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Obsessive–compulsive symptoms in eating disorders: A network investigation

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Abstract

Objective: Eating disorders (EDs) are complex, heterogeneous, and severe psychiatric syndromes. They are highly comorbid with obsessive–compulsive disorder (OCD) which exacerbates the course of illness and impedes treatment. However, the direct functional relations between EDs and OCD symptoms remain largely unexplored. Hence, using network analysis, we investigated the relationship between ED and OCD at the level of symptoms in a heterogeneous clinical sample.

Method: We used cross sectional data of 303 treatment-seeking patients with clinically relevant ED and OCD pathology. We constructed a regularized partial correlation network that featured both ED and OCD symptoms as nodes. To determine each symptom's influence, we calculated expected influence (EI) as an index of symptom centrality (i.e., "importance"). Bridge symptoms (i.e., symptoms from one syndromic cluster that have strong connections to symptoms of another syndromic cluster) were identified by computing bridge expected influence metrics.

Results: Fear of weight gain and dietary restraint were especially important among the ED symptoms. Interference due to obsessions was the key feature of OCD. ED and OCD clustered distinctly with few potential bridges between clusters.

Discussion: This study underscores the importance of cognitive symptoms for both ED and OCD although direct functional links between the two clusters are missing. Potentially, a network incorporating nodes capturing features of personality may account for diagnostic comorbidity better than specific symptoms of EDs or features of OCD do.

KEYWORDS

comorbidity, core psychopathology, eating disorders, network analysis, obsessive–compulsive disorders

1 | INTRODUCTION

Eating disorders (EDs) and obsessive–compulsive disorder (OCD) co-occur with great frequency. Approximately 41% of individuals with a primary ED diagnosis (35% of those with anorexia nervosa [AN], 40% of those with bulimia nervosa [BN]) have a lifetime OCD comorbidity (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). Ten percent of individuals with OCD have an ED (3% AN, BN, and eating disorder not otherwise specified [EDNOS], respectively) at some point in their life (Pinto, Mancebo, Eisen, Pagano, & Rasmussen, 2006). The comorbid occurrence of ED and OCD is associated with a prolonged period of illness (Milos, Spindler, Ruggiero, Klaghofer, & Schnyder, 2002), worse prognosis for overall functioning (Wentz, Gillberg, Gillberg, & Råstam, 2001), higher rates of other comorbidities, and greater risk

for suicide attempts (Sallet et al., 2010). Finally, genomic research has uncovered a large genetic correlation between OCD and AN that suggests shared vulnerability for these syndromes (Yilmaz et al., 2019).

Given these alarming features, studies have investigated factors influencing this comorbidity, revealing similarities with regard to age of onset, gender distribution, and comorbidity rates (Kaye et al., 2004; Pinto et al., 2006), genetics (Cavallini, Bertelli, Chiapparino, Riboldi, & Bellodi, 2000), abnormalities in the serotonergic system (Kaye, 2008; Murphy et al., 1989), and good responsivity to high dose selective serotonin reuptake inhibitors in both patient groups (Pigott, & Seay, 1999; Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992). Moreover, individuals with ED and those with OCD show similar personality features, for example, perfectionism, obsessionality, impulsivity, and concern over mistake (for a review see Altman & Shankman, 2009). On a functional level, researchers found both pathologies to be marked by intensive preoccupations, that is, obsessions, with a certain stimulus (e.g., for ED food or weight/shape, for OCD obsessions about for example, contamination) that evoke negative affect and are followed by compensatory behavior (e.g., engaging in purging in the case of ED or in compulsive washing in the case of OCD), which decreases negative affect or anxiety (Altman & Shankman, 2009). Preliminary evidence further indicates that intrusions appear with comparable frequency and with similar emotional disturbance among individuals with OCD and ED (García-Soriano, Roncero, Perpiñá, & Belloch, 2014), although it remains unclear whether patients with ED have similar dysfunctional appraisals as patients with OCD (García-Soriano et al., 2014; Lavender, Shubert, de Silva, & Treasure, 2006).

However, although the similarities of ED and OCD have been extensively studied, the specific illness pathways between these syndromes remain largely unexplored. Network theory provides a compelling framework for disclosing the functional relations abiding among the symptoms of comorbid conditions (Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011; Cramer, Waldorp, van der Maas, & Borsboom, 2010). Networks comprise circles ("nodes") and lines ("edges") that depict associations between nodes. In a typical cross sectional psychopathology network, the nodes are symptoms and the thickness of the edges connecting two nodes signify the strength of association (e.g., correlation or partial correlation) between the two connected symptoms and thus the probability of their co-occurrence. Moreover, network theory postulates that a disorder emerges from interactions among the nodes. For example, concerns about one's shape and size may cause restricted eating that leads to hunger, which triggers binge eating and subsequent purging. Hence, network theorists appeal to the functional relations among these nodes to explain why symptoms cluster syndromically. This view differs from traditional medical views that postulate an underlying common cause whose presence is reflected by the symptoms it causes. For example, a fever of 102°, pain upon swallowing, white blotches in one's throat, and fatigue co-occur result from the underlying common cause of the streptococcus bacterium, not because of any direct causal relations among the symptoms themselves.

