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# Illness Pathways between Eating Disorder and Post Traumatic Stress Disorder Symptoms: Understanding Comorbidity with Network Analysis.

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## Abstract

Eating disorders (ED) and post-traumatic stress disorder (PTSD) are highly comorbid. However, specific mechanisms by which PTSD-ED comorbidity is maintained are unknown. The current study constructed two PTSD-ED comorbidity networks (25 ED and 17 PTSD symptoms) in two samples: a clinical ($N$ = 158 individuals with an ED diagnosis) and a non-clinical sample ($N$ = 300 college students). Glasso networks were constructed to identify (a) pathways between disorders (bridge symptoms) and (b) core symptoms. Three illness pathways emerged: between *binge eating* and *irritability*, between *desire for a flat stomach* and *disturbing dreams*, and between *concentration problems* and *weight and shape-related concentration problems*. Our findings suggest that pathways between binge eating and irritability, body dissatisfaction and trauma reminders, and concentration difficulties may be the mechanisms by which comorbidity is maintained. Interventions disrupting these pathways and targeting core and bridge symptoms may be more efficient than traditional treatment approaches.

### Keywords

Eating disorders; posttraumatic stress disorder; comorbidity; network analysis

Post-traumatic stress disorder (PTSD) is one of the most frequently comorbid disorders with eating disorders (EDs; Brewerton, 2007; Hudson, Hiripi, Pope, Kessler, & Kessler, 2007; Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012; Swinbourne & Touyz, 2007; Tagay, Schlottbohm, Reyes-Rodriguez, Repic, & Senf, 2014), with comorbidity estimated to range between 4% and 62% (Brewerton, 2007; Swinbourne & Touyz, 2007; Tagay et al., 2014). Furthermore, in a national comorbidity survey, 90–100 % of individuals with all types of ED diagnoses reported having experienced a traumatic event (e.g., witnessing a car accident or someone being injured, rape, life-threatening illness, or natural disaster; Mitchell et al., 2012). To date, research on comorbidity between PTSD and EDs has focused on identifying how traumatic events may contribute to the development of both disorders. Less research has focused on how these disorders might maintain each other. Given these high

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rates of comorbidity (Brewerton, 2007; Swinbourne & Touyz, 2007; Tagay et al., 2014), it is necessary to examine how symptoms of PTSD and EDs might maintain and exacerbate each other, which could inform treatment.

Previous research suggests that traumatic events precede the development of EDs (Cachelin, Schug, Juarez, & Monreal, 2005; Collins, Fischer, Stojek, & Becker, 2014; Pike et al., 2006; Tagay et al., 2014). However, it has been suggested that PTSD symptoms rather than trauma history itself are associated with ED psychopathology (Holzer, Uppala, Wonderlich, Crosby, & Simonich, 2008). It is still unclear, though, whether PTSD symptoms maintain EDs or vice versa. Trottier and colleagues (2016) proposed that PTSD symptoms serve as a maintaining factor for ED symptoms. ED behaviors, such as binge eating, purging, and restriction may facilitate escape and avoidance of distressing memories, thoughts, and feelings of PTSD (Mitchell, Porter, Boyko, & Field, 2016; Trottier & MacDonald, 2017; Trottier et al., 2016). Although avoidance provides short-term relief, it prevents an individual from addressing the distressing symptoms and maintains the disorder long-term. Thus, it may be that instead of PTSD causing EDs or vice versa, specific symptoms of each disorder interact to maintain and exacerbate comorbidity.

Some pathways between specific symptoms of PTSD and EDs have been examined. Researchers found that having a traumatic experience is more often associated with binge eating and purging than other ED symptoms (Brewerton, 2007; Tagay et al., 2014). Brewerton (2007) notes that abuse history, including physical and sexual abuse, is associated with increased body dissatisfaction, suggestive of one pathway linking trauma and risk of developing an ED. Supportive of this potential link, Tagay and colleagues (2014) found in a sample of individuals with AN and BN that those with higher levels of PTSD symptoms (compared to those with lower PTSD) scored highest on body dissatisfaction. This relationship may be explained by the development of a self-critical view of self, which then drives individuals to use ED behaviors to achieve an ideal body image (Dunkley, Masheb, & Grilo, 2010). It has also been proposed that deficits in emotion regulation and impulsivity resulting from a traumatic experience may maintain ED symptoms (Mitchell et al., 2012; Trottier & MacDonald, 2017). This literature suggests that binge eating, purging, fasting, body dissatisfaction, self-criticism, and emotion dysregulation may be implicated as illness pathways connecting ED and PTSD.

## Network Analysis and Psychological Comorbidity

Network analysis has opened new possibilities for conceptualizing mental disorders in general and comorbidity in particular (Borsboom & Cramer, 2013; Cramer, Waldorp, van der Maas, & Borsboom, 2010; McNally, 2016; for a review of recent literature on the use of network analysis in psychopathology see Fried et al., 2017). From a network theory perspective, comorbidity occurs because there is a direct relationship between the symptoms of multiple disorders. The comorbidity network is represented by two disorder-specific networks in which symptoms are closely connected to one another, with some symptoms being connected or “bridged” across disorders (Cramer et al., 2010). These bridge symptoms can be conceptualized as illness pathways that causally connect symptoms of two disorders and therefore may maintain the comorbidity (Levinson et al., 2017). For example, if there is

a strong connection between flashbacks (PTSD) and binge eating (ED), experiencing many flashbacks may lead to increased binge eating or vice versa.

Another parameter that may be helpful in understanding psychological comorbidity using network analysis is node centrality. The symptoms with the highest centrality (core symptoms) in a combined network that consists of symptoms of both PTSD and ED may have an impact on all symptoms in the network. A core symptom exhibits a large number of connections in a network, and according to network theory, switching on this symptom will likely spread symptom activation throughout the network (Borsboom & Cramer, 2013; McNally, 2016), such as removing a central card in a house of cards, causing the entire deck to collapse. Even though a core ED symptom is likely to be most strongly connected to other ED symptoms and would affect them first, it is also likely to impact a bridge symptom. A change in that bridge symptom might deactivate the connected PTSD symptoms, dismantling the comorbidity network.

