participants were administered the SCID by trained clinical interviewers, and data were blind-diagnosed by two independent clinicians in order to ensure reliability. The diagnostic ability of this group in previous studies has been found to demonstrate excellent reliability (LaSalle et al., 2004).

PTSD Checklist-Civilian Version (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1993). The PCL-C is a 17-item self-report inventory that assesses DSM-IV criteria of PTSD. Individuals are asked to rate the degree to which they have been bothered by each symptom across the previous month, on a scale from 1 (not at all) to 5 (extremely). Examination of the psychometric properties of the PCL has established it as a strong, robust, measure, with good internal

consistency, test-retest reliability, and convergent validity in both individuals who have experienced trauma (e.g., Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Keen, Kutter, Niles, & Krinsley, 2008; Wilkins, Lang, & Norman, 2011) as well as in nonclinical samples (e.g., Conybeare, Behar, Solomon, Newman, & Borkovec, 2012; Ruggiero, Ben, Scotti, & Rabalais, 2003). Within the present sample, all individuals were asked to rate symptoms on the PCL-C regardless of whether or not they have experienced a traumatic event meeting full criteria for PTSD.

Yale-Brown Obsessive Compulsive Scale Interview (Y-BOCS; Goodman et al., 1989). The Y-BOCS is a gold-standard measure of OCS severity, and has demonstrated good reliability and validity (Goodman et al., 1989). The Y-BOCS Symptom Checklist first assesses the presence or absence of specific types of OCD symptoms. Participants are then asked about their endorsed obsessions and compulsions, respectively, with regards to the following factors: duration, distress, interference, resistance, and control. Each item is rated on a scale from 0 (least severe) to 4 (most severe). Responses to these five items are summed to yield the obsessions versus compulsions subscales.

Beck Depression Inventory, 2nd edition (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item measure of depressive symptom severity, with excellent internal consistency and convergent validity in both nonclinical and psychiatric populations (e.g., Beck et al., 1996; Storch, Roberti, & Roth, 2004). Individuals rate the extent to which they have experienced symptoms in the past two weeks, with each item scored on a scale of 0 to 3.

Beck Anxiety Inventory (BAI; Beck & Steer, 1990). The BAI is a 21-item measure of anxiety severity, with individuals rating each item on a scale of 0 to 3. The BAI's psychometric properties – including reliability, including internal consistency, test-retest reliability, and validity – have been found to be strong, and it has been validated in a range of populations (e.g., Hewitt & Norton, 1993; Steer, Ranieri, Beck, & Clark, 1993).

Data Analytic Approach

Estimated Regularized Network. The R package *qgraph* (Epskamp et al., 2012) was used to compute the networks. The initial network included 27 nodes as described above, with 10 OCD symptoms (Y-BOCS item scores) and 17 PTSD symptoms (derived from the PCL-C). Depression (BDI total score), and anxiety (BAI total score) will also be incorporated in the final network as two separate nodes. Each edge depicted in the network will represent the strength of association between each pair of symptoms, after statistically controlling for all the other (ten) factors in the network; essentially, an adjusted partial correlation estimated with the "least absolute shrinkage and selection operator" (LASSO) technique. LASSO is used to help circumvent the large number of parameters that would have to be estimated in a comprehensive model; by minimizing the edge estimates that are negligible, it returns a sparse (i.e., conservative) network. This estimation technique increases interpretability of the model, such that the smallest number of edges that explain the data's covariance are ultimately included (Epskamp et al., 2017). A tuning parameter is selected to adjust the sensitivity of the estimation; this parameter is calculated using the

Extended Bayesian Information Criterion (EBIC), incorporated into the *qgraph* package (Epskamp et al., 2017). Node centrality is quantified (again using *qgraph*) by examining the strength of a node, which is calculated by summing the weights (in other words, the correlation magnitudes) of each edge connected to the node. A higher node strength value indicates greater centrality, such that a node is more directly connected to other nodes in the network.

Bootstrapping Analyses. Relatively few studies have progressed beyond estimating networks to examine their stability (Santos et al., 2017). In line with procedures established by (Epskamp et al., 2017) and utilized in the most up-to-date network studies (McNally, Mair, et al., 2017; Santos et al., 2017), we conducted bootstrapping analyses with the R package *bootnet*. Results from 1000 bootstrapped networks provide an estimation of how stable centrality indicators are (i.e., by providing a coefficient between 0 and 1, with larger values reflecting higher stability). Moreover, bootstrapping results also provide confidence intervals (CIs) for the strength of each retained edge in the network, indicating accuracy and significance (i.e., whether the CI encompasses zero).

Missing Data and Power Analyses

The full sample size consists of 1028 treatment-seeking individuals with OCD; however, some were missing data on one or more of the above measures. With regards to network analyses, there are no established guidelines to our knowledge, but studies are generally considered adequately powered if there are at least 5 participants per node in the network. For Aim 1, there are 27 nodes to be considered in the network; thus, a sample size of $N \geq 135$ is sufficient. For

both Aims 1 and 2, even when utilizing listwise deletion – resulting in n = 415 – our analyses should still be sufficiently powered.

Results

Descriptive Data

Table 3.1 shows the mean, standard deviation, and ranges for the variables of interest, while Tables 3.2 and 3.3 show their zero-order correlations. 586 participants endorsed experiencing any kind of trauma, while 148 denied a trauma history (294 had missing data on the trauma questionnaire). Out of the ones who had experienced a trauma, 409 endorsed a crime-related trauma, 192 endorsed a trauma of a sexual nature, and 518 endorsed a general trauma (e.g. car accident, natural disaster, witnessing a death, or losing a loved one). However, only 15.6% of the sample met clinical criteria for PTSD.

Aim 1: A First Look at the Network Structure of OCD and PTSD Symptoms.

Figure 3.1 shows the estimated network including the 10 OCD symptoms and 17 PTSD symptoms. Figure 3.2 shows the corresponding centrality plot for this network, while Table 3.4 shows the significant edge weights. The three indicators of centrality were found to moderately correlate with one another (.70 for closeness and betweenness; .46 for strength and betweenness; .35 for strength and closeness). However, bootstrapping analyses indicated that this initial network was not stable. As can be seen in Figure 3.3, all three indicators of centrality decrease in reliability with dropped samples. Epskamp et al. (2017)'s guidelines for estimating and interpreting networks suggest a preferable stability coefficient of at least >.25 and ideally >.50; meanwhile, the bootstrapped network

only yielded a coefficient of .13 for strength (and was 0, or not applicable, for betweenness and closeness). It is likely that given our large number of nodes (and thus large number of parameters to estimate), our test sample lacked the power to present a stable bootstrapped network; though a sample of over 400 clinical OCD individuals is considerably large, it may not be sufficient for use with network analyses. The rule of thumb for the number of samples per node is a tentative suggestion, and no definitive guidelines have been established for sufficient power, to our knowledge. We thus proceeded with steps to achieve a more parsimonious and interpretable network.

As OCD symptoms were the primary focus of our investigation, we honed in on PTSD symptoms to simplify the overall network. A network of just the 17 PTSD symptoms was estimated to examine potential relations and subgroups of symptoms, as Figure 3.4 shows; the corresponding centrality plot is displayed in Figure 3.5. Factor analyses of PTSD (e.g., Asmundson et al., 2000), as well as the DSM-5's diagnostic characterization of PTSD (Association, 2013), are in support of four symptom clusters: *re-experiencing* and intrusive recollections of the traumatic event, *avoidance* of relevant stimuli, *emotional numbing*, and *hyperarousal*.

While the symptoms did appear to be generally grouped into these previously established clusters, we conducted a quantitative verification of these factors within our particular sample using the spin glass algorithm to detect symptom communities within our PTSD network and to objectively improve network stability. The spin glass community test is part of the R package *igraph*

and is used to determine clusters of nodes, or “communities”, within a network (Csardi & Nepusz, 2006). A community is detected when the number of edges and edge weights within a particular cluster is greater than the number of edges and edge weights within another group of nodes (Heeren & McNally, 2016).

Results revealed four communities within the PTSD network, which largely reflected the previously established four-factor analyses of PTSD symptoms and is also in line with the DSM-5 conceptualization of PTSD (American Psychiatric Association, 2013). The avoidance cluster remained the same, including avoidance of thoughts/feelings as well as avoidance of activities/situations reminiscent of the stressful life event. The re-experiencing cluster was almost identical as well, including intrusive memories, traumatic dreams, flashbacks, feeling upset, and physiological activity; the spin glass community included amnesia in this group as well. The main difference between the spin glass results and that of previous factor analyses was the division of the hyperarousal group of symptoms, which in the current literature includes sleep difficulties, anger, difficulty concentrating, hypervigilance, and feeling easily startled. Our spin glass algorithm detected hypervigilance and feeling easily startled as comprising their own community (see Figure 3.4). Conceptually, this grouping is intuitive as these two symptoms are physiological features of hyperarousal, while the others tend to be more related to affective hypersensitivity. It is thus reasonable that the spin glass community grouped sleep difficulties, anger, and difficulty concentrating, with other features of emotional disturbances – feeling distant from others, emotionally numb, a loss of interest in activities, and a sense of future

foreshortening. We named this modified cluster “emotional dysregulation”, and clarified the hypervigilance-startle cluster as “hyperarousal-physiological”. Given that the symptoms largely align with previous factor analyses, and that the minor changes are rational especially in light of our sample of treatment-seeking OCD individuals (i.e., may differ slightly compared to a primary PTSD sample), we were confident in retaining these four communities – avoiding, re-experiencing, emotional dysregulation, and physiological hyperarousal – for the next step of our network analyses.

Figure 3.6 displays the adjusted network, incorporating the four spin glass-derived PTSD clusters and 10 OCD symptom nodes. Given our modifications to our initial network, we also included depression and anxiety as clinically relevant comorbid features in this step, to determine whether this network would be adequately stable or whether these features would necessitate further adjustment as well. Figure 3.7 shows the corresponding centrality plot for this adjusted network. The three indicators of centrality were found to moderately correlate with one another, with strength bearing the highest correlations (.47 and .46 with closeness and betweenness, respectively; .37 for closeness and betweenness). Bootstrapping analyses indicated that this adjusted network can be considered stable. As can be seen in Figure 3.8, both strength and closeness maintain a high level of reliability across dropped samples, each bearing a stability coefficient of .60, well over the guideline of at least >.25 and ideally >.50 (Epskamp et al., 2017). As strength was the centrality measure that yielded both a high stability coefficient as well as the greatest correlation with closeness and

betweenness, we will focus on strength as the primary measure of node centrality for our network.

As a whole, results were surprisingly not in support of our hypotheses for Aim 1. PTSD and OCD symptoms formed disparate groups and were not uniquely linked to one another, above and beyond the existing associations between each node pair. In fact, when conducting the spin glass algorithm in order to stabilize the PTSD network and increase reliability and interpretability, four communities were detected: 1) the PTSD clusters, 2) depression and anxiety, 3) OCD symptoms – resistance and control aspects, and 4) OCD symptoms – time occupied, distress, and interference aspects. Thus, interpretations of node strength should be cautiously interpreted, as nodes with high strength were not necessarily central within the entire network, but bore strong edge weights within their own respective community. For instance, the re-experiencing symptom cluster evidenced the highest overall node strength, significantly stronger than both the hyperarousal-physiological and avoidant symptom clusters (see Figure 3.9). However, as Table 3.5 demonstrates, re-experiencing was only significantly linked to the three other PTSD cluster nodes, and not with the OCD, anxiety, or depression nodes. Thus, although having intrusive memories/thoughts – a main symptom of the re-experiencing cluster – was predicted to be highly central (Hypothesis 1.1), our findings only support this notion with regards to other PTSD clusters, and not in relation to OCD and clinical correlates.

Similarly, our adjusted network did not support our Hypothesis 1.2 in that distress associated with obsessions (ObsDis) was not found to be highly central. Out of the OCD symptoms, interference due to obsessions (ObsInt) and interference due to compulsions (ObsComp) yielded the highest node strength (Figure 3.7). However, their relative strength in the network was explained by their interconnectedness with other OCD symptoms – in particular, interference and time occupied by OCD – rather than with the PTSD clusters or with anxiety or depression (Figure 3.6). As noted above, the OCD symptoms formed two distinct communities as detected by the spin glass algorithm – one being the resistance and control aspects, the other including time occupied, distress, and interference due to obsessions and compulsions. Thus, each of the 10 OCD symptom nodes was interconnected within their own community, but none of them demonstrated a significant edge to a PTSD node, nor to anxiety or depression (Table 5). In other words, no pairs of PTSD and OCD nodes were significantly linked, in contrast to Hypothesis 1.3.

With regards to Aim 2, findings were in line with that above, such that anxiety and depression formed their own community of symptoms – and in fact demonstrated the strongest edge weight in our adjusted network, significantly stronger than any other edge (Figure 3.10). However, these two hypothesized clinical correlates were not critical in linking OCD symptoms and PTSD clusters past the level of existing covariance. As can be seen in Table 3.5, neither node was significantly linked to any other node in the network, with the exception of depression and the emotional dysregulation cluster of PTSD symptoms. This

connection makes sense given this cluster's symptom overlap with the BDI items reflected by the depression node (e.g., anhedonia, sleep disturbances, and concentration difficulties), and is also in line with recent network findings that PTSD and major depressive disorder may be linked via dysphoric symptoms (Afzali et al., 2017).

Discussion

Overall, this study was novel in its network approach to investigating OCD and PTSD symptoms, and illuminating in its findings. Though the initial network including all 17 PTSD and 10 OCD symptoms was not found to be stable, the spin glass algorithm proved useful in revealing communities of PTSD symptoms in line with previously established factor analyses. The adjusted network proved to be a reasonable constellation of nodes, if not generally surprising in face of our original hypotheses, yielding the following groups: (a) four PTSD symptom clusters, (b) an OCD symptom cluster associated with control/resistance, (c) an OCD symptom cluster comprising time occupied, interference, and distress, and (d) depression and anxiety as clinically relevant correlates. Yet, with few exceptions, nodes between these communities were not significantly connected above and beyond the covariance of every other node in the network.

Measurement errors may in part help to explain the pattern of results. The PCL-C, as an assessment of PTSD symptoms in the civilian population, asks participants to rate their response to "stressful life experiences", which may be interpreted in various ways by different individuals (e.g., a life-threatening

accident or assault, versus a more normative work conflict). Limiting the sample to those who have undergone a more severe or distressing event may alter responses. The PCL-C also does not specify when the SLE(s) in question occurred, but asks about the specific symptoms over the past month; those undergoing a more acute and/or recent event versus a more chronic or amorphous stressor may rate symptoms in a different way. Of note, the PCL-C inquires about symptoms over the past month, in contrast to the YBOCS measure of OCD symptoms, which in this sample asked participants to rate their current symptoms on average; meanwhile, the BDI and BAI focus on the past two weeks. Perhaps standardizing the time frame of the measures utilized would provide a more accurate cross-sectional perspective of these symptoms concurrently.

Idiosyncrasies of our sample may also partially account for the findings of our network. Out of the total sample, less than half completed the PCL-C, which limited the power of our analyses. It is also important to consider that our findings are only relevant in the context of treatment-seeking OCD individuals; results may differ in those with primary PTSD or those with comorbid OCD and PTSD, not to mention subgroups of patients who have endured a traumatic event in early childhood versus adulthood, or a severe traumatic event meeting diagnostic criteria for PTSD rather than a SLE. Moreover, the PCL-C utilizes the DSM-IV conceptualization of PTSD; it is possible that alternative diagnostic frameworks, such as the ICD-11, may alter network results. The ICD-11 hones in on a “narrower and briefer” set of PTSD symptoms – excluding nonspecific ones such

as sleep problems and trouble concentration (Stein et al., 2014, p. 4), which could not only reduce the number of estimated parameters, but also reveal stronger links between the remaining symptom nodes.

Nonetheless, our network provides a unique viewpoint from which to interpret the OCD and PTSD symptom networks separately. The 10 OCD symptoms, reflected by items from the YBOCS, formed two clusters of control/resistance and time occupied, interference, and distress. This may complement our overall understanding of OCD, which is usually characterized by the two YBOCS subscales of obsessions versus compulsions. Especially in the context of PTSD, it is interesting that control over and resistance against both obsessions and compulsions formed its own community. Specifically, a characteristic often associated with post-trauma reactions and trajectory is individual differences in “loss of control” related to the event (Başoğlu & Mineka, 1992; Foa, Zinbarg, & Rothbaum, 1992; Maes, Delmeire, Mylle, & Altamura, 2001). Perceived loss of control – which can include not only of one’s physical surroundings but also of one’s emotional and mental state (Ehlers, Maercker, & Boos, 2000) – has been linked with worse coping ability and higher PTSD severity, after both war-related trauma (Dekel, Mandl, & Solomon, 2011) and sexual assault (Ullman & Peter-Hagene, 2014). This may be similar to how many individuals with OCD endorse a strong urge to exert control over their thoughts and behaviors (Moore & Abramowitz, 2007; Moulding & Kyrios, 2006; Reuven-Magril, Dar, & Liberman, 2008), which points to the importance of effective management of rigid controllability beliefs, such as acceptance and focused

distraction (Najmi, Riemann, & Wegner, 2009). Exposures and mindfulness techniques could routinely address not only the symptom-specific intrusions and rituals, but also the fear of “going crazy” or “losing all autonomy” if one lacks absolute control.