In contrast to the traditional reflective common cause model that postulates symptoms to arise from an underlying disorder (Borsboom, Mellenbergh, & Van Heerden, 2003), network theory holds that the symptoms causally interact with each other and are thus themselves constitutive of the disorder (Borsboom & Cramer, 2013). By analyzing networks of psychopathological symptoms of a specific disorder, researchers can thus identify single symptoms that have many connections to other symptoms and are thus potentially important ("central") to the network as a whole. Such highly connected symptoms are plausible candidates for maintaining, and possibly causing, an episode of disorder. Similarly, network analysis can help to identify so-called bridge symptoms (Cramer et al., 2010). Bridge symptoms may play a key role in comorbidity by connecting two syndromic clusters, and if the connection is causal and the direction of influence issues outward from the bridge symptom to symptoms of both clusters. Conversely, successful treatment that deactivates bridge symptoms may hasten recovery from the comorbid disorders that they connect (Borsboom et al., 2011; Borsboom & Cramer, 2013; McNally, 2016). Network analysis thus identifies core maintaining symptoms and illness pathways.

Over the last few years, a growing number of network studies has investigated the symptomatic interplay *within* ED clusters, for example, for AN (e.g., Forrest, Jones, Ortiz, & Smith, 2018), BN (e.g., Levinson, Rapp, & Riley, 2014), and binge eating disorder (BED; Wang, Jones, Dreier, Elliott, & Grilo, 2019). Consistent with Fairburn's (2008) transdiagnostic model of EDs, they have confirmed the importance ("centrality") of overevaluation of shape and weight (DuBois, Rodgers, Franko, Eddy, & Thomas, 2017; Forrest et al., 2018; Wang et al., 2019) across ED subtypes. Moreover, fear of weight gain emerged as a highly important symptom in both BN (Levinson et al., 2014) and AN networks (Forrest et al., 2018).

Despite the high rates of comorbidity, few studies have examined the symptomatic interplay between ED and OCD. Studying the comorbid occurrence of ED and OCD in a heterogeneous ED sample with clinically relevant OCD by means of network analysis thus has theoretical and clinical implications. First, the investigation of the functional relations helps understand which symptoms are closely related and potentially influence each other, within and across disorders. The identification of such illness pathways, important nodes, and potential bridge symptoms may then guide clinicians to first target such symptoms and links to other symptoms relevant to the emergence of comorbidity. Second, by analyzing the network structure of this comorbidity, this study can further contribute to the current debate whether OCD and ED should be seen as distinct pathologies or if they should rather be conceptualized on a spectrum (as for example, suggested by Altman & Shankman, 2009). Lastly, investigations of the separate ED syndrome cluster could extend prior ED network analytic studies limited by small sample size, subclinical severity, or both.

2 | METHOD

2.1 | Participants

The initial sample consisted of 1,443 individuals who sought treatment for ED in five treatment centers in the United States in the

Rogers Behavioral Health System. Upon admission, they completed a battery of questionnaires. All provided written informed consent to use their de-identified data for research purposes. Because the archival data analyzed in the present study were de-identified, our protocol was deemed exempt by Harvard University's Committee on the Use of Human Subjects.

For this study, we excluded 629 individuals (44%) who were younger than 18 by the time of baseline assessment and 201 individuals (14%) who did not complete the necessary ED and OCD measures. To ensure clinically relevant psychopathology, we excluded 160 individuals (11%) who scored below a clinical cutoff on the eating disorder examination questionnaire (EDE-Q) version 6.0 (Fairburn & Beglin, 2008; threshold of two) and 150 (10%) who scored below a clinical cutoff on the Yale–Brown obsessive compulsive scale—self-report (Y-BOCS-SR; Steketee, Frost, & Bogart, 1996; threshold of 16). Because using a symptom severity threshold as an inclusion criterion can alter network structure via Berkson's Bias (De Ron, Fried, & Epskamp, 2019; Rohrer, 2018), we also analyzed the data without applying an EDE-Q threshold (n = 370; see Supplement) as a sensitivity analysis.

Our sample thus comprises cross sectional admission data from 303 individuals (257 women, 45 men, 1 nonbinary, M_{age} = 30.29, SD_{age} = 11.80, age range = 18–79). The patients were treated in inpatient (n = 143), partial (n = 93), residential (n = 61), and intensive outpatient (n = 6) programs. The majority (95%) was Caucasian, and 68% held a college or university degree. The mean body mass index (BMI) of the sample was 25.36 (SD = 10.56, range = 12.17–73.85). When we applied a diagnostic algorithm to the EDE-Q data that derives DSM-5 (APA, 2013) ED diagnoses (Berg, Peterson, Franzier, & Crow, 2012), 40 individuals (13%) qualified for AN, 44 individuals (15%) for BN, 83 individuals (27%) for BED, and 136 individuals (45%) for eating disorder not otherwise specified (EDNOS). The algorithm uses 85% of the ideal body weight as weight criteria. For an AN diagnosis, patients must endorse fear of weight gain or compensatory behaviors as well as overevaluation of shape or weight. For a BN diagnosis, patients must report at least one bulimic episode per week and compensatory behaviors more than once a week, and an overevaluation of shape or weight. For a BED diagnosis, bulimic episodes had to occur more than once per week and compensatory behaviors less than once a month.