## Network Comparison

Additionally, advances in network analysis can be utilized to test if networks of symptoms differ across populations (van Borkulo et al., 2015). The Network Comparison Test (NCT) can be used to compare whether two or more networks are different in network structure (i.e., if the way the nodes within the network are connected differs across samples) and/or different in global strength (i.e., if the sum of the strengths of all edges in the network differs across samples; van Borkulo et al., 2015). If a clinical and a non-clinical network do not differ in structure, this finding would suggest that symptoms interact with each other in a similar way, regardless of their severity. A greater density in a network would reflect that the symptoms are more strongly related and have potentially greater impact on one another (versus a less dense network).

The NCT has been used to determine if network density can predict treatment response, and results are conflicting (Schweren, van Borkulo, Fried, & Goodyer, 2017; van Borkulo et al., 2015). Van Borkulo et al. (2015) found that networks of patients with major depression who did not respond to treatment had higher global strength than the group that responded to treatment. However, Schweren et al. (2017) found no differences between groups. A recent study by Heeren and McNally (2018) found greater network connectivity in a sample of individuals with social anxiety disorder compared to a non-clinical sample. We hypothesize that the population with the more strongly connected network exhibits more densely connected symptoms. Such a finding has implications for understanding how psychopathology symptoms interact with each other in ill versus non-ill populations and would suggest that treatments are needed to loosen connections between symptoms.

## Application of Network Theory to PTSD and EDs

Network analysis has recently been applied to the conceptualization of both PTSD and EDs separately. There have been five studies using network analysis to examine PTSD across different types of samples (e.g., veterans, traumatic injury; Armour, Fried, Deserno, Tsai, & Pietrzak, 2017; Bryant et al., 2017; Fried et al., 2018; McNally et al., 2015; Mitchell et al.,

2017). Overall, though there are slight differences in the core symptoms identified, most studies have found that intrusive cognitions, distressing dreams, physiological reactivity, and concentration difficulties were central to PTSD psychopathology networks.

With regard to EDs, Levinson and colleagues (2017) conducted a network analysis of bulimia nervosa (BN) symptoms in a sample of individuals diagnosed with BN and found that fear of weight gain, desire to lose weight, preoccupation with weight, and over-evaluation of weight were core BN symptoms. These researchers suggest that weight-related fears and thoughts serve primary roles in the maintenance of BN (Levinson et al., 2017). In another application of network analysis in the examination of EDs, Forbush, Siew, & Vitevitch, (2016) found that body checking served as a core symptom of ED psychopathology networks. Differences in the findings may have resulted from differences in the methodology for network construction. DuBois, Rodgers, Franko, Eddy, and Thomas (2017) found that overvaluation of weight and shape was the most central symptom across all diagnostic groups (AN, BN, and BED). Finally, a study by Olatunji, Levinson, and Calebs (2018) found that interoceptive awareness (i.e., awareness of physical sensations) and ineffectiveness (i.e., feeling flawed), were central to the ED network at both admission and discharge from inpatient treatment. The different findings may be explained by Olatunji and colleagues (2018) using the 11 subscales of the Eating Disorder Inventory-2 (Garner, Olmstead, & Polivy, 1983). None of the other three studies included symptoms such as interoceptive awareness or ineffectiveness. Overall, these studies show how network analysis can be used to identify central symptoms of PTSD and EDs separately, but they do not address the high comorbidity between disorders.

## Present Study

Despite these important advances in the understanding of PTSD and ED psychopathology networks individually, network analysis has not been applied to the study of comorbid PTSD and ED symptoms, nor used to test differences between clinical and non-clinical samples. The primary aim of the present study was to identify the bridge symptoms (or illness pathways) that connect PTSD and EDs and core symptoms in the comorbid network, thereby enhancing our understanding of what might maintain this type of comorbidity. Our secondary aim was to test if networks would differ for clinical vs non-clinical samples to examine if network connectivity is associated with symptom severity. The results of the second aim intend to add to the recent literature about whether stronger network connectivity is related to stronger symptom severity, which can be used to inform treatment outcome research.

Based on previous research (Brewerton, 2007; Tagay et al., 2014), body dissatisfaction (e.g., shape and weight dissatisfaction) and bulimic symptoms were hypothesized to serve as bridge symptoms between PTSD and other ED symptoms. We hypothesized that the core symptoms of the comorbid PTSD-ED networks would include weight-related fears, intrusion, and concentration-related symptoms. We are building on previous literature focused on binge-purge ED symptoms by examining ED symptoms in a primarily AN sample. Following previous research (van Borkulo et al., 2015), it was also hypothesized that greater symptom severity would be associated with greater network density as measured by

global strength, with the clinical sample network having both greater levels of PTSD and ED symptoms and stronger relations between symptoms than the non-clinical sample network. We hypothesized that the networks would not differ in structure.

## Methods

### Participants

The clinical sample consisted of 158 individuals recently discharged from a residential and/or partial hospitalization eating disorder facility in the Midwestern United States. Individuals who gave permission to be contacted for research opportunities at discharge were contacted by email about participating in the study. Those who agreed to participate filled out measures online and were compensated for their time. All participants in the clinical sample met criteria for a diagnosis of ED at the time of data collection. ED diagnoses were determined using Eating Disorder Diagnostic Scale (see below: Stice, Telch, & Rizvi, 2000). A majority of the sample (84.8%) had a diagnosis of AN or atypical AN. Forty six percent of participants endorsed having had a binge episode in the past month, 23% endorsed vomiting, 13% endorsed taking laxatives, and 45% endorsed compensatory exercise. Please see Table 1 for more information on diagnoses. PTSD diagnoses were determined using the cut-off score on PTSD Checklist – Civilian Version (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). The National Center for PTSD (n.d.) suggests different cut-off scores for determining PTSD diagnosis for different types of clinical settings. Considering the controversy about thresholds of PTSD diagnosis and their clinical utility (Mitchell et al., 2012; Palm, Strong, & MacPherson, 2009), we report prevalence rates for both PCL-C cut-off scores of 44 and 35 (National Center for PTSD, n.d.). A total score above 44 is considered indicative of PTSD diagnosis in specialized clinics and VA primary care (Blanchard et al., 1996) and a score above 35 is considered indicative of PTSD in general populations and primary care samples (National Center for PTSD, n.d.). Seventy percent of participants in the clinical sample had scores above the threshold of 35 and 51% were above the threshold of 44. These prevalence rates are consistent with those reported in other literature (Mitchell et al., 2012; Tagay et al., 2014). No other any inclusion or exclusion criteria were implemented.