Within the other cluster of OCD symptoms: time occupied, distress, and interference – each associated with obsessions as well as compulsions – none of the six symptoms was significantly more central than one another. Though it is possible that greater power may demonstrate otherwise, our current model indicates that each symptom is important to consider in the conceptualization of OCD, in line with the cognitive-behavioral model of its etiology and maintenance. Moreover, for obsessions and compulsions, each corresponding symptom type yielded a significant edge (i.e., time occupied by obsessions was linked to time occupied by compulsions, and so forth). As in the other cluster of OCD symptoms, this may point to the importance of considering the obsessions and compulsions subscales not only separately, but in conjunction for matching symptom types. Further research may explore the possibility that reducing time occupied by obsessions may *directly* – as well as indirectly – reduce time occupied by compulsions (and likewise for distress, interference, resistance, and control). With regards to intervention implications, these corresponding edges may highlight how clinicians should target OCD symptoms from both angles of obsessions and compulsions. It may benefit outcomes to focus not only on exposures to feared consequences, but also to emphasize the perhaps less

acknowledged response prevention aspect of treatment, cutting down on present rituals and behavioral avoidance.

With regards to the PTSD network, re-experiencing was the most central symptom cluster, and had significant edges to each of the other three PTSD clusters. This is reasonable within our sample of treatment-seeking OCD individuals, as those with unwanted intrusive thoughts may be more likely to endorse re-experiencing symptoms – including repeated, disturbing memories, thoughts, images, or dreams – as a reaction to trauma or SLEs. Interestingly, results may extend to primary PTSD samples as well – a recent network study found intrusive memories to be a central symptom in both the acute (hospital admission) and chronic (12 month follow-up) phases of PTSD for trauma survivors, with strengthened connectivity between re-experiencing symptoms in the chronic phase (Bryant et al., 2017). Further investigations could clarify whether severity of re-experiencing symptoms may exacerbate that of other clusters, which could warrant greater incorporation of mindfulness and acceptance techniques, or strategies to combat dysfunctional beliefs involving importance of thoughts. These techniques may be particularly effective in a sample with comorbid OCD and PTSD, which treatment studies could explore. Finally, as the two clinical correlates included, depression and anxiety actually yielded the strongest edge between any node pairs in the network. This may hold true in many if not all psychiatric samples, pointing to the need to alleviate one or both in efforts to improve quality of life. Though they were not significantly connected to most other nodes above and beyond controlling for all other

relations in the network, it may be the case that these features are linked to overall symptom severity, rather than one particular symptom.

Findings of this study should be considered with several limitations in mind. Of note, while our adjusted sample of over 400 individuals – comparatively large for a clinical OCD study – our network does include many nodes, and thus, a relatively large number of parameters to estimate (see McNally, Heeren, & Robinaugh, 2017 for further discussion about robustness of networks relative to sample size). To-date, there are no relevant fit indices that can be determined to assess network findings' model of the data; in general, networks' stability and thus interpretability is improved with larger samples, which further studies could accomplish with more complete data. Given our reduced power, we were unable to examine more specific group differences that may play a role in the nature of OCD and trauma (e.g., gender, type of SLE, or age of OCD or SLE onset). Similarly, the sample size and retrospective database prevented inclusion of additional clinical correlates that may be relevant, such as anxiety sensitivity, distress tolerance, importance of thoughts, inflated sense of responsibility, or obsessive beliefs. Furthermore, given the heterogeneous nature of both OCD and PTSD in conjunction with the subjective nature of network conceptualization, it is possible that alternative formations are equally valid, such as clustering symptoms in another manner, dropping specific symptoms from analyses, or substituting other nodes (for instance, OCD symptom dimensions). Finally, the Brazilian sample may preclude cross-cultural generalizations; it may be possible that Latino populations may tend to perceive and/or report psychiatric symptoms

differently compared to other ethnic groups (e.g., Minsky, Vega, Miskimen, Gara, & Escobar, 2003; Wheaton, Berman, Fabricant, & Abramowitz, 2013).

This study is the first to our knowledge to utilize network analysis to examine the interplay between OCD and trauma symptoms, particularly in a clinical population. Though power was somewhat limited, our sample of treatment-seeking OCD individuals is considered large in the OCD literature. Our findings provided insight into the OCD and PTSD symptom networks, and indicated that they may be more independent than initially expected, and not related via anxiety or depression (above and beyond existing covariance). Results provide a solid foundation from which to further explore network systems to conceptualize OCD, PTSD, and other correlates that may play a role in symptom overlap.

Chapter 4: General Discussion

The overarching aim of this investigation was to conduct an initial foray into the interrelation between OCD and trauma by utilizing novel network analyses, as an alternate approach to the traditional latent model perspective. With two separate OCD samples, Studies 1 and 2 provided complementary perspectives in conceptualizing how trauma and OCD may be related. Study 1 broadly investigated the network of OCD symptom dimensions along with obsessions and compulsions severity, in relation to diagnostic status of common comorbid syndromes. Findings highlighted the importance of aggressive, sexual, religious obsessions and checking compulsions as a central feature, through which other OCD features are connected. With regards to trauma, Study 1 compared this OCD network between subgroups of those with versus without a trauma history. Though lack of power likely prohibited significant group differences, qualitative observations provided initial insight that those without a trauma background yielded a much sparser, loosely connected OCD network, in line with previous evidence that trauma is an important vulnerability factor for OCD (e.g., Cromer et al., 2007; Real et al., 2011). Meanwhile, Study 2 zoomed in on symptom-level relationships between OCD and PTSD. In contrast to expectations, PTSD symptom clusters were largely not connected to OCD hallmark features of obsession and compulsion severity (above and beyond existing covariance between network nodes). Meanwhile, OCD symptoms formed clusters – not obsessions versus compulsions, as the subscales would suggest – but rather one associated with control and resistance, and another

including time occupied, interference, and distress. This provides fodder for future exploration into the potential link between greater perceived loss of control after a trauma (Başoğlu & Mineka, 1992; Foa et al., 1992; Maes et al., 2001) and heightened desire for control in OCD (Moore & Abramowitz, 2007; Moulding & Kyrios, 2006; Reuven-Magril et al., 2008).

As a whole, this investigation illustrated the utility of the network approach in studying psychopathology, in line with its recent applications with other disorders and questions of comorbidity (e.g., Frewen et al., 2013; Heeren & McNally, 2016; McNally, Mair, et al., 2017; Robinaugh et al., 2014; Santos et al., 2017; van Borkulo et al., 2015). Not only are network graphs strikingly visual in facilitating interpretation of results, but findings provide insight into the dynamic interrelations between symptoms that may perpetuate cycles of onset and maintenance – some of which may spark further clinical and/or theoretical exploration (Borsboom & Cramer, 2013; McNally, 2016). Group differences between network structure and strength can be explored, as well as specific clusters of nodes or symptoms within a larger network.

Naturally, cons of the network approach should be addressed as well. For one, the conceptualization of which nodes to include in each analysis can often be up for interpretation. At times, it can be more straightforward in strictly including diagnostic criteria for a singular disorder like PTSD (McNally et al., 2015) or depression (Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2015). Yet, a more complex study question (e.g., involving comorbidity issues, or how to best operationalize a heterogeneous disorder such as OCD) can understandably

complicate node selection. For each of our studies, the primary aim helped guide our selection of nodes, but several other options may have been valid as well. In our case, node selection was also constrained by our use of archival data. Additionally, networks are more stable with increased sample size (Epskamp et al., 2017; Epskamp et al., 2012). Despite our samples being relatively large for clinical OCD studies, they admittedly limited the extent of the fine-grained analyses within the scope of our studies. Larger samples may allow investigators to examine various types of trauma, or to compare subgroups of OCD symptom dimensions. This also highlights the importance of complete data when using network analyses. For instance, the NCT cannot handle missing data, which reduces power in making group comparisons. Also, using pairwise versus listwise deletion may yield different findings. Future investigators may also benefit from applying extensions of network approaches, such as Bayesian algorithms that could generate more directional interpretations of symptom cycles (e.g., McNally, Mair, et al., 2017). Another possible venue that some researchers have begun to explore is comparing temporal differences of networks over time, using experience sampling methods (e.g., Bringmann, Lemmens, Huibers, Borsboom, & Tuerlinckx, 2015; Bringmann et al., 2016). Of course, with such a relatively novel method, it is important to not overreach with regards to interpretations; for instance, some groups have compared networks between subgroups of varying symptom severity (Wigman et al., 2013), but others have cautioned that differences may be accounted for by disparities in variance between groups (Terluin, de Boer, & de Vet, 2016).

Moreover, it may be the case that additional factors outside of the scope our two studies may be important for other researchers to explore with regards to OCD and trauma. For instance, it is possible that variability in cognitive variables associated with SLEs (e.g., high thought suppression, and/or low perceived control) may be more important in predicting OCS (McLaren, & Crowe, 2003). Other individual differences in levels of overinflated sense of responsibility, importance of thoughts, tolerance of uncertainty, and desire for control may also be illuminating in helping connect OCD and PTSD networks, along with comorbid symptoms. Specifically, it may be fruitful to further explore repetitive negative thinking – recent evidence points to this as a transdiagnostic factor spanning a range of affective disorders (Arditte, Shaw, & Timpano, 2016) and may underlie both rumination characteristic of depression and the intrusive thoughts present in OCD (Shaw, Carbonella, Arditte, & Timpano, in press); this raises the possibility that it may be linked with overlapping PTSD symptoms as well, particularly that of the re-experiencing cluster. On an even more fine-grained level, future research could explore these traits in relation to specific types of OCD dimensions and/or categories of SLEs. There are a myriad of nodes that could be interesting to explore when considered conjointly in a network. For instance, type of SLE has been found to relate to different depressive profiles (Keller, Neale, & Kendler, 2007) and impact PTSD symptom severity (Lancaster, Melka, & Rodriguez, 2009). It may be the case that SLEs of an interpersonal nature (versus non-interpersonal ones) would be differentially linked to dysfunctional beliefs associated with OCD such as low perceived control and inflated responsibility

(McLaren & Crowe, 2003; Moulding & Kyrios, 2007; Moulding, Kyrios, & Doron, 2007), which may act as a bridge to actual OCD symptoms.

It is also important to note that as a notoriously heterogeneous syndrome, OCD as a disorder is difficult to conceptualize, particularly as nodes in a network. There are many permutations of features that may be worthy of investigation, depending on the particular research question (i.e., symptom dimensions, symptom frequency count, total severity, obsessions versus compulsions subscale severity, and/or more specific severity indicators). Based on each study's overarching aim, and the constraints of each database, we attempted to best capture the nature of OCD in each study. With our goal of taking a broad look at the OCD network and comparing that between trauma versus no trauma history group, Study 1 made a compelling argument for including both symptom dimension factor scores and obsessions and compulsions severity in the network. Alternatively, with Study 2, we were more interested in focusing on a symptom-level analysis of severity with overlapping PTSD symptoms; thus, we went with specific YBOCS item scores to look at potential mechanisms of obsessions and compulsions that may cut across symptom dimensions. Yet, future studies may make a valid case for various network conceptualizations, such as comparing networks between primary symptom dimensions, contrasting levels of overall OCD diagnostic status, examining waxing and waning of OCD symptoms over time, or delineating childhood versus adult onset OCD.

Overall, these two studies complement one another as an initial foray into the link between OCD symptoms and trauma. The present studies were focused

on a sample of OCD individuals (lifetime OCD in Study 1; treatment-seeking current OCD in Study 2). Our particular networks may produce different findings in a sample of those diagnosed with PTSD or in a subset of individuals who have all experienced a traumatic event. Key findings reflected in our results include: repugnant and unwanted obsessions and checking compulsions as a key factor tying together the OCD symptom network; initial evidence that OCD symptoms are more tightly connected in those with a background of trauma; symptom dimensions yielding differential links to the rest of the network; and contrary to expectations, no outstanding edges bridging OCD and PTSD symptoms above and beyond all other symptoms. With an eye towards future research, it may be interesting to explore clusters of YBOCS items (as indicators of obsessions and compulsions severity) in relation to themes of control and resistance in trauma, as well as transdiagnostic features that may play a role in bridging symptoms of OCD and PTSD.

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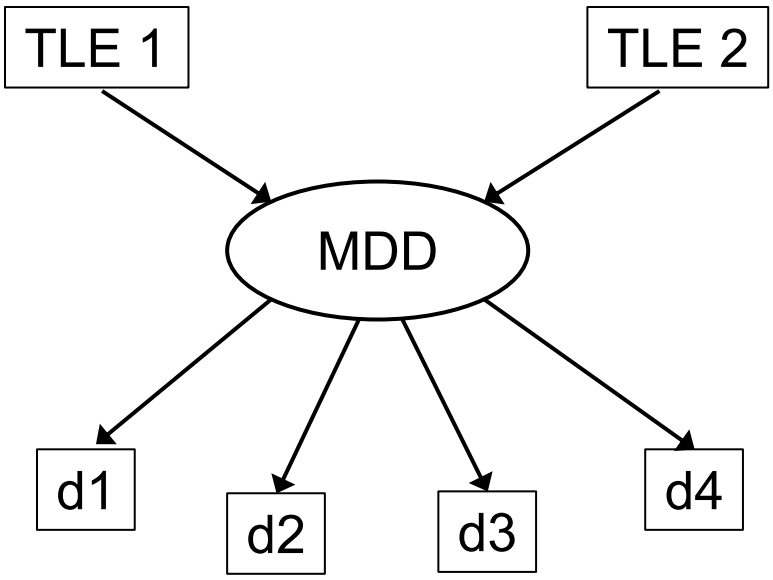


Figure 1.1. Latent variable model of the impact of traumatic life events (TLEs) on the latent condition major depressive disorder (MDD), which gives rise to depressive symptoms (d1-d4).

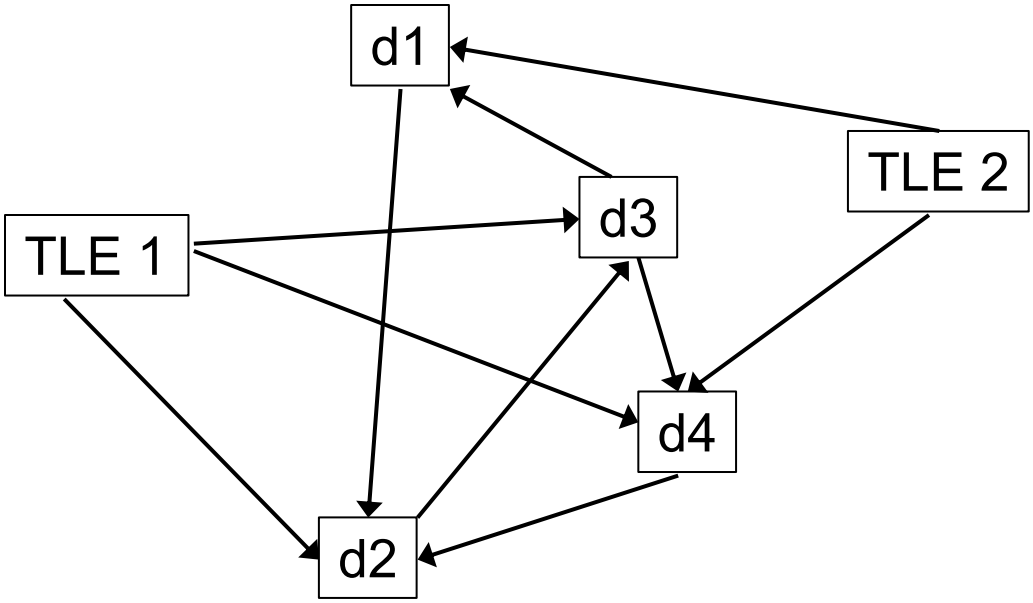


Figure 1.2. Network model of how traumatic life events (TLEs) directly impact depressive symptoms (d1-d4), which causally interact with one another.