2.2 | Measures

2.2.1 | Eating disorder examination questionnaire

The EDE-Q version 6.0 (Fairburn & Beglin, 2008) is the self-report questionnaire version of the eating disorder examination (EDE) semi-structured interview (Cooper & Fairburn, 1987). It assesses ED behaviors (e.g., binge eating, purging) and attitudinal features of ED (e.g., overimportance of shape) over the last 28 days. The six behavioral items assess frequencies of ED behaviors in terms of number of days over the last 28 days the specific behavior occurred. The 22 attitudinal items are assessed with a seven-point forced-choice format ranging from 0 to 6, with higher scores indicating greater levels of pathology. The EDE-Q has good psychometric properties (internal consistency: 0.70–0.93; concurrent validity with daily food records: 0.31–0.63; Berg et al., 2012) and reliably differentiates between clinically impaired and healthy individuals (e.g., Mond, Hay, Rodgers, Owen, & Beumont, 2004). Studies with clinical samples indicated that nearly half of those with an ED diagnosis score lower than the clinical cutoff (Fairburn & Beglin, 1994) of four on the EDE-Q (Aardoom, Dingemans, Slof Op'Landt, & Van Furth, 2012). In line with studies using receiver operating characteristics analysis (e.g., Mond et al., 2004), we lowered to two.

2.2.2 | Yale–Brown obsessive compulsive scale – Self-report

The Y-BOCS-SR (Steketee et al., 1996) is the self-report version of the Y-BOCS-SR interview (Goodman et al., 1989) that assesses OC symptoms over the previous week. Both interview and questionnaire show very good psychometric properties (for the Y-BOCS-SR: internal consistency = 0.78–0.89; convergent validity with the Y-BOCS: 0.75–0.79; Steketee et al., 1996). The obsessive and compulsive subscales each contain five items that measure five aspects of OC pathology on a five-point Likert scale from 0 (no symptom) to 4 (extreme): spent time, level of interference, distress, resistance (lower levels of resistance are assigned higher scores), and perceived control (lower levels of control are assigned higher scores). Total scores thus range from 0 to 40 with higher scores indicating higher severity. In line with previous research, we used a clinical cutoff of 16 (e.g., McNally, Mair, Mugno, & Riemann, 2017).

2.3 | Network estimation

For data analysis, we used R (version 3.5.2; R Core Team, 2018). We applied the R packages *qgraph* (version 1.5; Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012) and *networktools* (version 1.2.0; Jones, 2017) to estimate and visualize regularized partial correlation networks. In psychopathological networks, nodes represent symptoms and the thickness of a line ("edge") connecting two nodes signifies the magnitude of their association. In partial correlation networks, edges represent the partial correlation coefficient between two symptoms while controlling for the influence of all remaining nodes in the network. In *regularized* partial correlation networks, regularization techniques control for trivial and likely *spurious*, connections between nodes by setting them to zero (Costantini et al., 2015). Using the extended bayesian information criterion (EBIC), we regularized the underlying covariance matrix by applying a graphical least absolute shrinkage and selection operator (LASSO, Friedman, Hastie, & Tibshirani, 2008). The LASSO is a likelihood penalization technique that chooses an inverse covariance matrix (precision matrix) characterized by (a) parsimony and (b) optimized model fit. In analogy to λ that defines the level of sparsity, the hyperparameter, that is, tuning