The non-clinical sample consisted of 300 female undergraduate students at a university in the Midwestern United States. Students completed measures online to receive class credit for their participation. Individuals in the non-clinical sample did not complete a diagnostic interview. See Table 1 for demographics and scores on the Eating Disorder Examination Questionnaire - IV (EDE-Q-IV; Fairburn and Beglin, 1994) and the PCL-C (Blanchard et al., 1996) for both samples.

All procedures were approved by the Washington University Institutional Review Board. Informed consent was obtained from all individual participants included in the study.

## Measures

### Eating Disorder Diagnostic Scale (EDDS; Stice, Telch, & Rizvi, 2000).

The EDDS was used to determine diagnosis of the participants in the clinical sample. The EDDS is a 22-item self-report measure used to diagnose EDs, such as anorexia nervosa, bulimia nervosa, and binge eating disorder. The EDDS includes items scored on a likert scale, yes or no questions, and open-ended questions. The EDDS has adequate test-retest reliability, internal consistency, and validity (Stice et al., 2000; Stice, Fisher, & Martinez, 2004). In the current study, internal consistency was good (α = .93). Example items include: *Has your weight or shape influenced how you judge yourself as a person?* And *During the past 3 months have there been times when you have eaten what other people would regard as an unusually large amount of food (e.g., a pint of ice cream) given the circumstances?*

### PCL-C.

The PTSD Checklist – Civilian Version (PCL-C) is a 17-item self-report measure of PTSD symptoms (Blanchard et al., 1996). Each item corresponds to a symptom of PTSD (e.g., "*Repeated, disturbing memories, thoughts, or images of a stressful experience from the past?*") as defined by the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV; American Psychiatric Association, 1994). The PCL-C uses a 5-point Likert scale to assess how much a participant has been bothered by each symptom over the past month, ranging from 1 = Not at all to 5 = Extremely. Cronbach's α for the PCL-C in the clinical and non-clinical sample was .93 and .88 respectively.

### EDE-Q-IV.

The Eating Disorder Examination Questionnaire – IV (EDE-Q-IV) is a 41-item self-report measure of disordered eating (Fairburn & Beglin, 1994). Questions correspond to symptoms of EDs (e.g., "*Have you attempted to avoid eating any foods which you like in order to influence your shape or weight?*") and participants are asked how frequently they engage in the behaviors. The EDE-Q-IV uses open response and a 7-point Likert scale to assess the frequency of disordered eating behaviors over the past 28 days. For the present network analysis, questions that contain an open response were excluded to mirror the Likert scale structure of the PCL-C and because network analysis does not allow for open-ended responses. A total of 25 items that comprise EDE-Q-IV Global score were included. Cronbach's α for the EDE-Q in the clinical and non-clinical sample was .90 and .94 respectively.

### Data Analytic Procedure

**Glasso Networks.—**Two comorbid PTSD-ED psychopathology networks were constructed: one for the clinical ED treatment sample ($n$ = 158), and one for the non-clinical undergraduate sample ($n$ = 300). Network analysis was conducted in *R* (Version 3.3.2). Multiple imputation was conducted using Amelia II (Honaker, King, & Blackwell, 2011) in order to account for missing data. The clinical sample contained 8.7% missing data, and the non-clinical sample had 0.3% missing data. The networks were estimated using the *glasso* function in the *qgraph* package (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom,

2012) and script from Epskamp (2014). The indices of centrality were calculated using the *centralityPlot* and *centralityTable* functions in qgraph (Epskamp et al., 2012). Three commonly used indices of centrality for each network were calculated: betweenness, closeness, and strength (McNally, 2016).

**Bridge Symptoms.—**We used the *bridge* function of the *networktools* package (Jones, 2017) to identify bridge symptoms between PTSD and ED in each network. By calculating bridge strength, we identified which ED symptom was most strongly connected to all PTSD symptoms, and vice versa. Bridge strength is defined as the sum of the absolute value of all edges that exist between a node and all nodes that are not in the same cluster. In this analysis, PTSD symptoms comprised one cluster, and ED symptoms comprised another cluster.

**Stability.—**We tested the stability of the PTSD-ED networks using the R package *bootnet* case-dropping function (Epskamp et al., 2016). We computed edge weight stability and stability of all centrality indices and calculated the centrality stability coefficients. It is suggested that for interpretation of centrality stability coefficient should be at least above .25 and preferably above .50 (Epskamp, Borsboom, & Fried, 2017).

**Network Comparison Test.—**We also performed the Network Comparison Test (NCT), using the *NetworkComparisonTest* package in R (van Borkulo et al., 2015). NCT can be used to test if there are differences in network structure (i.e., if the way the nodes within the network are connected differs across samples), differences in edge strength (i.e., if a specific edge differs in strength across samples), and/or differences in global strength (i.e., if the sum of the strengths of all edges in the network differs across samples; van Borkulo et al., 2015).

## Results

### Stability Analyses.

Stability of the edge weights for both networks was good (see supplemental materials Figure S4 and Figure S5) with moderate edge weight confidence intervals. Strength centrality stability coefficient was acceptable for clinical network (SC = .44) and good for non-clinical network (SC = .50). Both were above the suggested cut-off of .25 (Epskamp, Borsboom, & Fried, 2017). Centrality stability for betweenness (Clinical SC = .05; non-clinical SC = 0.05) and closeness (Clinical SC = .21; non-clinical SC = 0.21) were poor and therefore these centrality indices were not interpreted. See supplemental figures S6 and S7 for centrality stability graphs.

### Model 1: Clinical PTSD-ED Network (*N* = 158)

In the clinical network (Figure 1), binge eating (ED; bridge strength = .49), irritability (PTSD; bridge strength = .36), desire for a flat stomach (ED; bridge strength = .30), and concentration problems (PTSD; bridge strength = .30) were identified as bridge symptoms. Binge eating (ED) and irritability (PTSD) were linked to each other and had the strongest pathway in the network (part $r = 0.22$). Desire for a flat stomach was most strongly linked to disturbing dreams (PTSD, part $r = 0.10$) and memory problems (PTSD, part $r = 0.05$) in the

PTSD cluster. Concentration problems (PTSD) were most strongly linked to weight and shape-related concentration problems (ED; part $r = 0.13$) and shape dissatisfaction (ED; part $r = 0.11$) in the ED cluster. Figure 2 shows bridge centrality plot for the network.