Table 2.1

Means and Ranges for Study 1 Variables of Interest

	Variable	Range	*M*	*SD*
1	TLE	0 - 5	.9	1.12
2	ObsSever	0 - 20	10.76	5.25
3	CompSever	0 - 20	10.51	4.79
4	Obs/Check	-1.81 - 1.96	-.11	.97
5	Symm/Order	-1.91 - 1.48	-.094	.99
6	Cont/Clean	-2.17 - 2.14	-.11	1.00
7	Hoard	-1.17 - 1.64	-.0012	.98
8	AnxDx	0 - 6	1.04	1.20
9	MoodDx	0 - 5	.96	1.15
10	SubsDx	0 - 2	.35	.63
11	OCSDx	0 - 11	2.07	2.32
12	OCDOnset	3 - 60	14.25	9.52

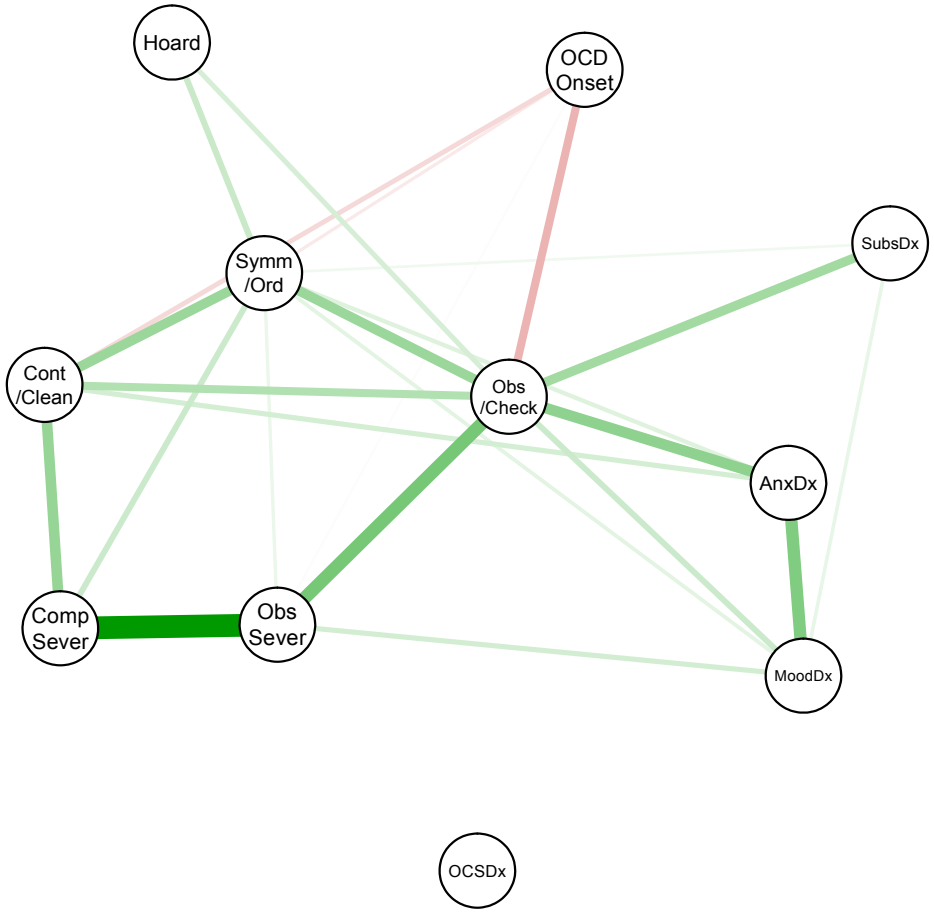
Note. TLE = count of lifetime traumatic life events; ObsSever = Yale–Brown Obsessive-Compulsive Scale (Y-BOCS) obsessions score; CompSever = Y-BOCS compulsions score; Obs/Check = Y-BOCS-CL obsessions/checking; Symm/Order = Y-BOCS-CL symmetry/ordering; Cont/Clean = Y-BOCS-CL contamination/cleaning; Hoard = Y-BOCS hoarding; AnxDx = count of total number of anxiety disorder diagnoses; MoodDx = count of total number of mood disorder diagnoses; SubsDx = count of total number of substance/alcohol disorder diagnoses; OCSDx = count of total number of obsessive-compulsive spectrum disorder diagnoses; OCDOnset = age of OCD onset.

Table 2.2

Correlations between Study 1 Variables of Interest

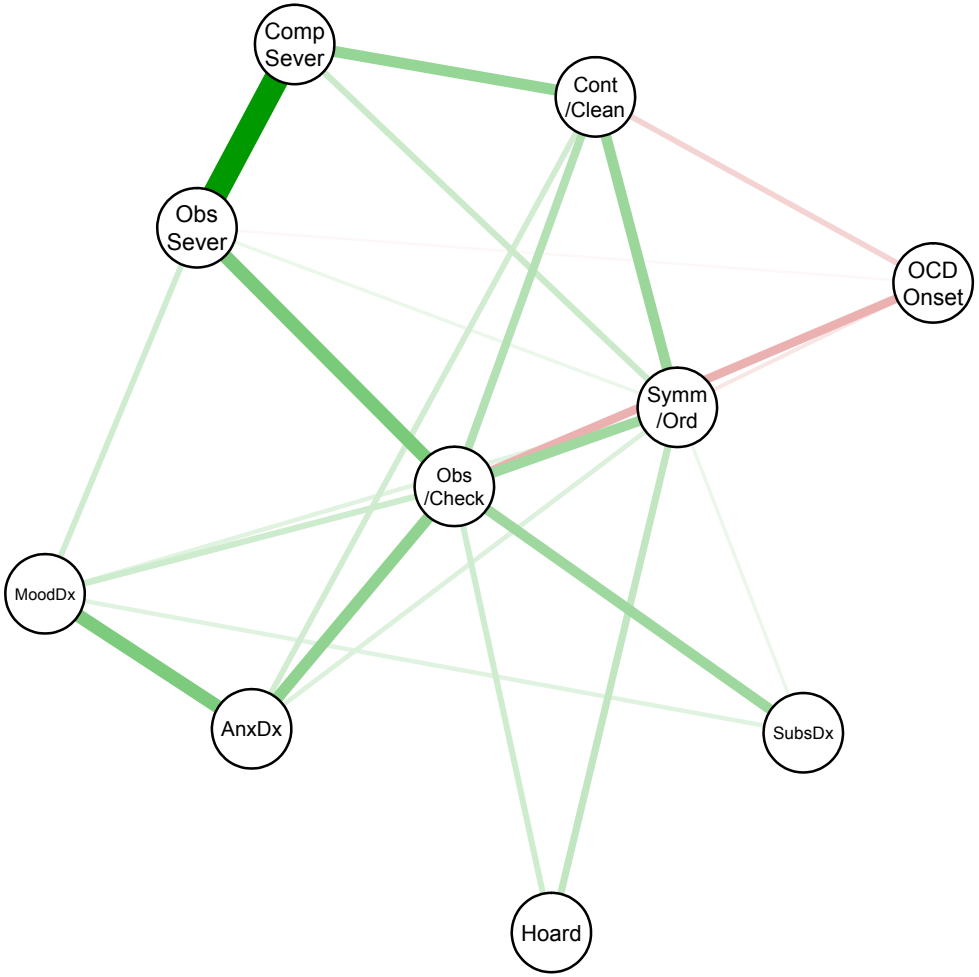
	Variable	2	3	4	5	6	7	8	9	10	11	12
1	TLE	.22**	.16*	.28**	.27**	.15*	.02	.19**	.20**	-.02	.00	-.10
2	ObsSever	-	.62**	.48**	.33**	.30**	.05	.25**	.24**	.10	-.01	-.07
3	CompSever		-	.28**	.34**	.40**	.13	.21**	.22**	.02	.06	.00
4	Obs/Check			-	.44**	.40**	.23**	.38**	.38**	.23**	-.02	-.14*
5	Symm/Order				-	.40**	.23**	.26**	.27**	.15*	-.03	-.15*
6	Cont/Clean					-	.11	.28**	.28**	.10	.06	-.08
7	Hoard						-	.11	.11	.07	.08	-.10
8	AnxDx							-	.97**	.13*	.03	-.03
9	MoodDx								-	.10	.00	-.04
10	SubsDx									-	.06	-.03
11	OCSDx										-	.10
12	OCDOnset											-

Note. *$p < .05$; **$p < .01$. TLE = count of lifetime traumatic life events; ObsSever = Yale–Brown Obsessive-Compulsive Scale (Y-BOCS) obsessions score; CompSever = Y-BOCS compulsions score; Obs/Check = Y-BOCS-CL obsessions/checking; Symm/Order = Y-BOCS-CL symmetry/ordering; Cont/Clean = Y-BOCS-CL contamination/cleaning; Hoard = Y-BOCS hoarding; AnxDx = count of total number of anxiety disorder diagnoses; MoodDx = count of total number of mood disorder diagnoses; SubsDx = count of total number of substance/alcohol disorder diagnoses; OCSDx = count of total number of obsessive-compulsive spectrum disorder diagnoses; OCDOnset = age of OCD onset.



ObsSever = Obsessions severity
CompSever = Compulsions severity
Obs/Check = obsessions/checking
Symm/Order = symmetry/ordering
Cont/Clean = contamination/cleaning
Hoard = hoarding
AnxDx = anxiety disorders
MoodDx = mood disorders
SubsDx = substance/alcohol disorders
OCD onset = age of OCD onset
OCSDx = OC-spectrum disorders

Figure 2.1. Initial network for full sample, including OCSDx as a node.



ObsSever = Obsessions severity
CompSever = Compulsions severity
Obs/Check = obsessions/checking
Symm/Order = symmetry/ordering
Cont/Clean = contamination/cleaning
Hoard = hoarding
AnxDx = anxiety disorders
MoodDx = mood disorders
SubsDx = substance/alcohol disorders
OCD onset = age of OCD onset

Figure 2.2. Revised network for full sample, excluding OCSDx as a node.

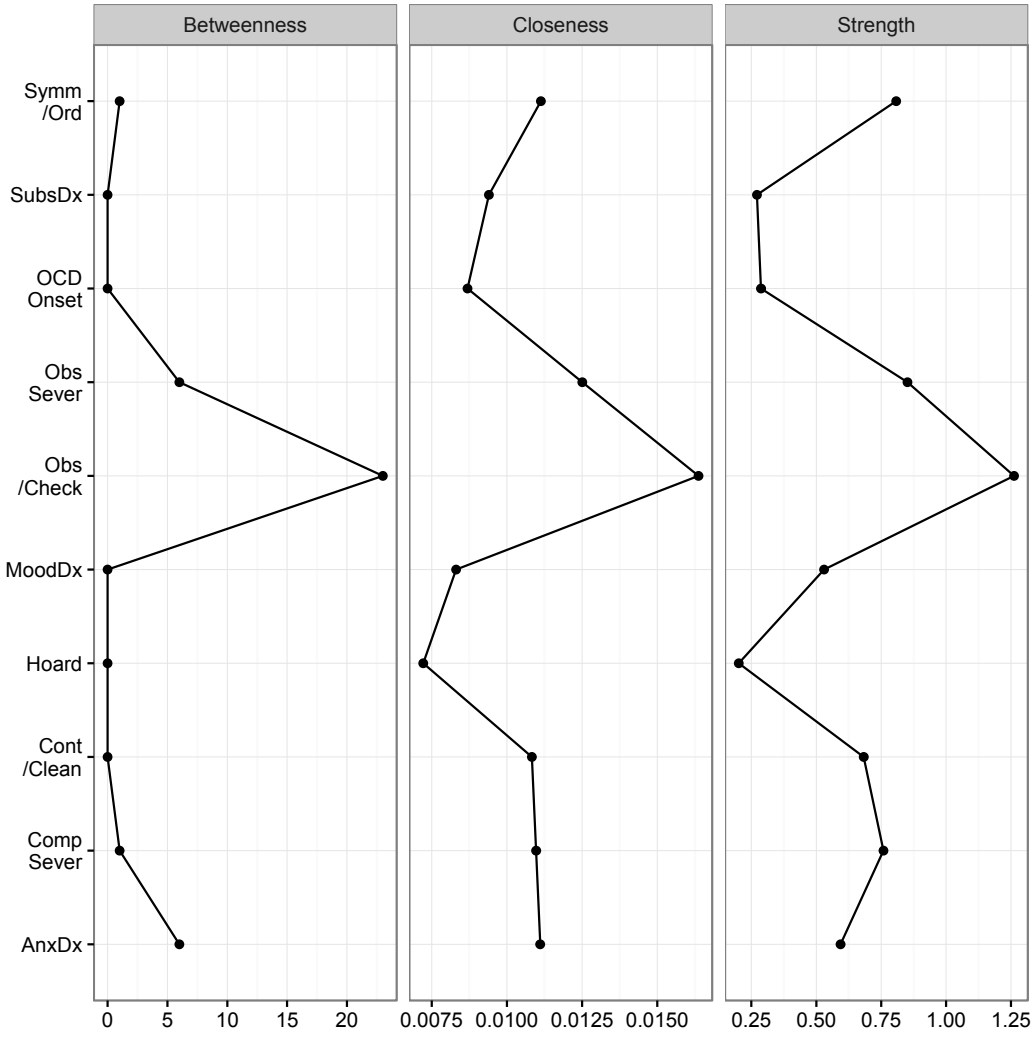


Figure 2.3. Centrality plot, including betweenness, closeness, and strength, for adjusted network shown in Figure 2.2.

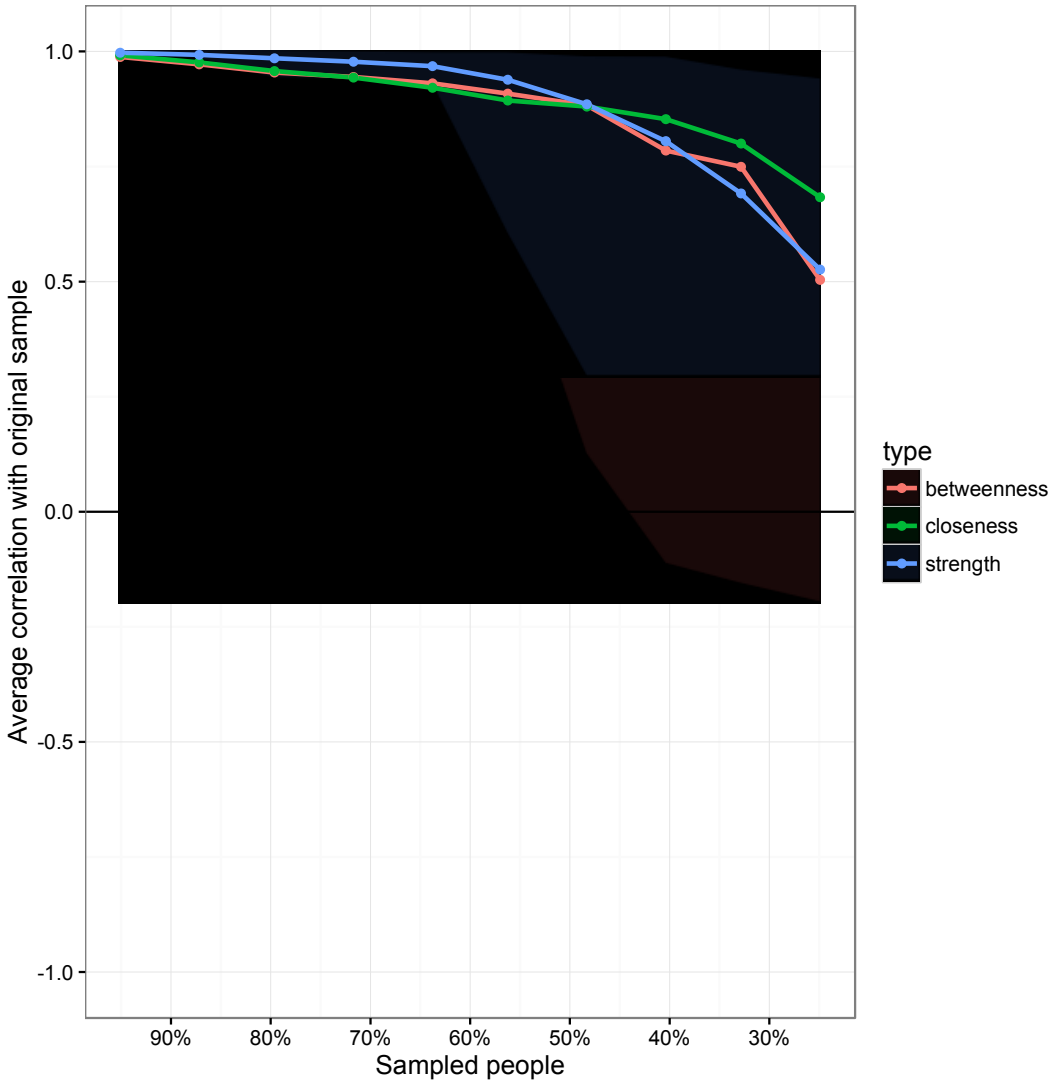


Figure 2.4. Bootstrapped dropped samples plot for bootstrapped results of adjusted network shown in Figure 2.2.