TABLE 1 Item scores on the Y-BOCS-SR and the EDE-Q and labels for the networks

Item	Mean (*SD*)	Label
Y-BOCS-SR		
1. Time consumed by obsessions	2.56 (0.93)	*otime*
2. Interference due to obsessions	2.17 (0.98)	*ointerf*
3. Distress caused by obsessions	2.42 (0.90)	*odistress*
4. Difficulty resisting obsessions	2.09 (0.94)	*oresist*
5. Difficulty controlling obsessions	2.55 (0.82)	*ocontrol*
6. Time consumed by compulsions	2.21 (0.92)	*ctime*
7. Interference due to compulsions	2.03 (1.02)	*cinterf*
8. Distress caused by compulsions	2.62 (0.87)	*cdistress*
9. Difficulty resisting compulsions	2.25 (0.82)	*cresist*
10. Difficulty controlling compulsions	2.45 (0.79)	*ccontrol*
EDE-Q		
1. Restrained eating	4.26 (2.12)	*restraint*
2. Restricted eating	2.44 (2.24)	*restrict*
3. Excluding foods	4.17 (2.20)	*exclude*
4. Dieting rules	4.10 (2.31)	*rules*
5. Desiring empty stomach	3.90 (2.40)	*emptystom*
6. Desiring flat stomach	4.96 (1.88)	*flatstom*
7. Preoccupation with food, eating, or calories	4.59 (1.83)	*occfood*
8. Preoccupation with shape or weight	4.51 (1.91)	*occshpwt*
9. Fear of loss of control over eating	4.57 (2.03)	*fearloc*
10. Fear of weight gain	5.22 (1.65)	*fearwt*
11. Feeling fat	5.13 (1.63)	*feelfat*
12. Desiring weight loss	4.93 (1.80)	*wishloss*
13. Times of overeating	11.17 (15.72)	*overeat*
14. Loss of control while overeating	11.83 (12.39)	*loc*
15. Times of binge eating	8.89 (10.62)	*binge*
16. Self-induced vomiting	10.43 (17.59)	*vomit*
17. Laxative use	2.92 (8.21)	*laxatives*
18. Over-exercising	7.06 (9.86)	*exercise*
19. Eating in secret	2.07 (2.08)	*secret*
20. Feeling guilty when eating	4.40 (1.78)	*guilt*
21. Concerns that others see one eat	4.16 (1.79)	*socialeat*
22. Weight overvaluation	5.18 (1.30)	*wtover*
23. Shape overvaluation	5.17 (1.30)	*shpover*
24. Upset when having to weight oneself	3.85 (2.08)	*rxweigh*
25. Dissatisfaction with weight	5.17 (2.08)	*disswt*
26. Dissatisfaction with shape	5.20 (1.38)	*dissshp*
27. Discomfort when seeing own body	5.22 (1.24)	*discown*
28. Discomfort when others see one's body	5.14 (1.40)	*discomf*

Note: EDE-Q, eating disorder examination questionnaire (Fairburn & Beglin, 2008); Y-BOCS-SR, Yale–Brown obsessive compulsive scale—self-report version (Steketee et al., 1996); *SD*, standard deviation. The EDE-Q contains seven-point scales ranging from 0 to 6 and count data. The Y-BOCS-SR is a five-point scale ranging from 0 to 4.

parameter, γ defines how much a simpler, that is, sparser, model is preferred (Chen & Chen, 2008). In line with previous research (Beard et al., 2016), we set the initial γ to 0.5.

Network analysis presupposes that each node represents a distinct entity (i.e., process, construct). Unfortunately, questionnaires in psychology sometimes include items that are synonyms (e.g., *feeling sad; feeling* blue). Having such synonymous items designated as distinct nodes can distort network analyses, especially measures of node importance (e.g., strength centrality; expected influence). Hence, researchers need to identify synonymous items and combine them to form a single node prior to computing networks. To detect redundant nodes, that is, nodes that reflect the same construct, we used the *goldbricker* function in the R package *networktools* (Jones, 2017). The *goldbricker* function assumes that variables that measure the same construct should covary in similar ways with other variables. Hence, *goldbricker* computes the proportions of those correlations that significantly differ for each pair of nodes. By applying a threshold of 0.25, the function returns a list of node pairs that differ in less than or equal to 25% of their correlations to other nodes (i.e., redundant nodes). After applying *goldbricker* to the separate EDE-Q and Y-BOCS-SR networks, we combined those nodes by means of Principal Component Analysis (PCA) that (a) fell below the threshold of 0.25 and (b) were reasonable to combine from a clinical perspective.

2.4 | Expected influence centrality and bridge expected influence centrality

Node strength centrality signifies the number and magnitude of edges connecting a node to other nodes in the network (Borsboom et al., 2011). Within the scope of psychopathology, symptoms that are especially central, that is, have many strong connections to other symptoms, are presumably important for the development and maintenance of the entire network and therefore crucial to target in clinical interventions if they represent the source of activation (Borsboom et al., 2011; Borsboom & Cramer, 2013; McNally, 2016).

We calculated the *one-step expected influence* (EI; Robinaugh, Millner, & McNally, 2016) as a measure of centrality via the package *networktools* (Jones, 2017). For each node, EI represents the summed weight of all its edges, positive and negative, with its immediate neighbor nodes in the network. That is, EI considers the sign of an association connecting two nodes (i.e., negative vs. positive partial correlation) when summing the magnitude of the edges connected to the node.

We further computed the *bridge expected influence* (bEI). The bEI of one node is the summed edge weights to all nodes of the other disorder. Higher bEI values indicate stronger and more numerous associations between an ED symptom and nodes in the other cluster (e.g., features of OCD), and stronger and more numerous associations between a feature of OCD and nodes in the other cluster (e.g., symptoms of ED). However, as we computed an undirected network, we cannot make claims about the direction of potential influence of these bridge symptoms (i.e., which symptoms rather receive activation from bridge symptoms, and which activate the bridge symptoms themselves).