Based on the stability analyses, only strength centrality was interpreted. As can be seen in Figure 1, in the clinical network, binge eating (ED; strength = 3.20), fear of weight gain (ED; strength = 2.21), disturbing dreams (PTSD; strength = 1.13), and being upset at reminders of trauma (PTSD; strength = 1.10) were core symptoms. Figure 3 shows the centrality plot for the network.

### Model 2: Non-Clinical PTSD-ED Network (*N* = 300)

In the non-clinical network (Figure 1) food-related concentration difficulties (ED; bridge strength = .17), weight and shape-related concentration difficulties (ED; bridge strength = .14), irritability (PTSD; bridge strength = .15), and loss of interest (PTSD; bridge strength = .14) were identified as bridge symptoms. Food-related concentration difficulties were most strongly connected to concentration (PTSD, part $r = 0.05$), sleep (PTSD, part $r = 0.05$), and memory problems (PTSD, part $r = 0.06$) in the PTSD cluster. Weight and shape-related concentration difficulties were similarly most strongly linked to memory (PTSD, part $r = 0.05$) and concentration difficulties (PTSD, part $r = 0.08$) in the PTSD cluster. Irritability (PTSD) was most strongly linked to fasting (ED; part $r = 0.04$) and guilt about weight and shape (ED; part $r = 0.04$) in the ED cluster. Loss of interest (PTSD) was most strongly linked to binge eating (ED; part $r = 0.06$) in the ED cluster. Figure 2 shows bridge centrality plot for the network.

The core symptoms in the non-clinical network were desire to lose weight (ED; strength = 1.95), being upset at reminders of trauma (PTSD; strength = 1.71), preoccupation with shape (ED; strength = 1.51), and weight dissatisfaction (ED; strength = 1.34). Figure 3 shows the centrality plot for the network.

### Network Comparison of Model 1 and Model 2

The results from the *network structure* invariance test showed that the two PTSD-ED networks were not significantly different ($p = .24$). We did not test the differences in edge strength because when the network structure is found to be invariant, there is no reason to pursue further testing of specific edges (van Borkulo et al., 2015). As hypothesized, the global strength invariance test showed that the difference between the global strength of the clinical network (global strength = 20.12) and the global strength of the non-clinical network (global strength = 18.87) was statistically significant ($p < .05$), suggesting that the PTSD-ED network had greater density in the clinical sample than in the non-clinical sample.

## Discussion

The present study used network analysis to conceptualize comorbid PTSD and ED symptoms and found that there were three major illness pathways that may connect the disorders. However, out of these three pathways, we identified that the primary (strongest) pathway was between the ED symptom *binge eating* and the PTSD symptom *irritability*, suggesting that *binge eating - irritability* may be a crucial illness pathway by which

comorbidity is maintained between PTSD and EDs. Another pathway was identified between the ED symptom *desire for a flat stomach* and the PTSD symptoms *disturbing dreams* and *memory problems*. Additionally, in both samples, PTSD symptoms of *concentration*, *memory*, and *sleep problems* were linked to the ED symptoms of *shape and weight dissatisfaction* and *concentration difficulties due to preoccupation with food and body concerns*, suggesting that cognitive difficulties may be another pathway through which ED and PTSD symptoms are connected and PTSD-ED comorbidity is maintained. These results are important because they present novel insights about which symptoms serve as pathways from one disorder to another and which symptoms may maintain PTSD-ED comorbidity. Interventions targeting these symptoms may be more efficient than traditional treatment approaches and may alleviate the need to choose which disorder to treat first.

## Illness Pathways between PTSD and EDs

Consistent with our hypotheses, binge eating and body dissatisfaction (desire for a flat stomach) were implicated as bridge symptoms in PTSD-ED comorbidity network. These results are consistent with the literature on PTSD-ED comorbidity identifying bulimic symptoms as most frequently co-occurring with a history of trauma and implicating body dissatisfaction as a potential link between PTSD and EDs (Brewerton, 2007; Tagay et al., 2014). The current study adds to the literature by elucidating how binge eating and body dissatisfaction are linked to PTSD symptoms.

**Binge eating - Irritability Pathway.—**In the clinical network, *binge eating* (ED) was strongly linked to *irritability* (PTSD), leading to the hypothesis that an increase in the emotion of irritability specifically might lead to a binge eating episode or vice versa. These findings support the idea that PTSD symptoms may act as maintaining factors for ED behaviors by functioning to regulate affect (Trottier et al., 2016). According to affect regulation theory of binge eating (Dingemans, Danner, & Parks, 2017), binge eating serves as a way to regulate distressing emotions, and specifically irritable states, which might then lead to other symptoms of PTSD. These results are particularly interesting considering that our sample consisted primarily of individuals with AN. These findings support previous literature that individuals with AN binge-purge type have higher prevalence of PTSD than individuals with AN restricting type (Reyes-Rodríguez et al., 2011), by showing that even within a primarily AN sample, binge eating is strongly related to PTSD symptoms.

Irritability is defined as a strong emotional response to a stimulus and subsequent unwarranted behavioral reaction (Eichen, Chen, Boutelle, & McCloskey, 2017). Irritability is a common symptom of depression, anxiety, PTSD, bipolar disorders, ADHD, and other mental disorders (American Psychiatric Association, 2013), but research on this emotion has been limited. Irritability has been studied in the context of emotion dysregulation, which is strongly associated with binge eating in the literature (Eichen et al., 2017; Kelly et al., 2016; Selby, Ward, & Joiner, 2010; Southward et al., 2014). Additionally, high levels of irritability may be explained by comorbid conditions such as anxiety and depression, as well as by continued restriction of food intake. Our finding suggests that we need additional research focused specifically on the relationship between irritability and binge eating, as it may be one of the key connections between PTSD and EDs.

**Body Dissatisfaction – Disturbing Dreams Pathway.—**A link between *desire for a flat stomach* (ED) and *disturbing dreams* (PTSD) emerged as another pathway in the clinical network. This pathway suggests that PTSD symptoms might maintain EDs via reminders of trauma (e.g., disturbing dreams), triggering self-critical thoughts about one's body and preoccupation with weight or shape (e.g., desire for a flat stomach). Mitchell et al. (2012) suggest that survivors of sexual abuse may wish to get thin and change their body shape to appear less sexually attractive to avoid a future trauma. These cognitions may then lead to weight-control behaviors such as restriction and trigger binge eating-purge cycles (Fairburn et al., 2009). Understanding these illness pathways is important in identifying a cognitive-behavioral chain during treatment of comorbid PTSD and EDs, which is currently a part of cognitive-behavioral and dialectical behavioral therapies (Fairburn, Cooper, & Shafran, 2003; Linehan, 2013). Emotion regulation strategies may be helpful in disrupting the irritability-binge eating pathway and cognitive restructuring may be effective in modifying trauma-body dissatisfaction cognition links.