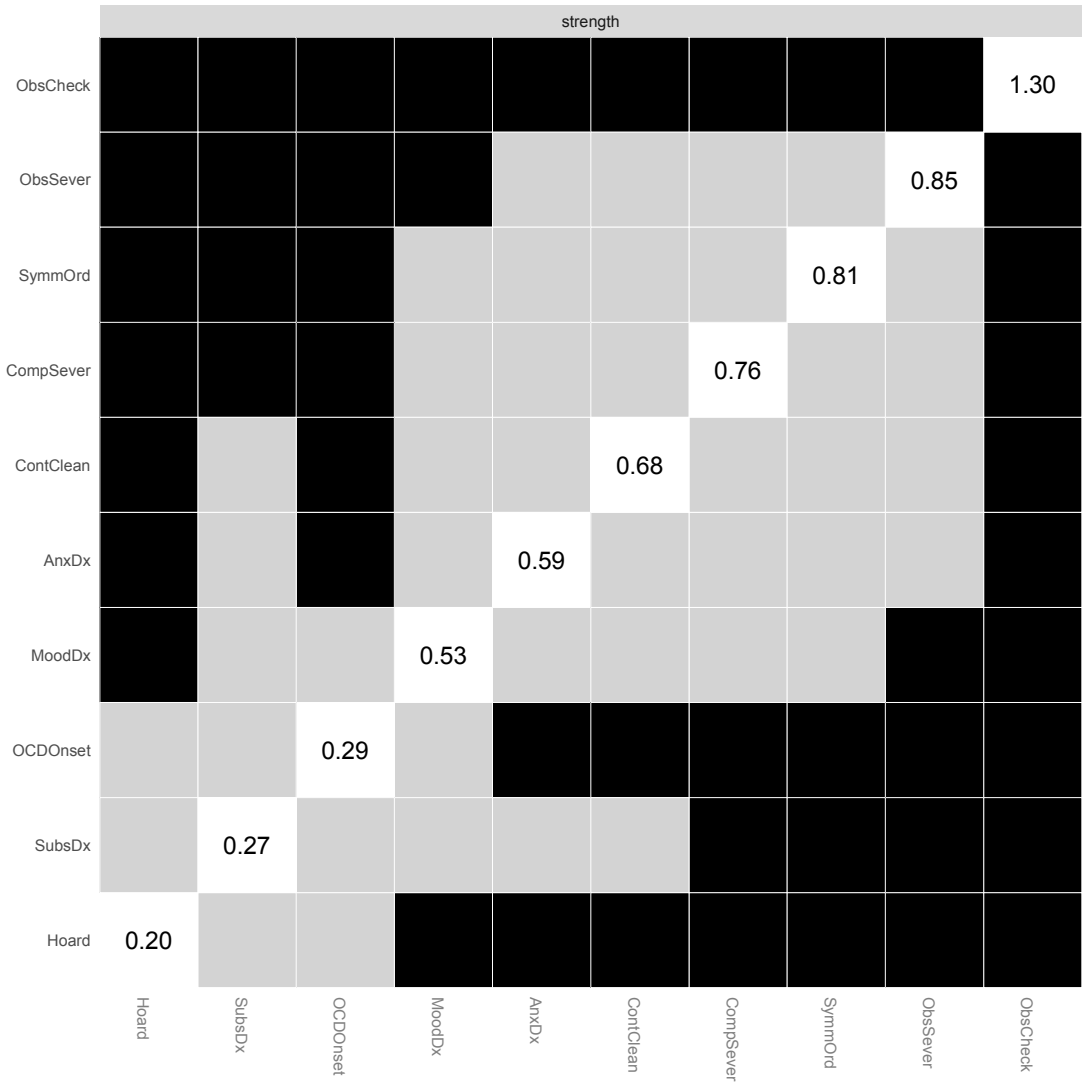


Figure 2.5. Graphical depiction of bootstrapped strength centrality results of adjusted network shown in Figure 2.2. Gray boxes indicate nodes that do not differ significantly from one another; black boxes represent nodes that do differ significantly from one another.

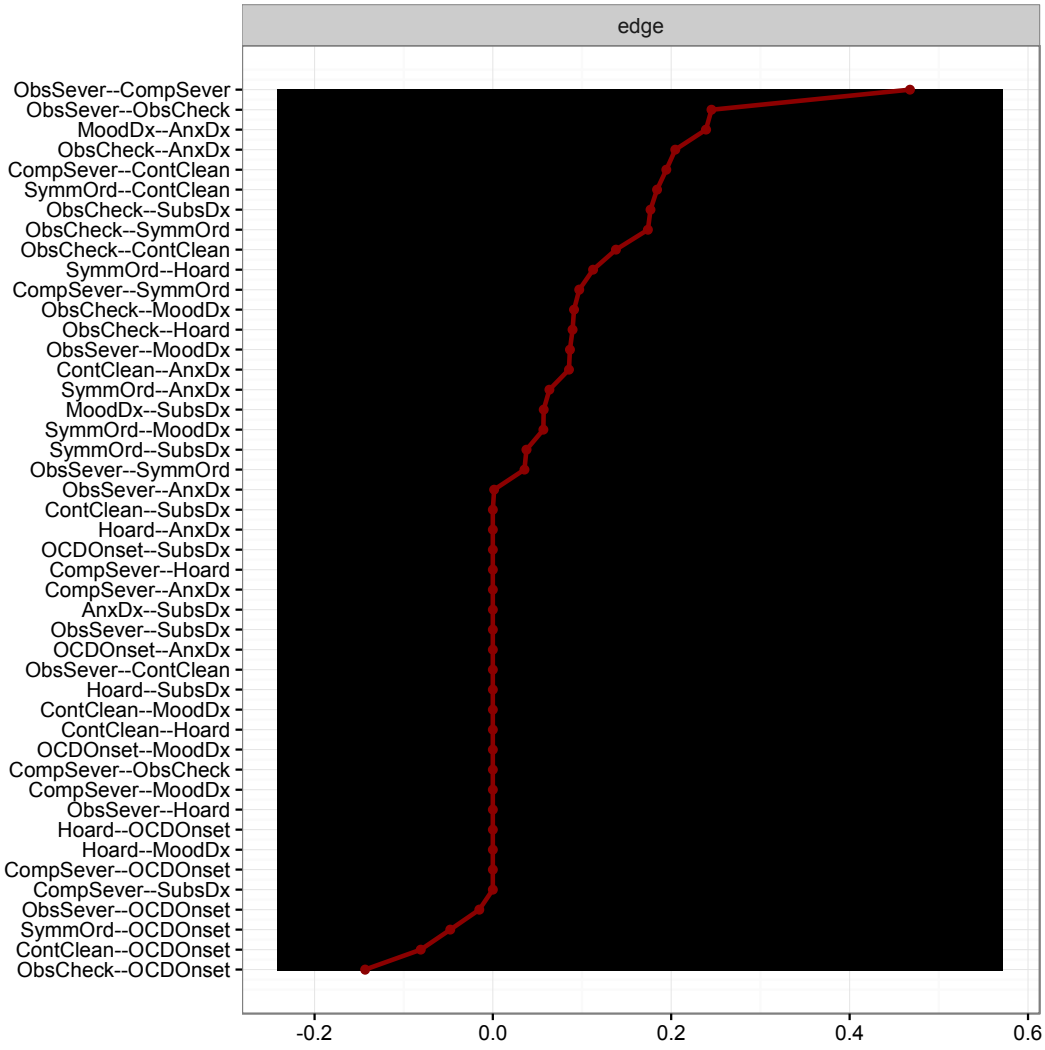


Figure 2.6. Confidence interval plot displaying bootstrapped results of adjusted network shown in Figure 2.2.

Table 2.3

Edge Weights and Confidence Intervals of OCD Network shown in Figure 2.6

Nodes	Sample Edge Weight	Lower CI (2.5%)	Upper CI (97.5%)
ObsSever--CompSever	0.47*	0.32	0.57
ObsSever--ObsCheck	0.25*	0.13	0.33
MoodDx--AnxDx	0.24*	0.084	0.36
ObsCheck--AnxDx	0.20*	0.073	0.29
CompSever--ContClean	0.19*	0.075	0.29
SymmOrd--ContClean	0.18*	0.063	0.29
ObsCheck--SubsDx	0.18	0.00	0.31
ObsCheck--SymmOrd	0.17*	0.071	0.28
ObsCheck--ContClean	0.14*	0.014	0.25
SymmOrd--Hoard	0.11	0.00	0.21
CompSever--SymmOrd	0.10	0.00	0.20
ObsCheck--MoodDx	0.091	0.00	0.22
ObsCheck--Hoard	0.089	0.00	0.18
ObsSever--MoodDx	0.086	0.00	0.20
ContClean--AnxDx	0.085	0.00	0.20
SymmOrd--AnxDx	0.063	0.00	0.18
MoodDx--SubsDx	0.057	0.00	0.20
SymmOrd--MoodDx	0.057	0.00	0.18
SymmOrd--SubsDx	0.038	0.00	0.20
ObsSever--SymmOrd	0.035	0.00	0.15
ObsSever--AnxDx	0.001	0.00	0.10
AnxDx--SubsDx	0.00	-0.041	0.14
CompSever--AnxDx	0.00	0.00	0.099
CompSever--Hoard	0.00	0.00	0.11
CompSever--MoodDx	0.00	-0.075	0.049
CompSever--ObsCheck	0.00	0.00	0.001
CompSever--OCDOnset	0.00	-0.092	0.00
CompSever--SubsDx	0.00	-0.14	0.018
ContClean--Hoard	0.00	0.00	0.066
ContClean--MoodDx	0.00	0.00	0.079
ContClean--SubsDx	0.00	0.00	0.15
Hoard--AnxDx	0.00	0.00	0.13
Hoard--MoodDx	0.00	-0.11	0.00
Hoard--OCDOnset	0.00	-0.086	0.00
Hoard--SubsDx	0.00	-0.079	0.11
ObsSever--ContClean	0.00	0.00	0.072
ObsSever--Hoard	0.00	-0.096	0.00
ObsSever--SubsDx	0.00	-0.058	0.12
OCDOnset--AnxDx	0.00	-0.013	0.12
OCDOnset--MoodDx	0.00	-0.052	0.085
OCDOnset--SubsDx	0.00	0.00	0.14
ObsSever--OCDOnset	-0.015	-0.11	0.00
SymmOrd--OCDOnset	-0.048	-0.14	0.00
ContClean--OCDOnset	-0.081	-0.18	0.00
ObsCheck--OCDOnset	-0.14*	-0.24	-0.036

Note. *$p < .05$.

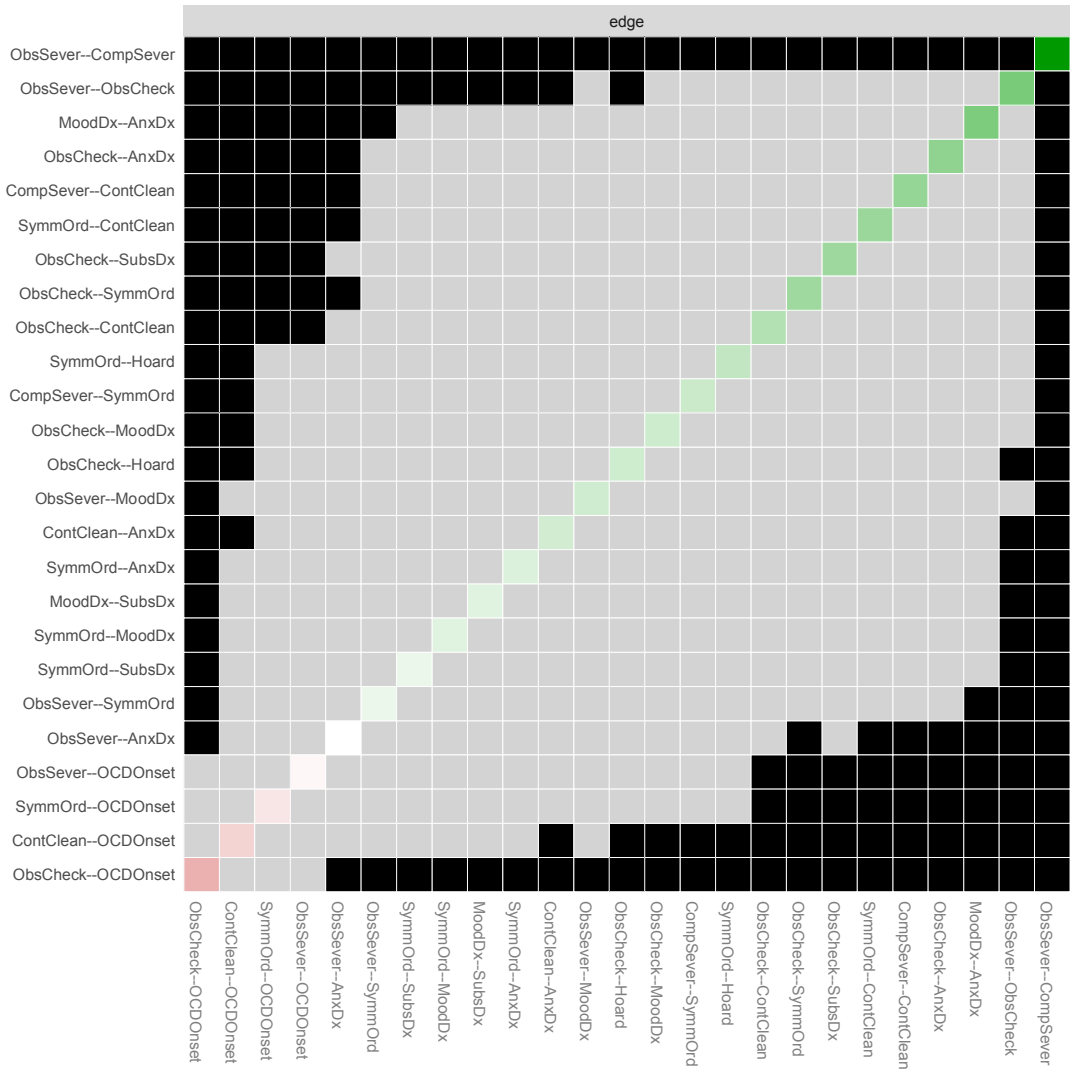


Figure 2.7. Graphical depiction of bootstrapped edge weight results of adjusted network shown in Figure 2.2. Gray boxes indicate edges that do not differ significantly from one another; black boxes represent edges that do differ significantly from one another.