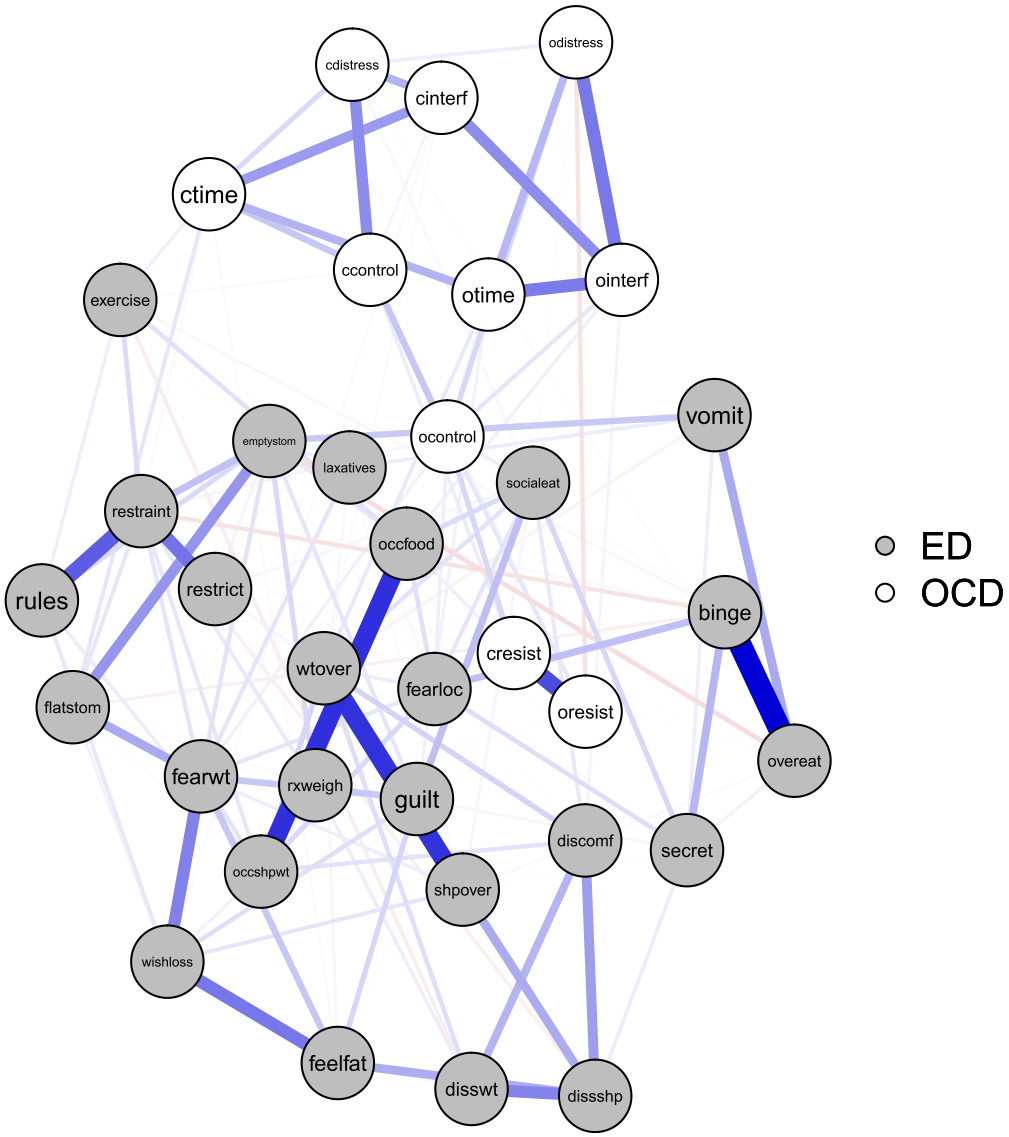

FIGURE 1 Network constructed by using the graphical least absolute shrinkage and selection parameter (LASSO). Edges depict regularized partial correlations between symptoms assessed with the eating disorder examination questionnaire (EDE-Q) version 6.0 (Fairburn & Beglin, 2008) and the Yale–Brown obsessive compulsive scale—self-report (Y-BOCS-SR; Steketee et al., 1996) [Color figure can be viewed at wileyonlinelibrary.com]

2.5 | Stability

We used the R package *bootnet* (version 1.2; Epskamp, Borsboom, & Fried, 2018) to assess the accuracy of the calculated estimated networks structures and centrality measures. To estimate the stability of the edges in terms of the EI and the bEI, we used *bootnet* to conduct 1,000 bootstraps of the EI/bEI for each node and for each edge with increasingly smaller subsets of subjects.

3 | RESULTS

3.1 | Missing data

Given the small amount of missing data (0.002% for the EDE-Q) and that the answers were missing for different individuals, we judged it to be missing at random (Rubin, 1976). The missing data points were imputed with the R package *mice* (version 3.3.0; van Buuren & Groothuis-Oudshoorn, 2011) in a single imputed dataset. Means and standard deviations of the EDE-Q and the Y-BOCS-SR items and the node labels appear in Table 1. Participants scored on average 4.39 on the EDE-Q (*SD* = 1.00, range = 2–6) and 23.35 on the Y-BOCS-SR (*SD* = 5.23, range = 16–39).