**Concentration Difficulties Pathway.—**Difficulties in concentration and memory emerged as another illness pathway in both the clinical and non-clinical PTSD-ED networks. Specifically, concentration problems (PTSD) were linked to shape dissatisfaction (ED) in the clinical network, and food, shape, and weight-related concentration problems (ED) were linked to memory and concentration problems in the context of PTSD. These pathways may be an example of how ED symptoms maintain PTSD psychopathology. It is possible that preoccupation with shape may lead to concentration and memory problems, which are common in both PTSD and EDs. It seems likely that impaired concentration in one area extends to difficulty concentrating in other areas, which might explain how difficulties concentrating progress from one disorder to the other. It is likely that concentration and memory difficulties are consequences of other symptoms like sleep and low body weight and may not themselves be potential targets for intervention. However, concentration and memory difficulties can be impairing and may present a barrier during treatment because patients may be less likely to retain new information.

## Core Symptoms of Clinical PTSD-ED Network

Consistent with our hypothesis, *binge eating*, ED weight-related fears (*fear of weight gain*), cognition alteration symptoms (*desire for flat stomach*), and PTSD intrusion symptoms (*disturbing dreams* and *being upset at reminders of trauma*) were central in the PTSD-ED network. Our findings are partially consistent with other network analyses results of ED psychopathology identifying fear of weight gain and dissatisfaction with shape and weight as core symptoms (DuBois, Rodgers, Franko, Eddy, & Thomas, 2017; Forbush et al., 2016; Levinson et al., 2017). However, other ED psychopathology networks did not identify binge eating as a central symptom, and binge eating was on the periphery of a BN psychopathology network (DuBois et al., 2017; Forbush et al., 2016; Levinson, et al., 2017). In the PTSD-ED comorbidity networks, binge eating was a central symptom with the highest strength centrality. This result is consistent with research that experiencing a traumatic event may be more related to EDs characterized by bulimic symptoms (Brewerton, 2007). These findings suggest that binge eating may be central in the PTSD-ED comorbidity network, but

not in psychopathology networks containing only ED symptoms, because of a strong association between bulimic symptoms and PTSD (Tagay et al., 2014).

We also hypothesized that mood/cognition alteration and intrusion symptoms of PTSD would be core symptoms in the clinical comorbidity network. Consistent with our hypothesis, *disturbing dreams* and *being upset at reminders of trauma* were highly central in the PTSD-ED comorbidity network. *Disturbing dreams* were not only central but were also involved in one of the PTSD-ED illness pathways, which supports the importance of this symptom in understanding the comorbidity. These results support previous literature highlighting sleep disturbance including nightmares as a "hallmark" feature of PTSD (Lamarche & Koninck, 2007, p. 1260). Imaginal exposure therapy (Levinson, Rapp, & Riley, 2014) and Imagery Rehearsal Therapy (Germain & Nielsen, 2003; Krakow et al., 2001) are being used in similar ways to address fear of weight gain and disturbing dreams. Our findings suggest that these symptoms may be important sites of intervention in novel treatment development. Overall, identifying central symptoms may be useful in understanding the comorbidity in the context of bridge symptom analysis (i.e., if central and bridge symptoms are the same). However, centrality itself is unlikely to signify an accurate representation of comorbidity.

## Clinical vs. Non-Clinical Networks

As hypothesized, we found that the networks differed in network density, but not in network structure. Our results replicate the findings of Heeren and McNally (2018) and indicate that individuals in a clinical sample may differ from healthy controls not in the way in which the symptoms interact, but in how strongly connected the symptoms are. For example, disturbing dreams would be unlikely to trigger other PTSD or ED-related symptoms in healthy individuals but would be more likely to do so in an individual with a diagnosed ED because of the strong connections between symptoms (rather than how the symptoms relate to each other). There is a discrepancy in the literature as to whether network density at baseline predicts treatment success (Schweren et al., 2017; van Borkulo et al., 2015). However, our results suggest that because non-clinical networks have lower density, symptom reduction would be associated with weakening of network connectivity. These findings have important clinical implications, because they suggest that treatments should focus on weakening connections as a whole within psychopathology networks, and that this reduction in symptom connections might be representative of healthy functioning. Network density might be used to compare clinical and healthy samples, to assess treatment outcomes (e.g., whether the network k is less dense post-treatment), and to identify groups of individuals who are less likely to respond to treatment. Future research should compare network density pre- and post-treatment, as well as in other healthy vs non-healthy samples. Overall, the similarity of central and bridge symptoms between the networks and lesser intensity of symptoms and weaker connections between them in the non-clinical network adds to the validity of the clinical network.

## Limitations and Future Directions

Certain limitations of this study are worth considering. First and foremost, we have a relatively small sample size considering recent recommendations of having at least three

participants per parameter (Fried & Cramer, 2017). However, both networks had acceptable and good stability, which allows us to interpret the network even with a smaller sample size (Epskamp, Borsboom, & Fried, 2017). We hope that future research will replicate these results in a larger sample. Further, our clinical sample was drawn from individuals who had been discharged from ED treatment and only a portion had comorbid PTSD. Selecting a sample of individuals with comorbid PTSD-ED screened by diagnostic interview may produce a different network. Additionally, we used self-report measures to establish participants' diagnoses. We also did not collect information on the nature of trauma, and networks may differ based on the type of traumatic experiences (e.g., combat vs. sexual assault). Additionally, our data is cross-sectional. Although network theory implies causal relationships between symptoms, we are not able to claim any directional patterns without examining prospective data. Therefore, bridge and core symptoms may influence other symptoms they are linked to or instead be on the receiving end of the causal chain. If symptoms with high centrality develop as consequences of other symptoms, they would not be the best target for intervention. Thus, our assertion that targeting core network symptoms in psychotherapy will lead to a reduction of other symptoms needs to be tested in prospective and experimental data. Additionally, it is possible that because of the high connectedness of the central symptoms, even if they are reduced during an intervention, they might be easily re-activated by other symptoms. We hope that future studies examine the efficacy of targeting bridge and core symptoms in pre-post designs and compare it to treatment as usual.