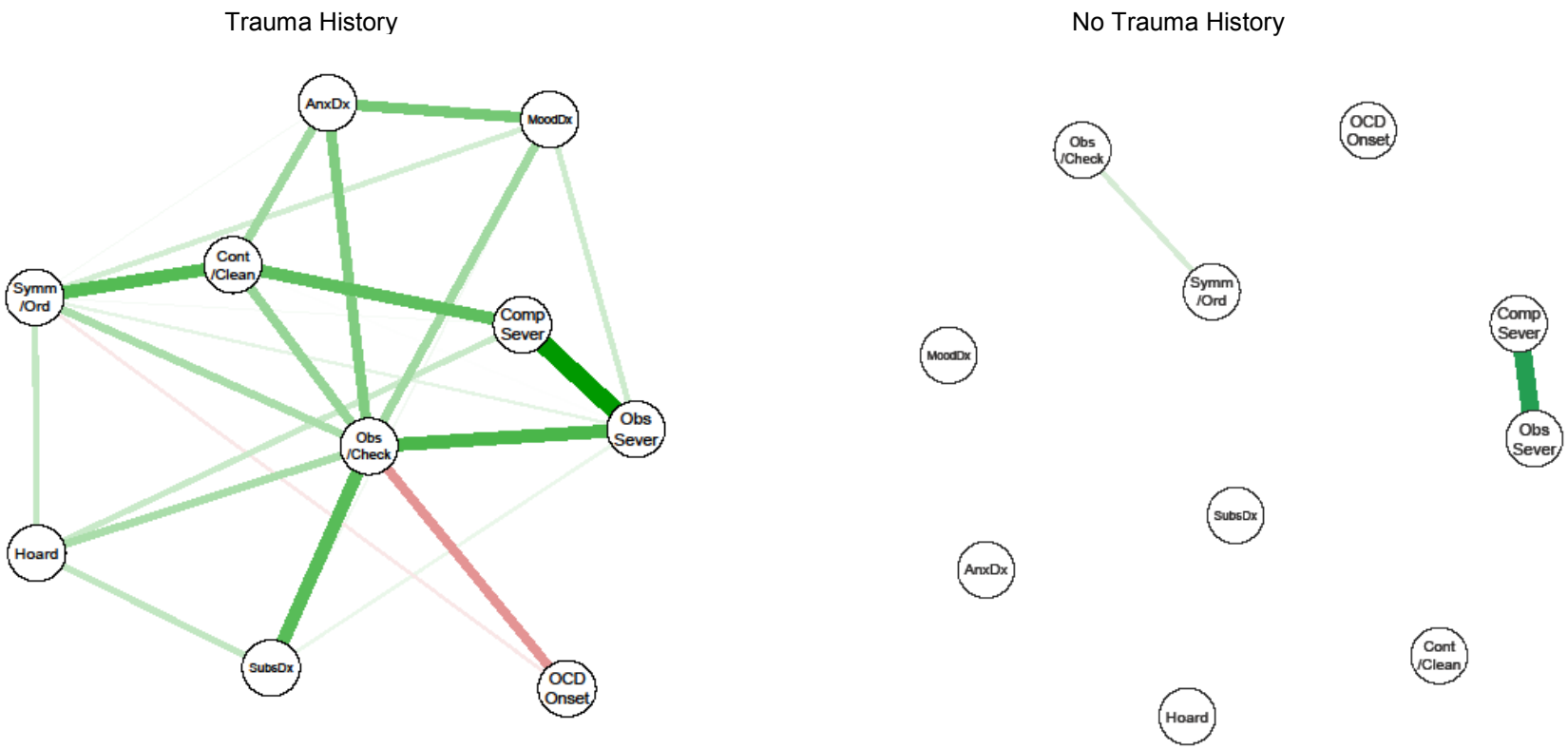


Figure 2.8. Networks for those with (left) and without (right) a trauma history, using pairwise deletion.

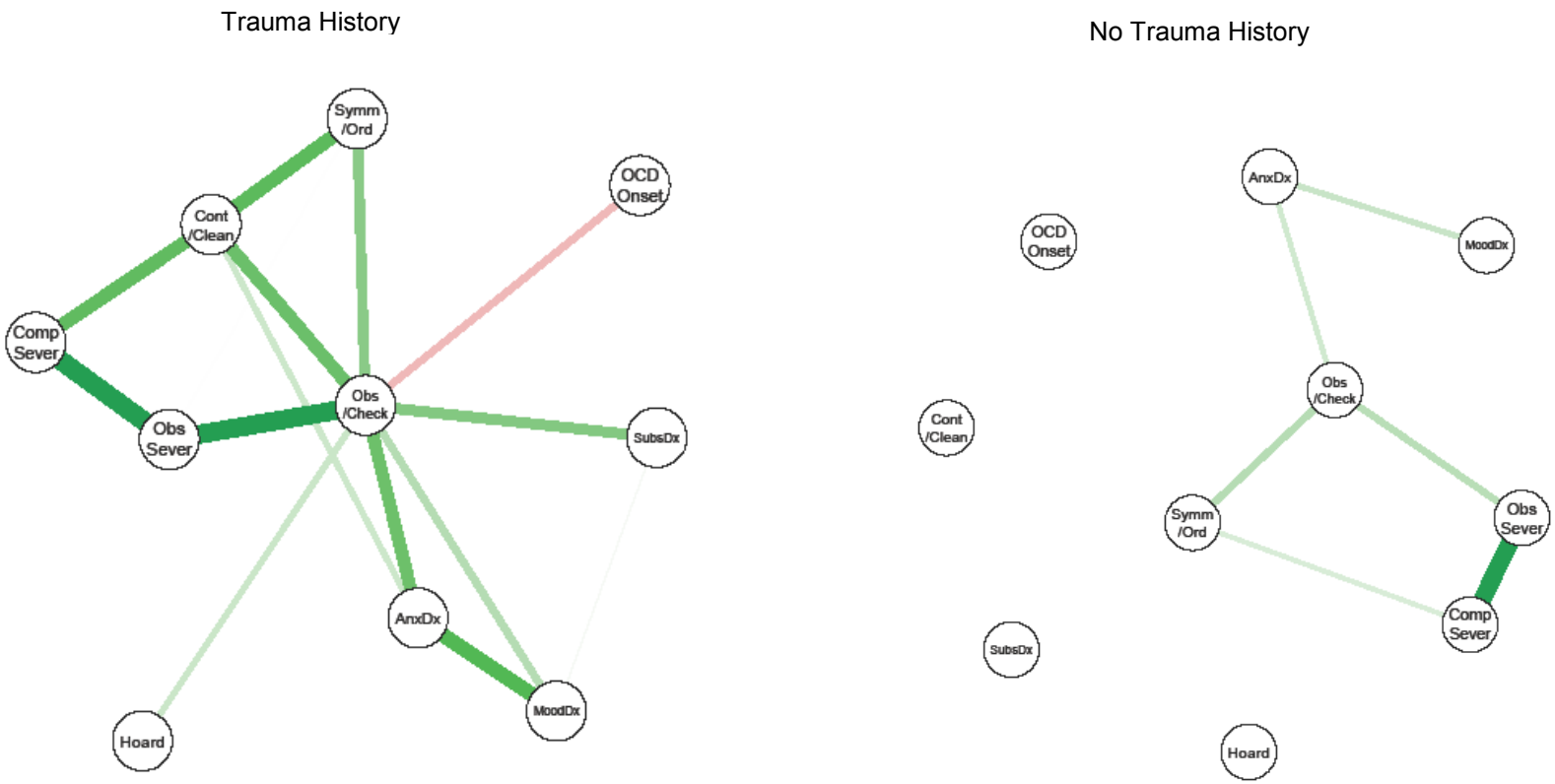


Figure 2.9. Networks for those with (left) and without (right) a trauma history, using listwise deletion.

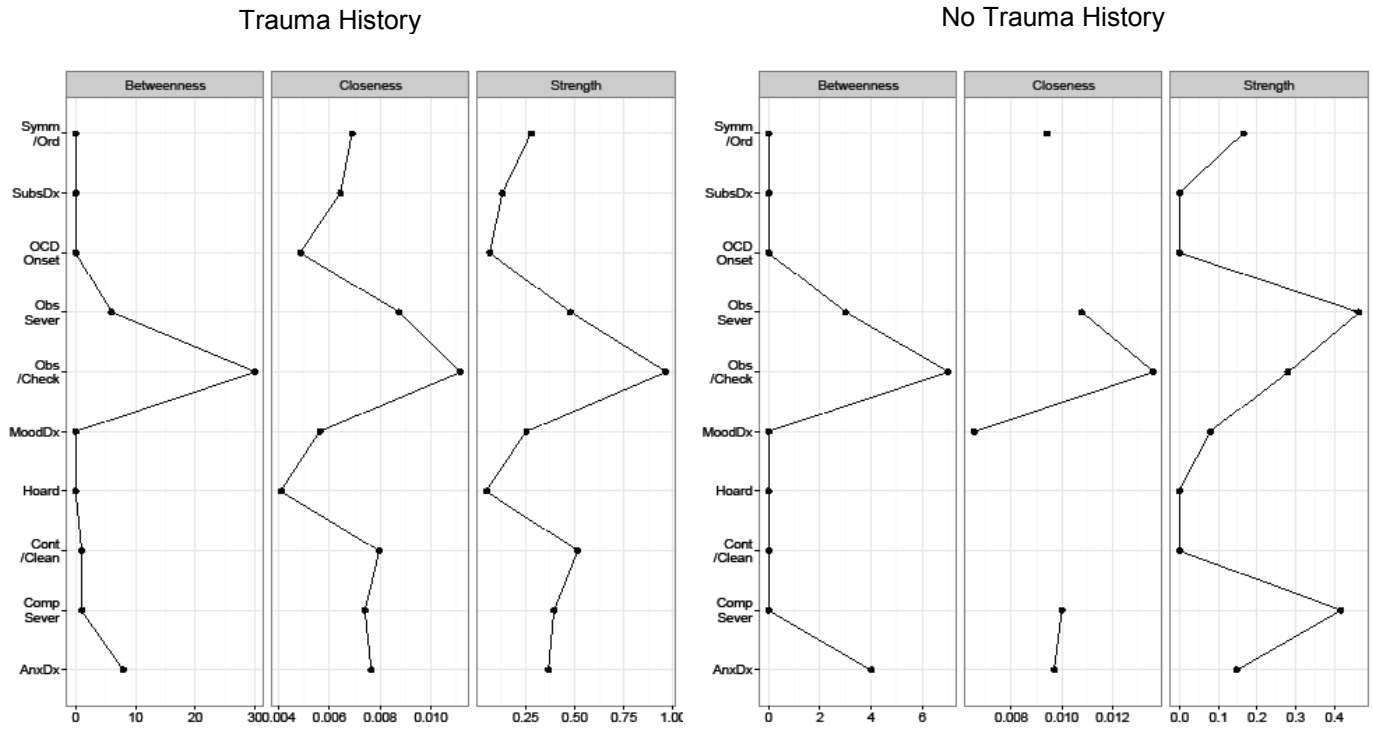


Figure 2.10. Centrality plots for those with (left) and without (right) a trauma history, based on the networks shown in Figure 2.9.

Table 3.1

Descriptives for Study 2 Variables of Interest

Variable	Range	*M*	*SD*
Y-BOCS Items			
Time occupied by obsessions	0 - 4	2.74	1.05
Interference caused by obsessions	0 - 4	2.26	.93
Distress caused by obsessions	0 - 4	2.74	.89
Difficulty resisting obsessions	0 - 4	2.11	1.25
Difficulty controlling obsessions	0 - 4	2.83	1.04
Time occupied by compulsions	0 - 4	2.58	1.00
Interference caused by compulsions	0 - 4	2.23	.97
Distress caused by compulsions	0 - 4	2.76	.97
Difficulty resisting compulsions	0 - 4	2.39	1.22
Difficulty controlling compulsions	0 - 4	2.86	1.06
PCL-C Items			
Intrusive memories, thoughts, images	1 - 5	1.67	1.14
Traumatic dreams	1 - 5	1.31	.80
Flashbacks	1 - 5	1.49	1.04
Feeling upset	1 - 5	1.98	1.39
Physiological reactivity	1 - 5	1.58	1.13
Avoidance of thoughts or feelings	1 - 5	1.80	1.33
Avoidance of activities or situations	1 - 5	1.73	1.30
Having trouble remembering	1 - 5	1.35	.89
Loss of interest in activities	1 - 5	1.60	1.17
Feeling distant from others	1 - 5	1.62	1.17
Feeling emotionally numb	1 - 5	1.35	.92
Feeling your future will be cut short	1 - 5	1.57	1.14
Difficulty falling or staying asleep	1 - 5	1.55	1.14
Feeling irritable or having angry outbursts	1 - 5	1.59	1.10
Difficulty concentrating	1 - 5	1.64	1.19
Hypervigilant, watchful, or super alert	1 - 5	1.75	1.25
Feeling easily startled or jumpy	1 - 5	1.76	1.24
PCL-C Clusters			
Re-experiencing symptoms	1 - 5	1.60	.91
Avoidant symptoms	1 - 5	1.77	1.23
Numbing symptoms	1 - 5	1.50	.83
Hyperarousal symptoms	1 - 5	1.66	1.02
BDI-II			
Depression	0 - 53	16.42	11.25
BAI			
Anxiety	0 - 53	15.94	11.34

Note. Y-BOCS = Yale–Brown Obsessive-Compulsive Scale, a five-point scale ranging from 0 to 4; PCL-C = PTSD Checklist-Civilian Version, a five-point scale ranging from 1 to 5. BDI-II = Beck Depression Inventory-II, a four-point scale ranging from 0 to 3; BAI = Beck Anxiety Inventory, a four-point scale ranging from 0 to 3.

Table 3.2

Correlations between OCD Symptoms and PTSD Symptoms

Variable	ObsTime	ObsInt	ObsDis	ObsRes	ObsCont	CompTime	CompInt	CompDis	CompRes	CompCont	Intrusion	Dreams	Flash	Upset	PhysioR	AvoidTh	AvoidAct	Amnesia	LossInt	Distant	Numb	Future	Sleep	Anger	Concen	Hyper	Startle
ObsTime	-																										
ObsInt	0.62**	-																									
ObsDis	0.61**	0.69**	-																								
ObsRes	0.28**	0.33**	0.28**	-																							
ObsCont	0.49**	0.48**	0.54**	0.56**	-																						
CompTime	0.61**	0.53**	0.50**	0.34**	0.45**	-																					
CompInt	0.49**	0.71**	0.54**	0.32**	0.42**	0.70**	-																				
CompDis	0.50**	0.54**	0.59**	0.32**	0.49**	0.65**	0.67**	-																			
CompRes	0.23**	0.24**	0.24**	0.58**	0.41**	0.36**	0.34**	0.39**	-																		
CompCont	0.39**	0.39**	0.43**	0.44**	0.60**	0.58**	0.54**	0.63**	0.64**	-																	
Intrusion	0.13**	0.14**	0.17**	0.033	0.092	0.13**	0.092	0.12*	0.074	0.064	-																
Dreams	0.13**	0.09	0.12*	-0.063	0.036	0.094	0.069	0.087	0.017	0.067	0.57**	-															
Flash	0.13**	0.18**	0.17**	0.062	0.089	0.13**	0.12*	0.11*	0.063	0.089	0.66**	0.51**	-														
Upset	0.12*	0.11*	0.16**	0.061	0.078	0.10*	0.088	0.15**	0.11*	0.092	0.74**	0.47**	0.64**	-													
PhysioR	0.11*	0.13**	0.11*	0.060	0.081	0.14**	0.10*	0.14**	0.096	0.070	0.67**	0.42**	0.53**	0.71**	-												
AvoidTh	0.084	0.089	0.099	0.024	0.067	0.066	0.093	0.079	0.037	0.059	0.59**	0.39**	0.50**	0.72**	0.55**	-											
AvoidAct	0.11*	0.15**	0.13**	0.029	0.095	0.12**	0.13**	0.14**	0.050	0.085	0.60**	0.43**	0.57**	0.68**	0.54**	0.74**	-										
Amnesia	0.033	0.093	0.062	0.048	0.092	0.040	0.098	0.050	0.081	0.055	0.31**	0.24**	0.42**	0.39**	0.41**	0.40**	0.36**	-									
LossInt	0.062	0.11*	0.11*	0.060	0.098	0.060	0.077	0.050	0.093	0.040	0.63**	0.42**	0.52**	0.59**	0.54**	0.48**	0.52**	0.35**	-								
Distant	0.091	0.082	0.12**	0.041	0.051	0.050	0.044	0.072	0.046	0.040	0.64**	0.53**	0.51**	0.62**	0.53**	0.48**	0.53**	0.26**	0.79**	-							
Numb	0.073	0.073	0.054	0.0041	0.033	-0.016	0.019	0.052	0.023	0.046	0.49**	0.43**	0.43**	0.44**	0.34**	0.42**	0.44**	0.26**	0.54**	0.63**	-						
Future	0.13**	0.11*	0.11*	0.067	0.027	0.090	0.086	0.11*	0.074	0.060	0.56**	0.46**	0.56**	0.54**	0.44**	0.44**	0.44**	0.30**	0.63**	0.71**	0.57**	-					
Sleep	0.13*	0.13**	0.11*	0.0040	0.046	0.056	0.076	0.10*	0.089	0.099	0.55**	0.52**	0.48**	0.56**	0.49**	0.42**	0.45**	0.31**	0.62**	0.65**	0.59**	0.70**	-				
Anger	0.11*	0.11*	0.10*	-0.012	0.033	0.064	0.040	0.085	0.043	0.053	0.61**	0.54**	0.55**	0.58**	0.48**	0.54**	0.51**	0.29**	0.58**	0.65**	0.61**	0.68**	0.68**	-			
Concen	0.11*	0.13**	0.095	0.025	0.034	0.072	0.072	0.075	0.040	0.042	0.63**	0.53**	0.57**	0.57**	0.45**	0.45**	0.50**	0.33**	0.64**	0.69**	0.55**	0.68**	0.68**	0.74**	-		
Hyper	0.083	0.10*	0.10*	-0.006	0.030	0.081	0.068	0.016	0.023	0.017	0.61**	0.45**	0.56**	0.60**	0.49**	0.46**	0.53**	0.22**	0.52**	0.54**	0.46**	0.53**	0.57**	0.61**	0.57**	-	
Startle	0.15**	0.15**	0.16**	0.078	0.14**	0.12**	0.096	0.097	0.091	0.084	0.67**	0.52**	0.61**	0.66**	0.55**	0.53**	0.55**	0.31**	0.60**	0.62**	0.51**	0.63**	0.67**	0.69**	0.67**	0.80**	-