3.1.1 | Redundant nodes

Among the ED symptoms, the *goldbricker* function returned six pairs of nodes that differed in less than 25% of their correlations with other nodes. We decided to collapse three pairs, namely, trying to exclude foods (*exclude*) and trying to follow rules (*rules*); loss of control while overeating (*loc*) and times of binge eating (*binge*), and dissatisfaction with weight *(disswt)* and discomfort when seeing own body *(discown)*. The node labels were accordingly adapted to *rules (exclude* and *rules)*, *binge (loc* and *binge)*, *disswt (disswt* and *discown)*. Using PCA to combine items increases the variance of the comorbid items relative to

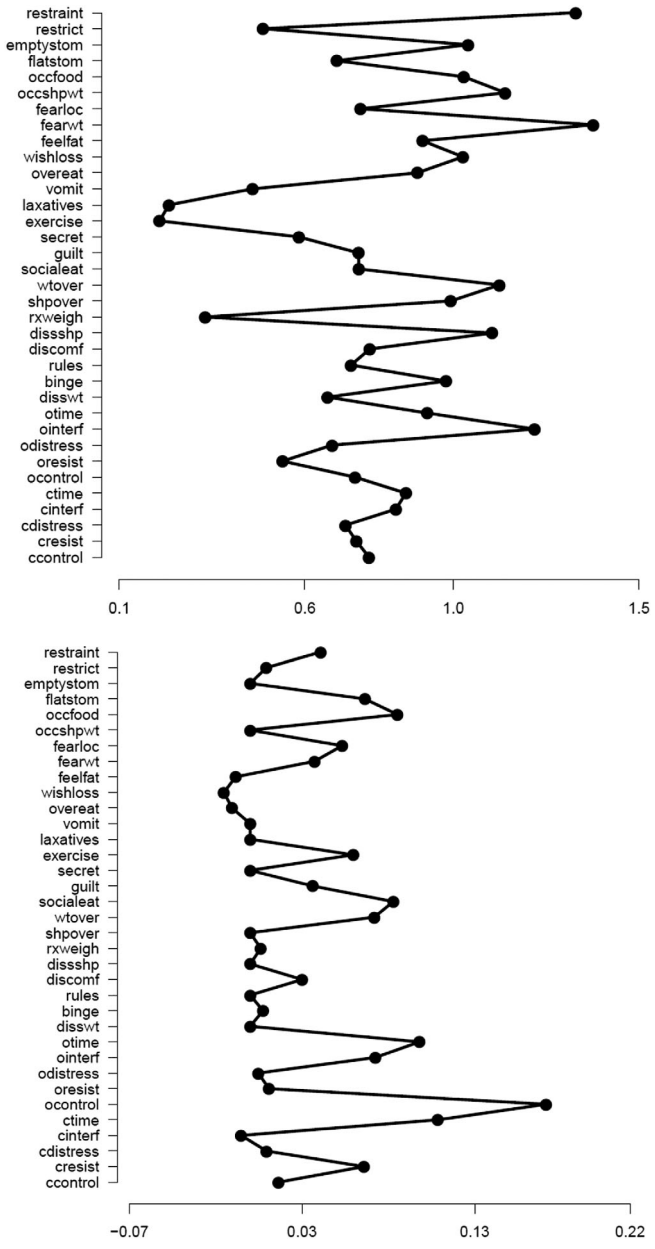

FIGURE 2 Centrality plots featuring expected influence (EI) on the top and bridge expected influence (bEI) on the bottom for each node in the network. Each plot depicts nodes on the *y* axis and the raw EI and bEI on the *x* axis

noncombined items. To account for the possibility that inflated variances biased centrality estimates, we conducted a sensitivity analysis with items standardized by variance. This analysis showed that centrality estimates of PCA-combined items were not inflated and centrality measures correlated highly when items were standardized versus non-standardized (r = .81). The goldbricker function did not indicate more redundancies after combining these pairs. There were no redundant nodes among the OC symptoms. The complete network thus comprised 35 nodes.

3.2 | Regularized partial correlation network

The regularized partial correlation network is depicted in Figure 1. Blue edges represent positive partial correlations and red edges represent negative partial correlations. Thicker and more saturated edges indicate stronger connections. As we used multidimensional scaling for visualization (Jones, Mair, & McNally, 2018), the distances between nodes can be meaningfully interpreted. The tuning parameter was adjusted to 0.25 as the tuning parameter of 0.5 returned an empty network with no edges. Overall, ED and OC symptoms appeared in two distinct clusters with few edges between them. Most edges represent positive correlations, with the strongest ones emerging within each of the two clusters such as times of overeating (*overeat*) and times of binging (*binge*; r = .72) and difficulty resisting obsessions *(oresist)* and difficulty resisting compulsions (*cresist*; r = .51). Few negative correlations emerged, such as between times of overeating (*overeat*) and desire for an empty stomach (*emptystom*; r = .12). The few edges that did emerge between OCD and ED symptoms were: time spent on compulsions *(ctime)* and the desire to have a flat stomach *(flatstom; r* = .07); time spent on obsessions *(otime)* and weight overevaluation *(wtover; r* = .06); difficulties controlling obsessions *(ocontrol)* and fear of loss of control *(fearloc; r* = .05); difficulties controlling obsessions *(ocontrol)* and concerns that others see one eat *(socialeat; r* = .05).