Also, our study may have been limited by the symptoms we chose to include, as using different measures for ED or PTSD symptoms may produce different psychopathology networks. Future research should examine the replicability of findings regarding core network symptoms using various measurements. Finally, it is worth noting that our sample consisted of primarily individuals with AN or Atypical AN, which limits the generalizability of the findings to other ED diagnoses. Because there are differences in comorbidity rates between PTSD and each type of ED (Tagay et al., 2014), comorbidity networks for specific ED diagnoses with PTSD may be different. It would be worthwhile to compare networks consisting of individuals with restricting AN and binge-purge AN. Additionally, we did not complete diagnostic interviews with the undergraduate sample, and a part of the sample likely also had an ED diagnosis. Finally, it is unclear if these findings will generalize beyond a treatment-seeking sample and to other ED diagnoses. Future research should replicate these findings in different types of samples, including among those with diverse ED diagnoses, non-treatment seeking samples, with individuals at different stages of illness, and in samples of fully comorbid PTSD and EDs. Despite limitations, this study has several strengths. First, to our knowledge, this is the first study that used network analysis to examine comorbidity between PTSD and ED. Second, this paper is contributing to the growing body of literature using network analysis in psychopathology. Network analysis is a quickly advancing methodology that allows for a novel conceptualization of mental disorders and their comorbidity. Third, we were able to compare comorbidity networks in both clinical and non-clinical samples and examine differences in the network structure and density.

## Conclusions

The current study provides a new perspective on the nature of comorbidity between PTSD and EDs and contributes to the growing area of research using network analysis to understand psychopathology. Our findings suggest that concentration difficulties, the connection between binge eating and irritability, and between body dissatisfaction and disturbing dreams may be pathways by which comorbidity is maintained. These results support the emotion regulation hypothesis linking PTSD and ED, as well as the role of body dissatisfaction in connection between trauma and ED symptoms. Comparisons of clinical and non-clinical networks revealed that the networks show similar structure but differ in density, which is associated with symptom severity.

Comorbid psychological conditions present a challenge to clinicians regarding which symptoms to address first. Bridge symptoms, such as irritability, identified in comorbidity networks may be reasonable targets for clinical intervention aimed at disrupting the pathways that maintain the comorbidity. Further, if bridge symptoms also have high strength centrality in the network, intervening on these symptoms may also decrease other symptoms within a psychopathology network (Borsboom & Cramer, 2013; Hofmann, Curtiss, & McNally, 2016). Identifying illness pathways and core symptoms may present a first step in developing a novel approach to treating comorbid disorders. We hope that future research will examine the clinical utility of targeting the bridge and core symptoms identified here in prospective and intervention designs.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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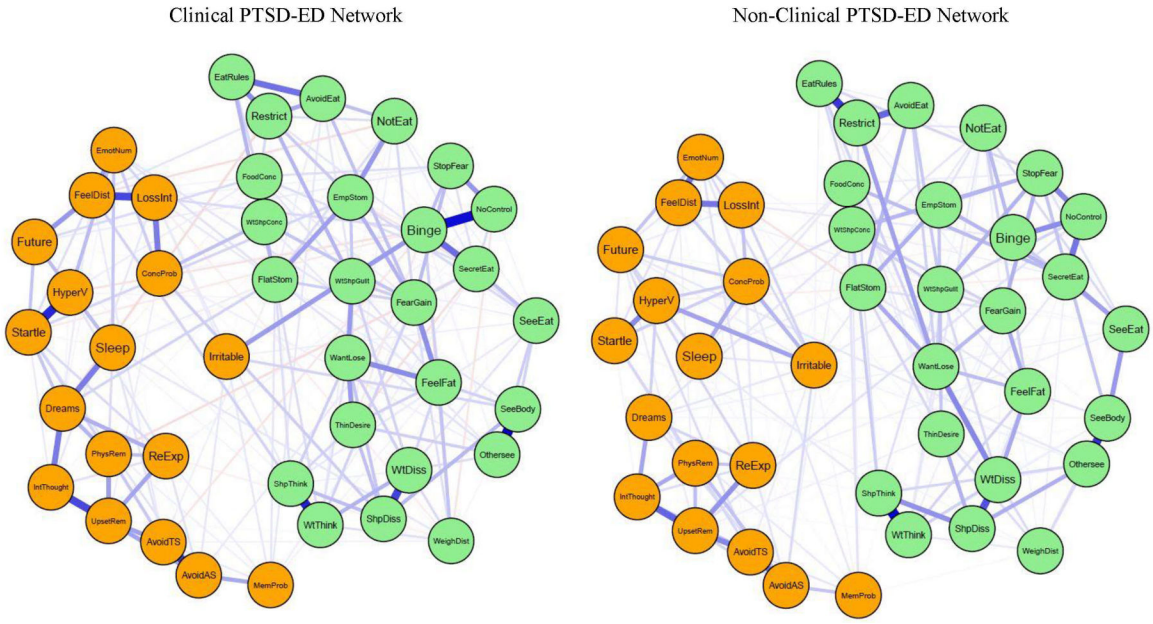


**Figure 1.**

Clinical and Non-Clinical PTSD-ED Networks.

*Note*. PTSD symptoms are in orange and ED symptoms are in green. Thicker lines between nodes represent stronger relationships. PTSD symptom label descriptions: Avoid.A.S = avoidance of activities and situations; Avoid.T.S = avoidance of thinking or speaking; Conc.Prob = concentration problems; Dreams = disturbing dreams; Emot.Numb = emotional numbness; Feel.Dist = feeling distant; HyperV = hypervigilance; IntrusiveT = intrusive and disturbing thoughts; Irritable = feeling irritable; Loss.Int = loss of interest; Mem.Prob = memory problems; No.Future = feeling like future will be cut short; Phys.Rem = physical reactions to reminders; Re.Exp = re-experiencing; Sleep.Prob = sleep problems; Startle = easily startled; Upset.Rem = upset at reminders. ED symptom label descriptions: AvoidEat = avoidance of eating liked foods; Binge = binge-eating episodes; DesireLoseWt = desire to lose weight; DiscOtherBody = discomfort at others seeing one's body; DiscSelfBody = discomfort at seeing one's own body; EatRules = following rules about eating; EmptyStom = desire for empty stomach; FeelFat = feeling fat; FlatStom = desire for flat stomach; FoodConc = concentration problems related to food; NoControl = loss of control of eating; NotEat = long periods of not eating; Restrict = restriction of food intake; SecretEat = eating in secret; SeeEat = concern over being seen eating; ShpDissat = shape dissatisfaction; ShpThink = shape-influenced self-evaluation; StopFear = fear of being unable to stop eating; ThinDesire = desire for thinness; WeighDistress = distress about weighing oneself; WtDissat = weight dissatisfaction; WtGainFear = fear of weight gain; WtShpConc = concentration problems related to weight or shape; WtShpGuilt = feeling guilty about weight or shape; WtThink = weight-influence self-evaluation.