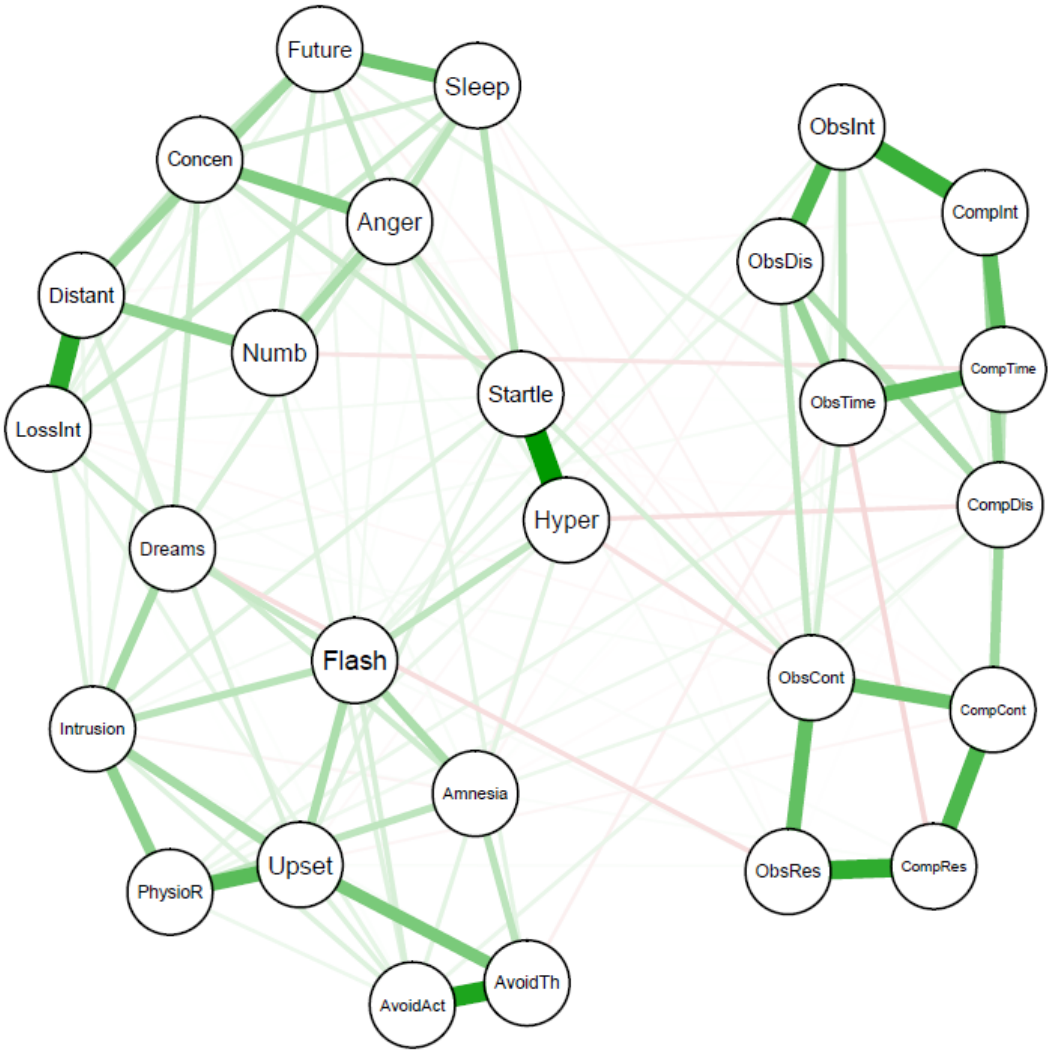
Note. *p < .05; **p < .01. Yale–Brown Obsessive-Compulsive Scale variables: ObsTime = time occupied by obsessions; ObsInt = interference caused by obsessions; ObsDis = distress caused by obsessions; ObsRes = difficulty resisting obsessions; ObsCont = difficulty controlling obsessions; CompTime = time occupied by compulsions; CompInt = interference caused by compulsions; CompDis = distress caused by compulsions; CompRes = difficulty resisting compulsions; and CompCont = difficulty controlling compulsions. PTSD Checklist-Civilian Version variables: Intrusion = intrusive memories, thoughts, or images of the SLE; Dreams = traumatic dreams; Flash = flashbacks; Upset = feeling upset in response to reminders of SLE; PhysioR = physiological reactivity to reminders of the SLE; AvoidTh = avoidance of thoughts or feelings about the SLE; AvoidAct = avoidance of activities or situations reminiscent of the SLE; Amnesia = having trouble remembering parts of the SLE; LossInt = loss of interest in previously enjoyed activities; Distant = feeling distant or cut off from people; Numb = feeling emotionally numb; Future = feeling that your future will be cut short; Sleep = difficulty falling or staying asleep; Anger = feeling irritable or having angry outbursts; Concen = difficulty concentrating; Hyper = hypervigilant, watchful or super alert; and Startle = feeling easily startled or jumpy.

Table 3.3

Correlations between OCD Symptoms and PTSD Clusters

Variable	ObsTime	ObsInt	ObsDis	ObsRes	ObsCont	CompTime	CompInt	CompDis	CompRes	CompCont	Re-exper	Avoid	Numb	Hyper	Depress	Anx
ObsTime	-															
ObsInt	0.62**	-														
ObsDis	0.61**	0.69**	-													
ObsRes	0.28**	0.33**	0.28**	-												
ObsCont	0.49**	0.48**	0.54**	0.56**	-											
CompTime	0.61**	0.53**	0.50**	0.34**	0.45**	-										
CompInt	0.49**	0.71**	0.54**	0.32**	0.42**	0.70**	-									
CompDis	0.50**	0.54**	0.59**	0.32**	0.49**	0.65**	0.67**	-								
CompRes	0.23**	0.24**	0.24**	0.58**	0.41**	0.36**	0.34**	0.39**	-							
CompCont	0.39**	0.39**	0.43**	0.44**	0.60**	0.58**	0.54**	0.63**	0.64**	-						
Re-exper	0.15**	0.15**	0.18**	0.044	0.092	0.14**	0.11*	0.15**	0.091	0.092	-					
Avoid	0.11*	0.13**	0.12**	0.029	0.086	0.099*	0.12*	0.12*	0.047	0.077	0.74**	-				
Numb	0.10*	0.12**	0.12**	0.058	0.076	0.060	0.082	0.085	0.082	0.061	0.75**	0.62**	-			
Hyper	0.14**	0.14**	0.13**	0.021	0.066	0.092	0.083	0.087	0.066	0.068	0.78**	0.62**	0.82**	-		
Depress	0.31**	0.34**	0.31**	0.066*	0.20**	0.23**	0.30**	0.24**	0.037	0.17**	0.32**	0.29**	0.36**	0.33**	-	
Anx	0.29**	0.33**	0.30**	0.064*	0.20**	0.23**	0.29**	0.25**	0.069*	0.15**	0.32**	0.26**	0.30**	0.29**	0.69**	-

Note. *$p < .05$; **$p < .01$. Yale–Brown Obsessive-Compulsive Scale variables: ObsTime = time occupied by obsessions; ObsInt = interference caused by obsessions; ObsDis = distress caused by obsessions; ObsRes = difficulty resisting obsessions; ObsCont = difficulty controlling obsessions; CompTime = time occupied by compulsions; CompInt = interference caused by compulsions; CompDis = distress caused by compulsions; CompRes = difficulty resisting compulsions; and CompCont = difficulty controlling compulsions. PTSD Checklist-Civilian Version symptom clusters: Re-exper = re-experiencing; Avoid = avoidant; Numb = numbing; and Hyper = hyperarousal. Depress = Beck Depression Inventory-II total score; Anx = Beck Anxiety Inventory total score.



OCD Symptoms
ObsTime = time occupied by obsessions
ObsInt = interference caused by obsessions
ObsDis = distress caused by obsessions
ObsRes = difficulty resisting obsessions
ObsCont = difficulty controlling obsessions
CompTime = time occupied by compulsions
CompInt = interference caused by compulsions
CompDis = distress caused by compulsions
CompRes = difficulty resisting compulsions
CompCont = difficulty controlling compulsions

PTSD Symptoms
Intrusion = intrusive memories/thoughts/images
Dreams = traumatic dreams
Flash = flashbacks
Upset = feeling upset
PhysioR = physiological reactivity
AvoidTh = avoidance of thoughts/feelings
AvoidAct = avoidance of activities/situations
Amnesia = having trouble remembering
LossInt = loss of interest in activities
Distant = feeling distant from others
Numb = feeling emotionally numb
Future = feeling that your future will be cut short
Sleep = difficulty falling or staying asleep
Anger = feeling irritable or having angry outbursts
Concen = difficulty concentrating
Hyper = hypervigilant, watchful or super alert
Startle = feeling easily startled or jumpy

Figure 3.1. Estimated regularized network of OCD symptoms and PTSD symptoms.

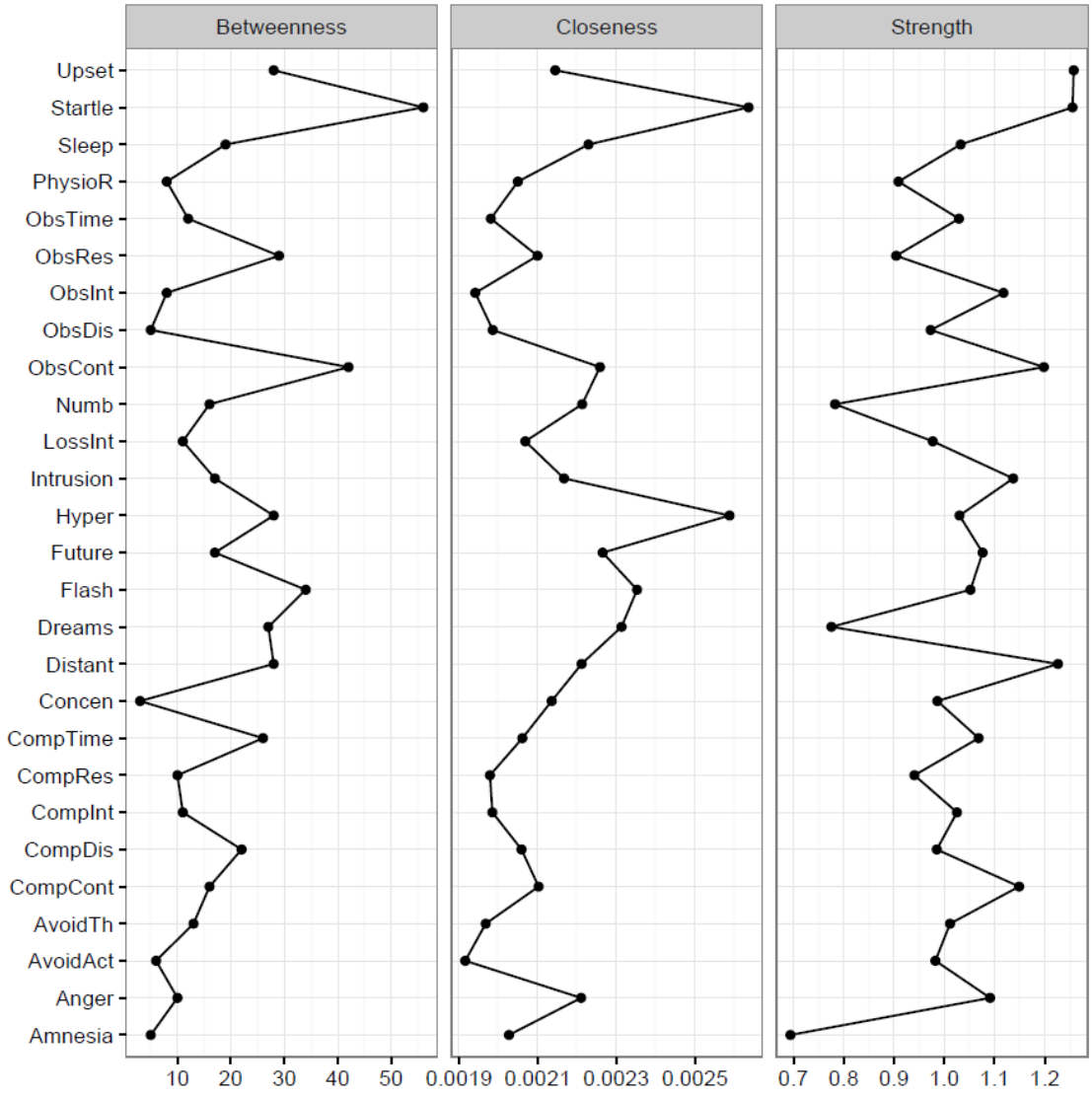


Figure 3.2. Centrality plot, including betweenness, closeness, and strength, for network of OCD symptoms and PTSD symptoms shown in Figure 3.1.

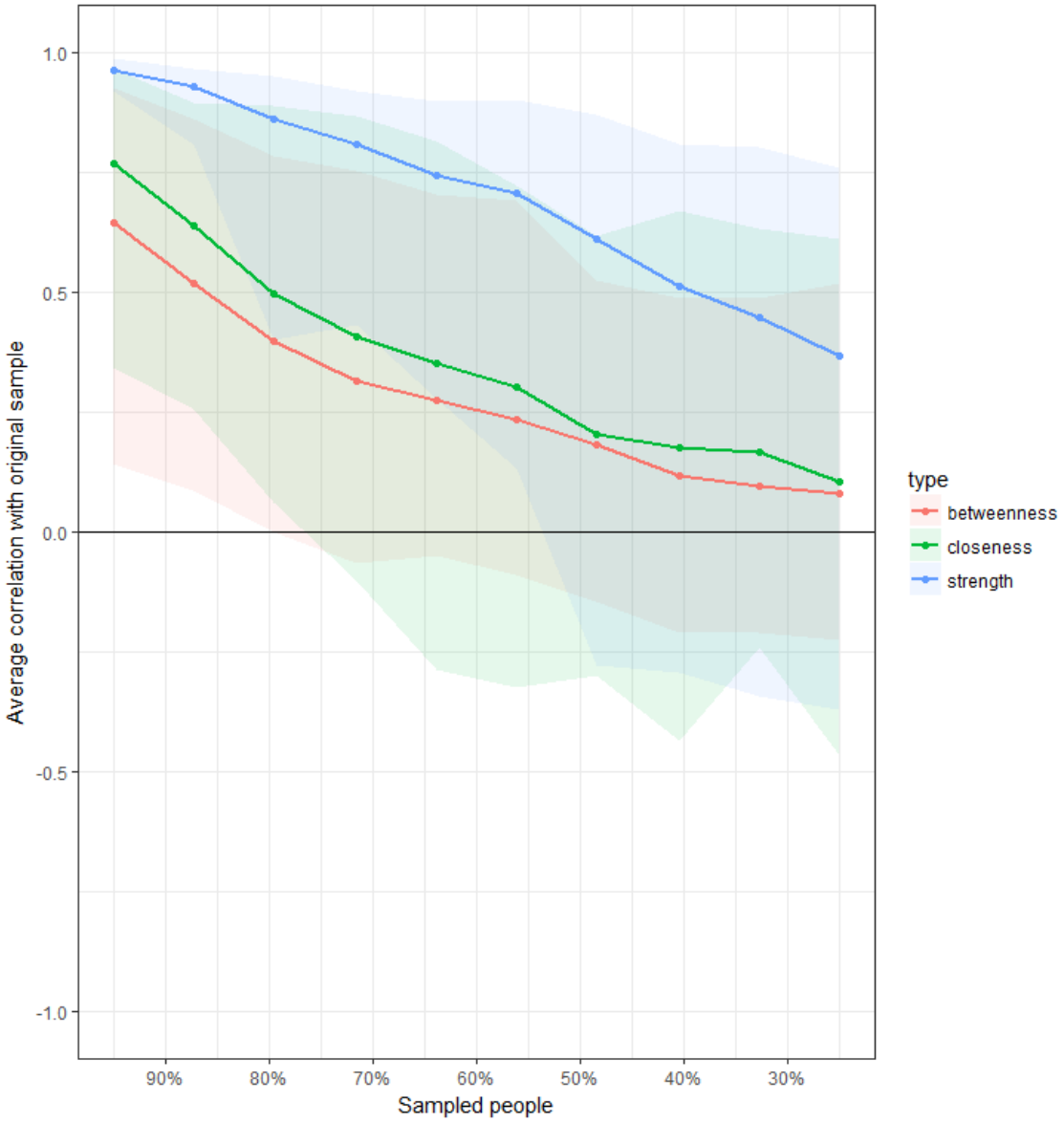


Figure 3.3. Dropped samples plot for bootstrapped results of network shown in Figure 3.1.