3.3 | Expected influence centrality and bridge expected influence centrality

The EI and bEI values for each node appear in Figure 2. Fear of weight gain *(fearwt)* was the most central node in terms of EI, and therefore potentially the most important node in the maintenance and perhaps the development of the network. Other high centrality nodes were restrained eating (*restraint*), interference due to obsessions (*ointerf*), weight overvaluation (*wtover*), and preoccupation with shape and weight (*occshptwt*). Nodes having scant importance in the network were over-exercising (*exercise*), laxatives use (*laxatives*), and feeling upset when having to weigh oneself (*rxweigh*).

Nodes that showed connectivity to the other clusters, respectively, and thus featured greater bridge expected influence were difficulty controlling obsessions (*ocontrol*), time spent on compulsions (*ctime*), and time spent on obsessions (*otime*). Bootstrapped centrality results, however, did not identify these symptoms to be reliably stronger bridges than any other symptom.

Overall, symptoms from the ED cluster showed higher overall connectivity whereas there were more OC symptoms bridging the distinct ED and OC clusters.

The nonthreshold sensitivity analyses yielded similar results (see Supplement). Although De Ron et al. (2019) have rightly alerted network researchers about the risks of the distorting effects of Berkson's Bias, our networks did not appreciably differ as a function of having

versus not having a symptom severity threshold as an inclusion criterion.

3.4 | Stability

The edges as well as the EI for each node in this network were sufficiently stable (correlation-stability [CS] coefficient = 0.75). Stability for the bEI, however, was low (CS = 0.244). Consequently, the reported bEI in the combined network should be interpreted cautiously, whereas the edges appear sufficiently stable for interpretations.

4 | DISCUSSION

This study is the first to apply network analysis to the comorbidity between ED and OCD at the level of symptoms in a heterogeneous ED sample. Overall, ED and OC symptoms emerged as distinct clusters with only a few moderately strong links between them. Both clusters showed cognitive symptoms to be more important ("central") than behavioral ones and to feature only few links between cognitions and behaviors.

4.1 | Functional relations between symptoms within clusters

Unsurprisingly, symptoms that featured similar components showed the strongest connections, for example, binging (overeating and experiencing loss of control while overeating) and overeating. Despite their strong resemblance, these symptoms differed in their correlation patterns with other nodes and are therefore nonredundant. The analysis of correlation patterns can be used to get a new perspective on these symptoms. For instance, whereas binging was closely associated with eating in secret, overeating was more closely associated with vomiting. As both symptoms describe increased food intake, the discriminating factor that leads to fear (and potential withdrawal) from eating in company with others appears to be the loss of control over eating.

4.2 | Central ED symptoms

Our data strongly underscore the importance of fear of weight gain for ED pathology. Consistent with network analyses on symptoms of AN (Forrest et al., 2018), BN (Forrest et al., 2018), and BED (Wang et al., 2019), fear of weight gain was most strongly connected to other cognitive symptoms, mainly the desire to have a flat stomach and the desire to lose weight. Similar patterns emerged for the preoccupation with shape and weight. According to the transdiagnostic theory of ED (Fairburn, 2008), these body image concerns result from the core pathologic overevaluation of shape and weight. Our results suggest that fear of weight gain and preoccupation with shape and weight may be an additionally useful treatment target that could be addressed by, for example, routine weighing (Waller et al., 2007) or imaginal exposure (Levinson et al., 2014). As the catastrophic fear of, for example, immediate weight gain, cannot be targeted via in vivo exposure, imaginal exposure is an opportunity to desensitize patients to the fear of being fat.

(Dietary) restraint was the only behavioral symptom that was highly central in the ED cluster with strong connections to other behavioral symptoms, namely, restrictive eating and following dieting rules. This corroborates previous research that showed high centrality for restraint in AN and BN networks (Forrest et al., 2018). With regard to these findings, ED treatment should focus on re-establishing regular eating patterns by for example, providing meal plans. In contrast to Fairburn's (2008) transdiagnostic model, however, dietary restraint was not connected to binge eating. Given that previous research indicates a rather time-sensitive association between dietary restraint and binging (e.g., Zunker et al., 2011), future research using multiple time points and matched ED and OCD questionnaires is needed to investigate this relationship more closely.