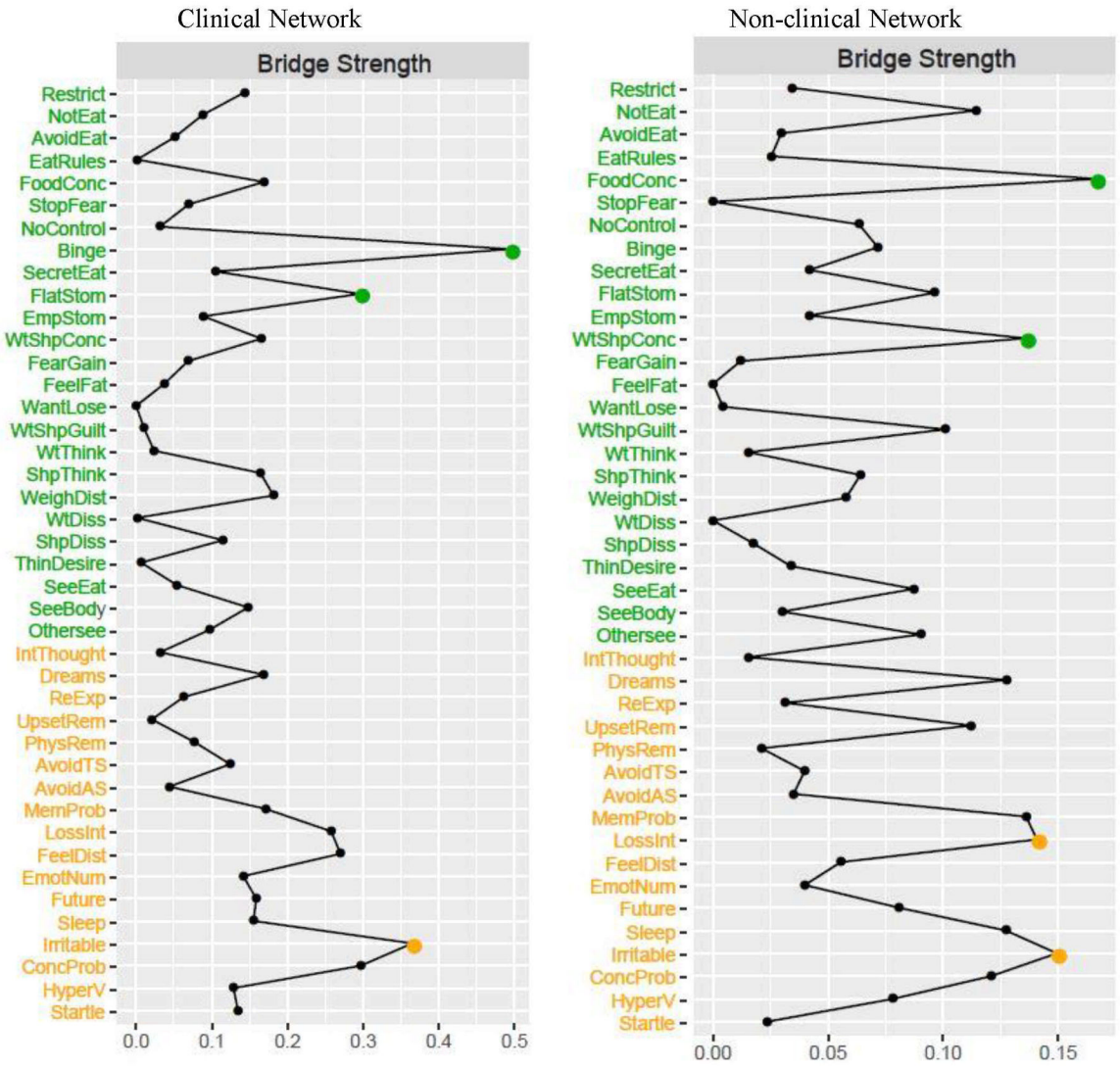


**Figure 2.**

Bridge Strength Plot for Clinical and Non-Clinical PTSD-ED Networks.

PTSD symptom label descriptions: Avoid.A.S = avoidance of activities and situations; Avoid.T.S = avoidance of thinking or speaking; Conc.Prob = concentration problems; Dreams = disturbing dreams; Emot.Numb = emotional numbness; Feel.Dist = feeling distant; HyperV = hypervigilance; IntrusiveT = intrusive and disturbing thoughts; Irritable = feeling irritable; Loss.Int = loss of interest; Mem.Prob = memory problems; No.Future = feeling like future will be cut short; Phys.Rem = physical reactions to reminders; Re.Exp = re-experiencing; Sleep.Prob = sleep problems; Startle = easily startled; Upset.Rem = upset at reminders. ED symptom label descriptions: AvoidEat = avoidance of eating liked foods; Binge = binge-eating episodes; DesireLoseWt = desire to lose weight; DiscOtherBody = discomfort at others seeing one's body; DiscSelfBody = discomfort at seeing one's own body; EatRules = following rules about eating; EmptyStom = desire for empty stomach; FeelFat = feeling fat; FlatStom = desire for flat stomach; FoodConc = concentration problems related to food; NoControl = loss of control of eating; NotEat = long periods of not

eating; Restrict = restriction of food intake; SecretEat = eating in secret; SeeEat = concern over being seen eating; ShpDissat = shape dissatisfaction; ShpThink = shape-influenced self-evaluation; StopFear = fear of being unable to stop eating; ThinDesire = desire for thinness; WeighDistress = distress about weighing oneself; WtDissat = weight dissatisfaction; WtGainFear = fear of weight gain; WtShpConc = concentration problems related to weight or shape; WtShpGuilt = feeling guilty about weight or shape; WtThink = weight-influence self-evaluation.