Table 3.4

Significant Edge Weights and Confidence Intervals of Initial OCD/PTSD Network shown in Figure 3.1

Nodes	Sample Edge Weight	Lower CI (2.5%)	Upper CI (97.5%)
Hyper--Startle	0.55	0.45	0.68
AvoidTh--AvoidAct	0.48	0.36	0.65
LossInt--Distant	0.45	0.32	0.60
ObsRes--CompRes	0.45	0.34	0.61
ObsInt--CompInt	0.42	0.38	0.62
CompRes--CompCont	0.38	0.30	0.57
ObsInt--ObsDis	0.38	0.27	0.54
Upset--PhysioR	0.36	0.14	0.53
ObsTime--CompTime	0.35	0.31	0.57
CompTime--CompInt	0.34	0.28	0.53
ObsRes--ObsCont	0.34	0.15	0.48
ObsCont--CompCont	0.32	0.19	0.49
Future--Sleep	0.30	0.15	0.46
Upset--AvoidTh	0.28	0.19	0.47
Anger--Concen	0.27	0.10	0.44
Distant--Numb	0.24	0.034	0.43
Intrusion--PhysioR	0.24	0.098	0.48
ObsTime--ObsDis	0.22	0.071	0.37
CompInt--CompDis	0.22	0.054	0.35
CompDis--CompCont	0.21	0.093	0.36
Distant--Future	0.21	0.036	0.39
ObsDis--CompDis	0.18	0.044	0.33
Flash--Upset	0.18	0.045	0.41
Flash--Amnesia	0.18	0.045	0.44
ObsTime--ObsInt	0.16	0.083	0.38
Sleep--Startle	0.15	0.0078	0.31
PhysioR--Amnesia	0.14	0.014	0.45
CompTime--CompCont	0.13	0.046	0.33
CompDis--Hyper	-0.067	-0.31	-0.026
ObsRes--Dreams	-0.071	-0.39	0.0022

Note. Yale–Brown Obsessive-Compulsive Scale variables: ObsTime = time occupied by obsessions; ObsInt = interference caused by obsessions; ObsDis = distress caused by obsessions; ObsRes = difficulty resisting obsessions; ObsCont = difficulty controlling obsessions; CompTime = time occupied by compulsions; CompInt = interference caused by compulsions; CompDis = distress caused by compulsions; CompRes = difficulty resisting compulsions; and CompCont = difficulty controlling compulsions. PTSD Checklist-Civilian Version variables: Intrusion = intrusive memories, thoughts, or images of the SLE; Dreams = traumatic dreams; Flash = flashbacks; Upset = feeling upset in response to reminders of SLE; PhysioR = physiological reactivity to reminders of the SLE; AvoidTh = avoidance of thoughts or feelings about the SLE; AvoidAct = avoidance of activities or situations reminiscent of the SLE; Amnesia = having trouble remembering parts of the SLE; LossInt = loss of interest in previously enjoyed activities; Distant = feeling distant or cut off from people; Numb = feeling emotionally numb; Future = feeling that your future will be cut short; Sleep = difficulty falling or staying asleep; Anger = feeling irritable or having angry outbursts; Concen = difficulty concentrating; Hyper = hypervigilant, watchful or super alert; and Startle = feeling easily startled or jumpy.

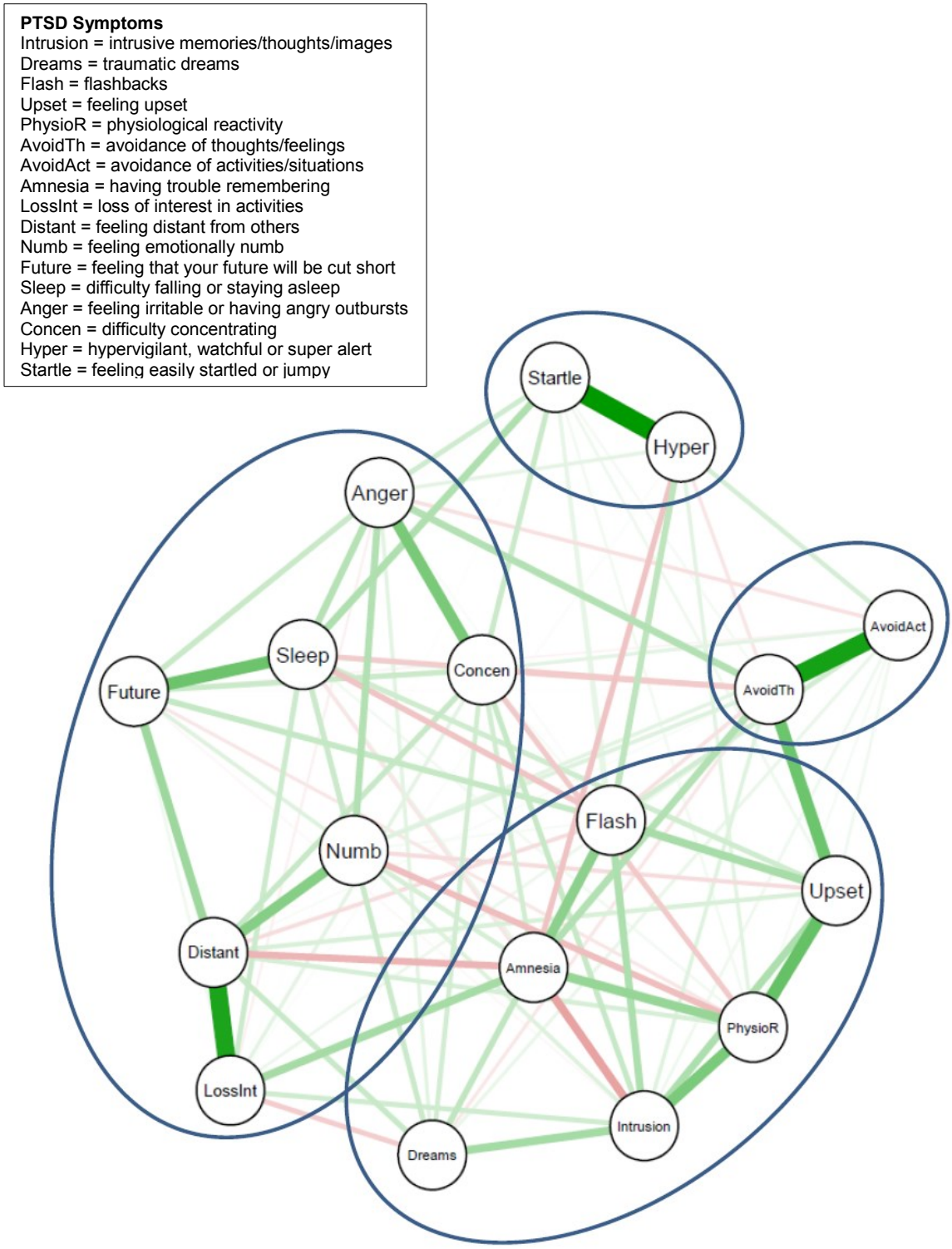


Figure 3.4. Estimated regularized network of PTSD symptoms. Blue circles indicate communities of symptoms as detected by the spinglass algorithm.

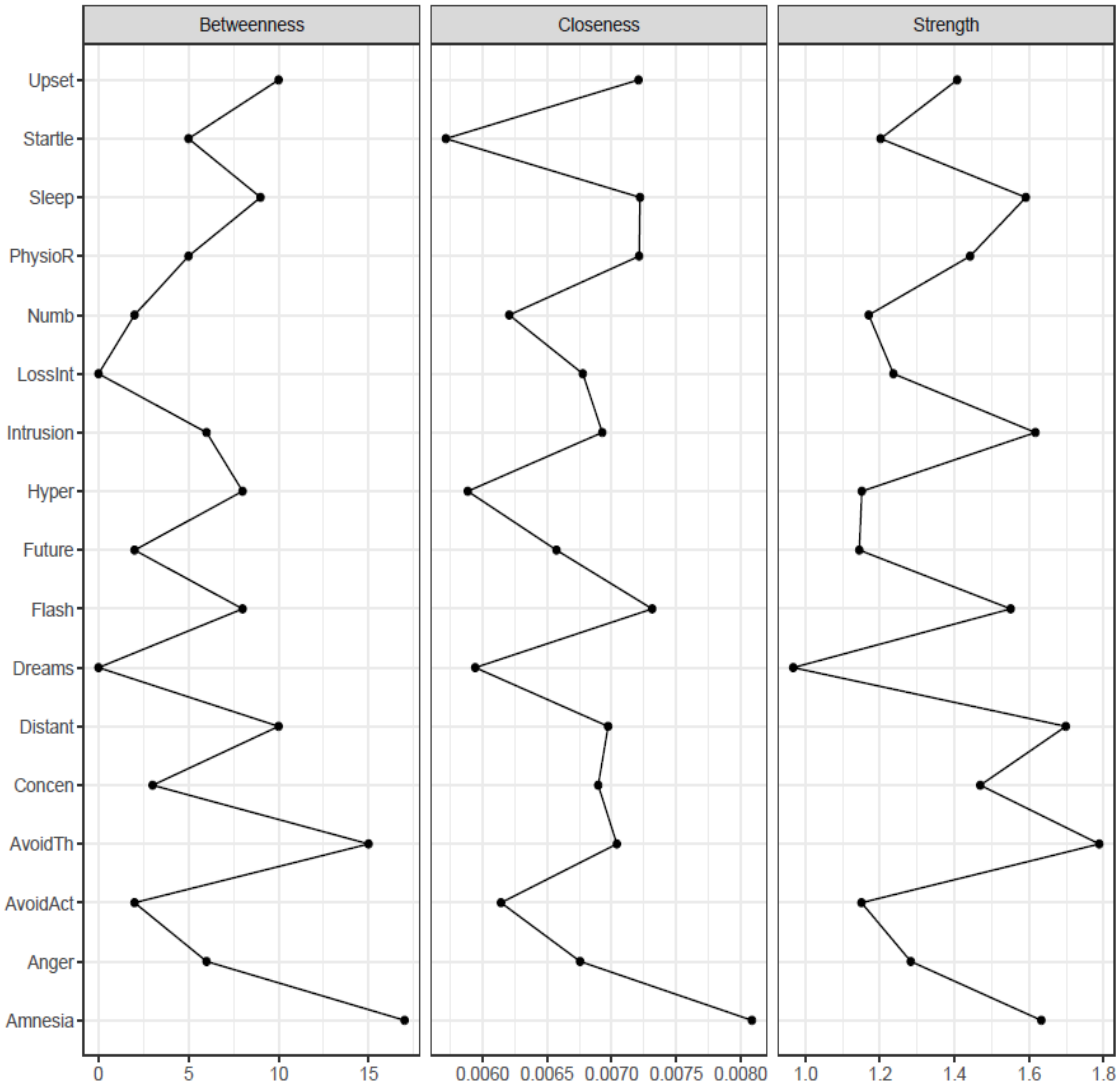


Figure 3.5. Centrality plot, including betweenness, closeness, and strength, for network of PTSD symptoms shown in Figure 3.4.

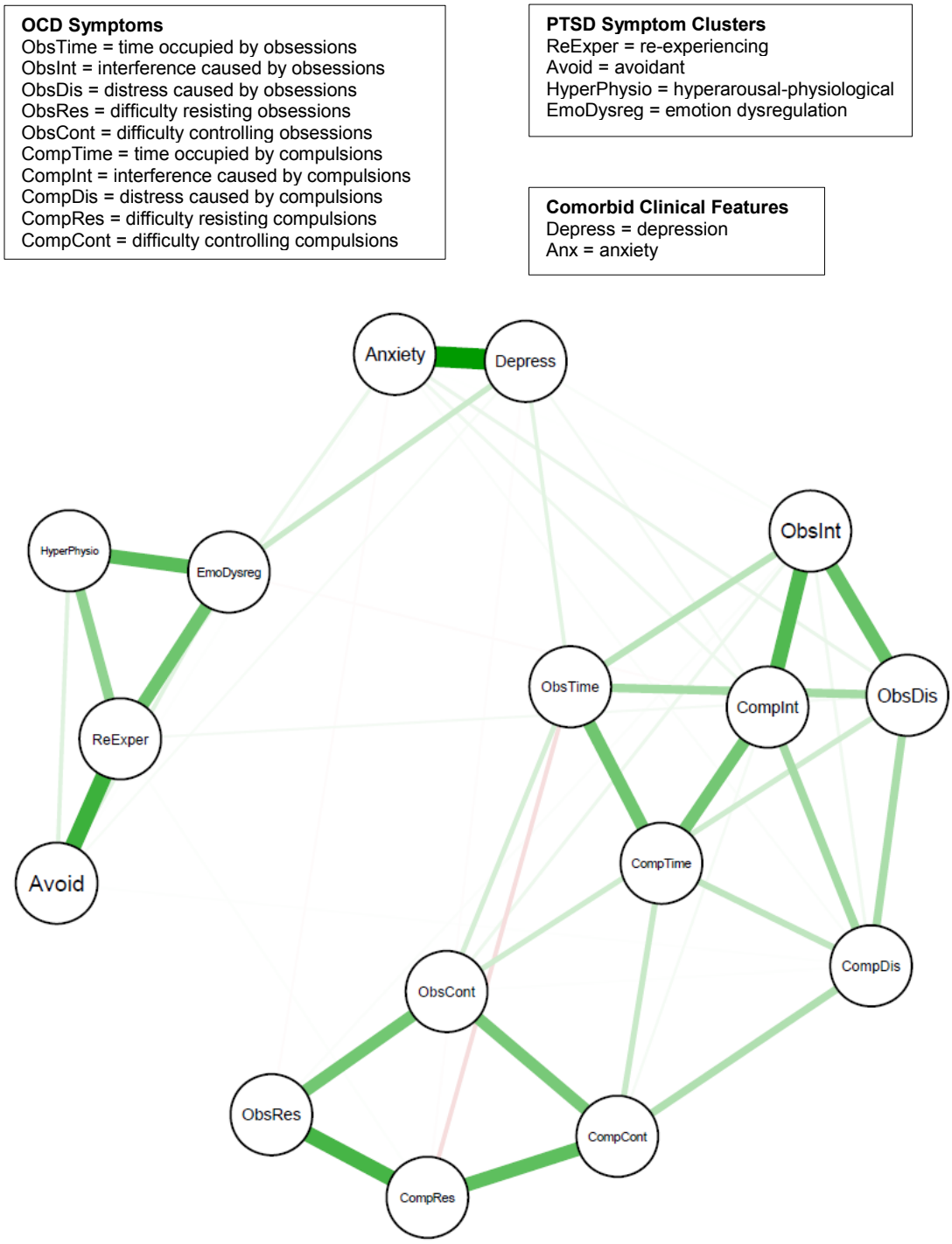


Figure 3.6. Adjusted estimated regularized network of OCD symptoms, PTSD clusters, depression, and anxiety.