Overall, this ED network shows that cognitive symptoms are highly interconnected as are behavioral symptoms. This is consistent with previous network analyses investigating symptomatic interplay in AN and BN (Forrest et al., 2018) and BED (Wang et al., 2019). Considering that the dominant rationale in cognitive behavioral therapy is to modify cognitions in the service of promoting positive behavioral change, this is especially striking. This study underscores the importance of examining links between cognitions instead of focusing exclusively on potential links to behaviors.

4.3 | Central OCD symptoms

Interference due to obsessions clearly emerged as the most central symptom in the OCD cluster. This dovetails with research showing that the severity of obsessions is a major contributor to the life interference OCD causes (Salkovskis, 1985). Given that compulsions arise to reduce obsession-induced distress, the dominant influence of obsession severity within the scope of OCD appears plausible. Moreover, negative cognitive appraisals amplify the emotional pain of obsessions (Salkovskis, 1985) and attempts to suppress obsessions often paradoxically increase their frequency (Salkovskis & Kirk, 1997).

We found similar connections between obsession-related interference and other OCD features as did McNally et al. (2017) in a Bayesian network analysis involving adults with OCD. However, a similar analysis involving adolescents with OCD did not (Jones, Mair, Riemann, Mugno, & McNally, 2018).

4.4 | Symptomatic interplay between OCD and ED

Both, the ED and the OCD cluster, emphasize the importance of cognitive symptoms (i.e., high EI centrality values). Edges between clusters mainly emerged between the difficulty controlling obsessions and

different ED cognitions (weight overvaluation, fear of loss of control, concerns that others see one eat). Overall, this dovetails with findings stressing the importance of intrusive cognitions and the diminished control over them in both disorders. It is reasonable that someone who experiences difficulties controlling obsessions (e.g., fear of contamination) encounters similar difficulties when controlling intense preoccupations with shape, weight, or food.

Our analyses implicated difficulty controlling obsessions, difficulty controlling compulsions, and time spent on obsessions as candidate bridge symptoms. Although these three features of OCD had the highest bEI values, our analyses indicated that these values were not reliably greater than the bEI values for the other features of OCD. Moreover, their low CS values raise questions about their stability. Hence, although theory implies that these three features of OCD are suitable targets for preventing comorbidity, insufficient reliability and stability temper such clinical implications.

As the EDE-Q and the Y-BOCS-SR endorse exclusively cognitive and behavioral symptoms directly related to ED and OCD, we attribute the lack of stable bridge symptoms and strong associations between clusters to unmeasured vulnerability variables. Research indicates that patients with lifetime comorbidities of OCD and ED share vulnerabilities (e.g., coping styles, personality traits, neurobiological variables) that, depending on specific external factors, foster either the development of ED, OCD, or both. These variables may not necessarily be symptoms that serve as criteria for diagnosis but rather as transdiagnostic risk factors. We hypothesize that, given the vast amount of research on shared personality features, such personality variables may be better candidates for bridges than disorder-specific cognitions and behaviors. Traditionally, personality features have been treated as the mere latent cause of behavior. However, by identifying affective, cognitive, and behavioral aspects of personality features, for example, perfectionism, personality can be reconceptualized as nodes that can figure in a network (Cramer et al., 2012).

4.5 | Limitations

This network investigation is marked by three limitations. First, we used cross sectional data which does not allow to model the effect of time and thus causality. Third-variable effects, such as shared genetic vulnerabilities (Yilmaz et al., 2019), are one potential explanation for the associations detected between disorders in our analysis. Longitudinal and experimental work is needed to see if fear of weight gain and restraint have causal effects on ED etiology and the development of other symptoms. Second, the combined network was likely underpowered. Claims about the bridge expected influence can only be made with great caution as the measure did not yield sufficient stability. Furthermore, the tuning parameter needed adjustment due to the potential power problem, which heightens sensitivity but lowers specificity (Epskamp & Fried, 2018). Studies using greater sample sizes are needed to obtain more robust results. Third, the Y-BOCS-SR and EDE-Q target different time periods, with the EDE-Q asking for reports over the last 28 days and the Y-BOCS-SR only about the last week. Future work is needed that uses matched questionnaires in order to control for shifts in pathology over time.

5 | CONCLUSION

In this first network study on ED-OCD comorbidity, we included a heterogeneous treatment-seeking sample with clinically relevant ED and OCD symptoms and performed up-to-date network computation by using EI as centrality measure and MDS for visualization. Consistent with previous studies limited by small samples and less precise metrics, ours underscores the importance of cognitive symptoms to both syndromes. Among ED symptoms, fear of weight gain played a key role whereas interference due to obsessions was especially important among the OC symptoms. Surprisingly, the many similarities between EDs and OCD did not emerge as strong bridges between syndromes which speak in favor of conceptualizing ED and OCD as distinct disorders. Future work is needed to investigate causal links and to include personality variables that potentially help explain the comorbid occurrence of ED and OCD on a functional level.

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CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

Data Availability: The data was collected within the Rogers Behavioral Health system. The raw data is available on https://osf.io/f4y92/?view_only=9c5c6e3ba532476a9910c2898efbee1c.

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