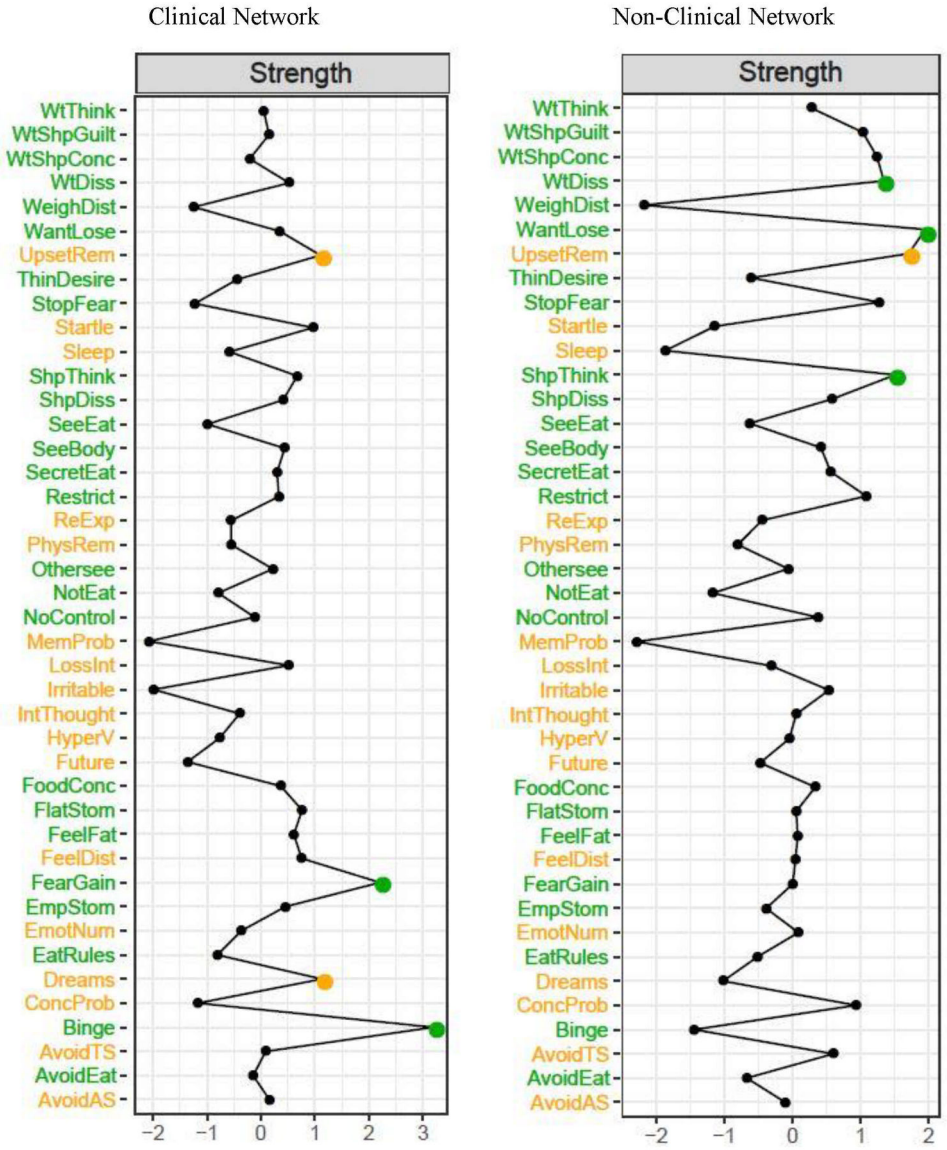


**Figure 3.**

Strength Centrality Plots.

*Note*. PTSD symptoms are in orange and ED symptoms are in green. Higher values indicate that a node is more central to the network.

**Table 1.**

Demographics and Diagnostic Descriptions

| | | | Clinical | | | Non-Clinical | | |
|---|---|---|---|---|---|---|---|---|
| | | ***N* (%)** | ***M (SD)*** | **Range** | ***N* (%)** | ***M (SD)*** | **Range** | ***p*** |
| **Age** | | | **25.77 (8.95)** | **14–59** | | **18.71 (1.05)** | **17–23** | **<0.001** |
| **EDE-Q** | | | **3.17 (1.54)** | **.05–4.68** | | **1.46 (1.13)** | **0–5** | **<0.001** |
| **PCL-C** | | | **44.33 (16.22)** | **17–82** | | **29.34 (9.84)** | **17–68** | **<0.001** |
| Female | | 151 (95.6) | | | 300 (100%) | | | |
| Ethnicity | | | | | | | | |
| | European American | 148 (93.7) | | | 182 (60.7) | | | |
| | Afr. American | 1 (0.6) | | | 12 (4) | | | |
| | Hispanic | 3 (1.9) | | | 8 (2.7) | | | |
| | Asian | 1 (0.6) | | | 82 (19.3) | | | |
| | Multiracial | 3 (1.9) | | | 15 (5) | | | |
| ED Diagnosis | | | | | | | | |
| | AN/Atypical AN | 134 (84.8) | | | | | | |
| | BN | 9 (5.7) | | | | | | |
| | BED | 1 (0.6) | | | | | | |
| | OSFED | 14 (8.9) | | | | | | |
| PTSD Diagnosis | Score over 35 | 84 (54) | | | 95 (32) | | | |
| | Score over 44 | 58 (35) | | | 28 (9) | | | |
| Treatment | | | | | | | | |
| | Inpatient | 1 (0.6) | | | | | | |
| | Residential | 4 (2.4) | | | | | | |
| | PHP | 3 (1.8) | | | | | | |
| | IOP | 8 (4.8) | | | | | | |
| | Outpatient | 66 (39.3) | | | | | | |

*Note.* EDE-Q = Eating Disorder Examination Questionnaire; PCL-C = The PTSD Checklist – Civilian Version; AN = Anorexia Nervosa; BN = Bulimia Nervosa; BED = Binge Eating disorder; OSFED = Other Specified Feeding or Eating Disorder; PTSD = post-traumatic stress disorder; PHP = Partial Hospitalization; IOP – Intensive Outpatient. Diagnostic information is based on self-report.