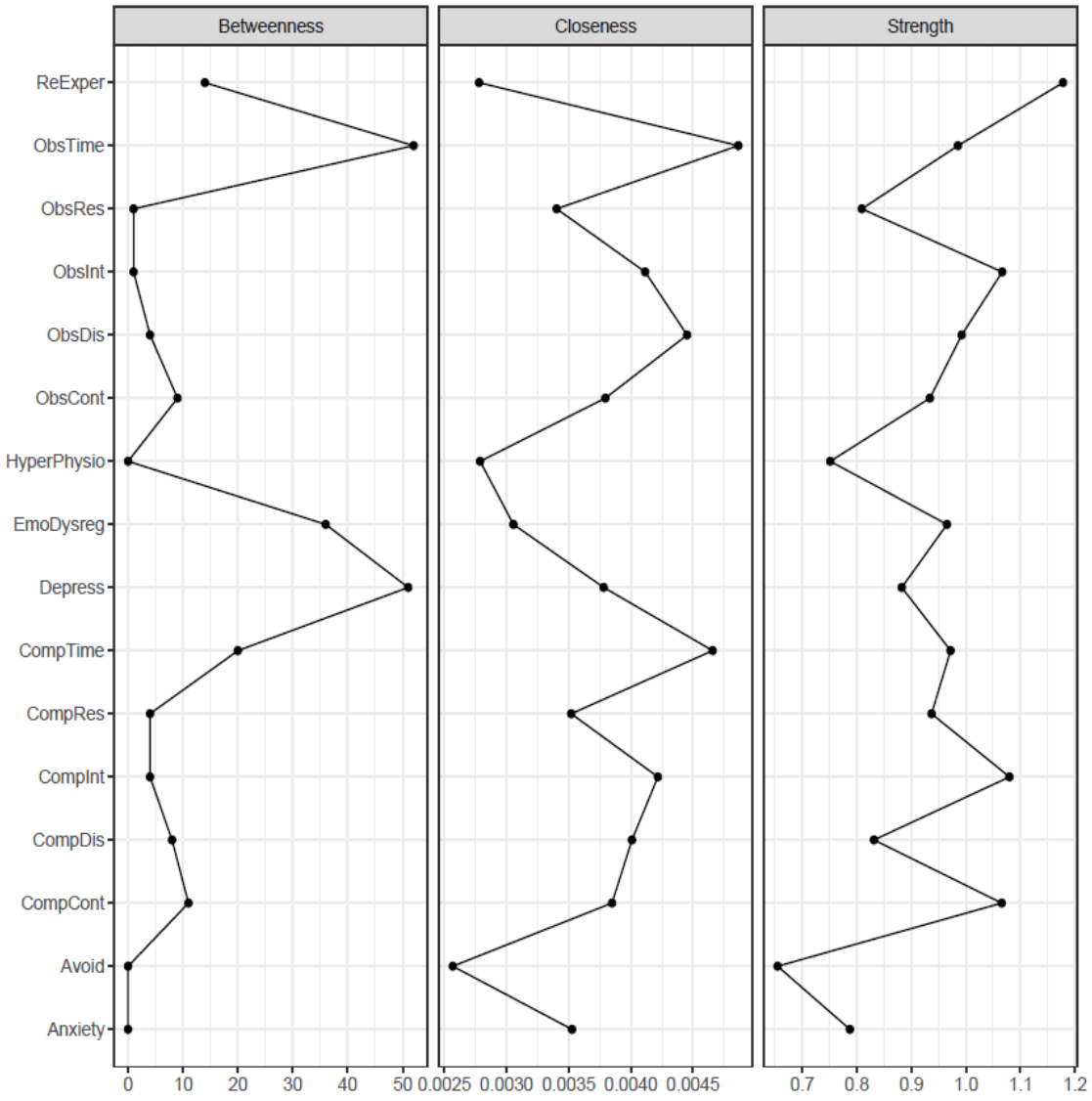


Figure 3.7. Centrality plot, including betweenness, closeness, and strength, for network of OCD symptoms, PTSD clusters, depression, and anxiety shown in Figure 3.6.

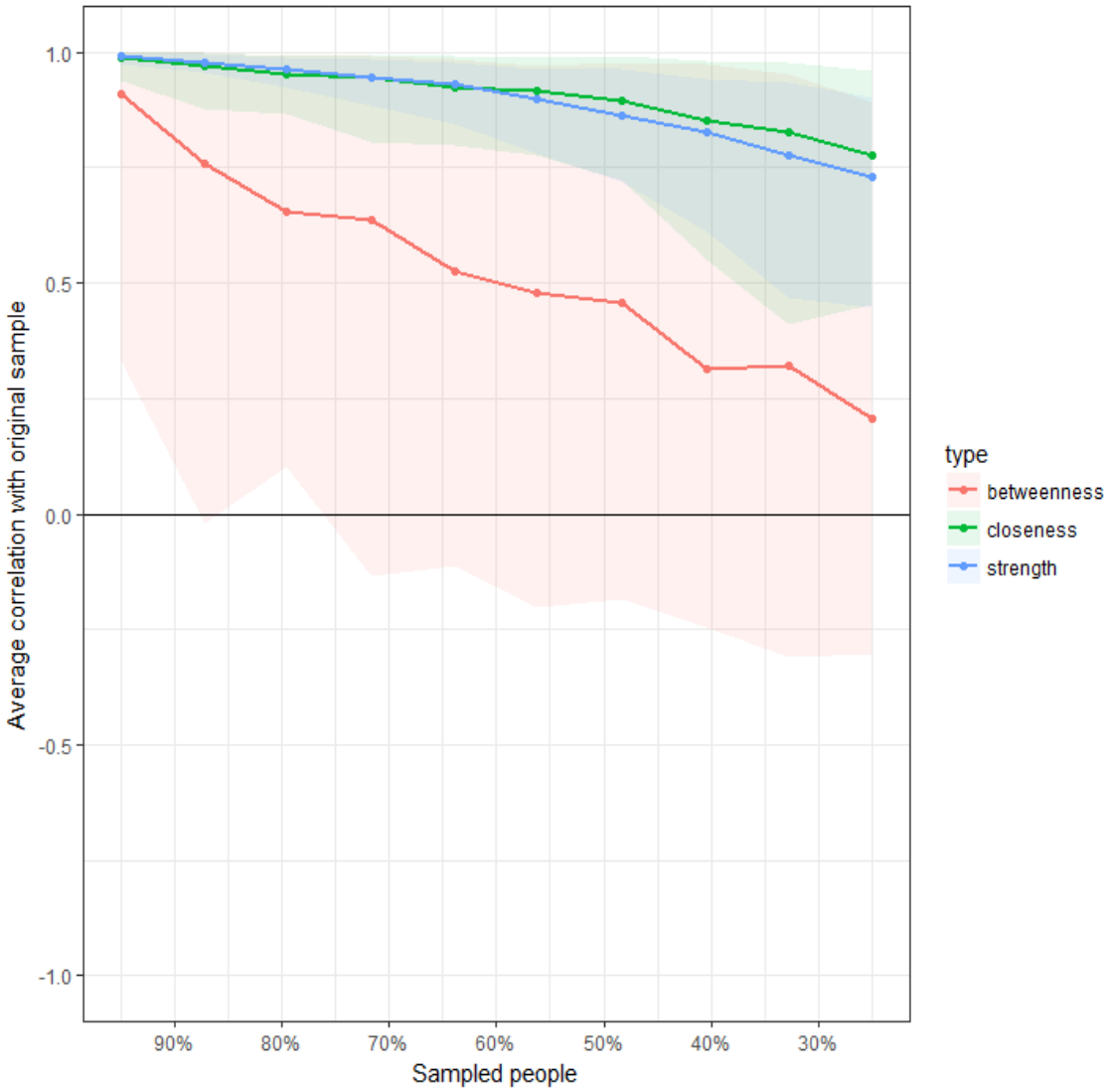


Figure 3.8. Dropped samples plot for bootstrapped results of adjusted network shown in Figure 3.6.

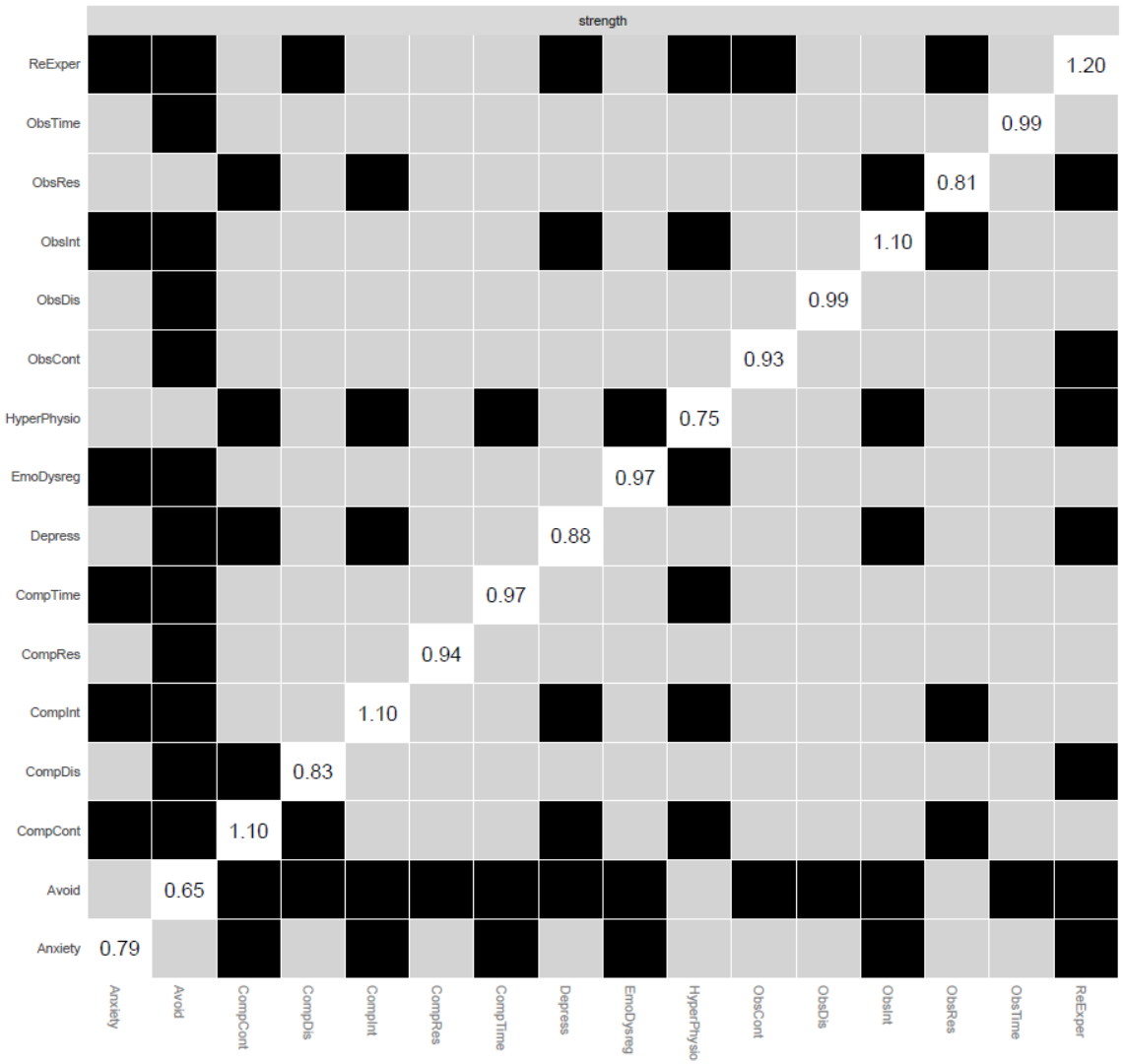


Figure 3.9. Graphical depiction of bootstrapped strength centrality results of adjusted network shown in Figure 3.6. Gray boxes indicate nodes that do not differ significantly from one another; black boxes represent nodes that do differ significantly from one another.

Table 3.5

Significant Edge Weights and Confidence Intervals of Adjusted OCD/PTSD Network shown in Figure 3.6

Edges	Sample Edge Weight	Lower CI (2.5%)	Upper CI (97.5%)
Depress--Anxiety	0.62	0.51	0.70
Avoid--ReExper	0.47	0.35	0.57
ObsRes--CompRes	0.45	0.33	0.55
ObsInt--CompInt	0.42	0.31	0.53
HyperPhysio--EmoDysreg	0.40	0.28	0.50
CompRes--CompCont	0.39	0.29	0.49
ObsInt--ObsDis	0.37	0.27	0.45
ReExper--EmoDysreg	0.35	0.25	0.45
ObsTime--CompTime	0.35	0.25	0.45
ObsRes--ObsCont	0.34	0.21	0.43
CompTime--CompInt	0.34	0.24	0.44
ObsCont--CompCont	0.33	0.20	0.44
HyperPhysio--ReExper	0.27	0.14	0.37
ObsTime--ObsDis	0.22	0.11	0.34
CompInt--CompDis	0.22	0.09	0.32
ObsDis--CompDis	0.21	0.09	0.33
CompDis--CompCont	0.19	0.09	0.29
ObsTime--ObsInt	0.17	0.06	0.28
CompTime--CompDis	0.16	0.02	0.27
CompTime--CompCont	0.13	0.03	0.21
EmoDysreg--Depress	0.13	0.06	0.19
ObsDis--ObsCont	0.12	0.011	0.22

Note. Yale–Brown Obsessive-Compulsive Scale variables: ObsTime = time occupied by obsessions; ObsInt = interference caused by obsessions; ObsDis = distress caused by obsessions; ObsRes = difficulty resisting obsessions; ObsCont = difficulty controlling obsessions; CompTime = time occupied by compulsions; CompInt = interference caused by compulsions; CompDis = distress caused by compulsions; CompRes = difficulty resisting compulsions; and CompCont = difficulty controlling compulsions. PTSD Checklist-Civilian Version symptom clusters: ReExper = re-experiencing; Avoid = avoidant; HyperPhysio = hyperarousal-physiological; and EmoDysreg = emotion dysregulation. Depress = Beck Depression Inventory-II total score; Anxiety = Beck Anxiety Inventory total score.

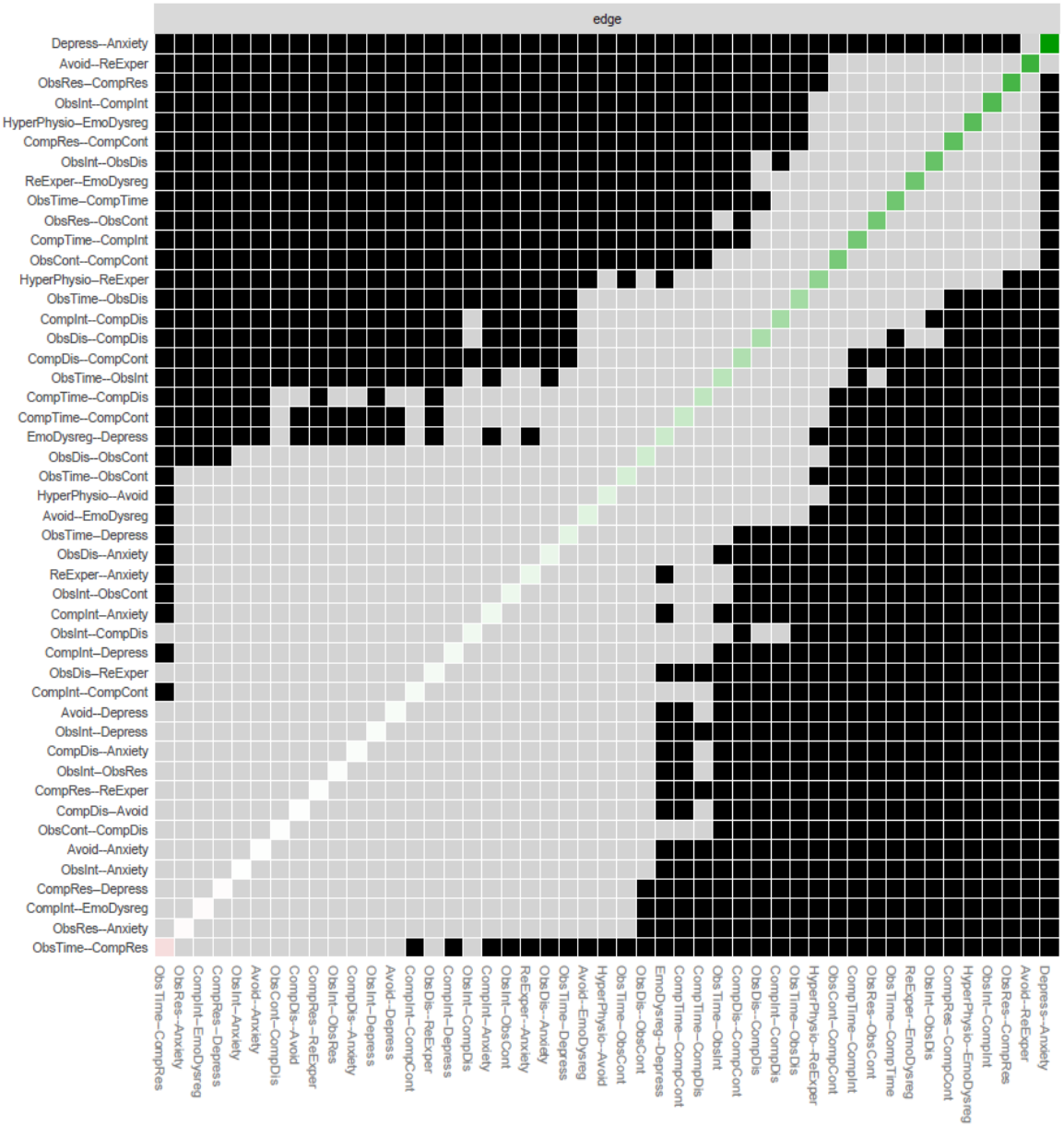


Figure 3.10. Graphical depiction of bootstrapped edge weight results of adjusted network shown in Figure 3.6. Gray boxes indicate edges that do not differ significantly from one another; black boxes represent edges that do differ significantly from one another.