Research paper

Co-occurring depression and obsessive-compulsive disorder: A dimensional network approach

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A B S T R A C T

Background: Depressive and obsessive-compulsive (OCD) symptoms often co-occur and a number of possible explanations for this co-occurrence have been explored, including shared biological and psychosocial risk factors. Network approaches have offered a novel hypothesis for the link between depression and OCD: functional inter-relationships across the symptoms of these conditions. The few network studies in this area have relied largely on item, rather than process-level constructs, and have not examined relationships dimensionally.
Methods: Network analytic methods were applied to data from 463 treatment-seeking adults with OCD. Patients completed self-report measures of OCD and depression. Factor analysis was used to derive processes (i.e., nodes) to include in the network. Networks were computed, and centrality, bridge, and stability statistics examined.
Results: Networks showed positive relations among specific OCD and depressive symptoms. Obsessions (particularly repugnant thoughts), negative affectivity, and cognitive-somatic changes (e.g., difficulty concentrating) were central to the network. Unique relations were observed between symmetry OCD symptoms and cognitive-somatic changes. No direct link between harm-related OCD symptoms and depression was observed.
Conclusions: Our results bring together prior findings, suggesting that both negative affective and psychomotor changes are important to consider in examining the relationship between OCD and depression. Increased consideration of heterogeneity in the content of OCD symptoms is key to improving clinical conceptualizations, particularly when considering the co-occurrence of OCD with other disorders.

1. Introduction

Obsessive-compulsive disorder (OCD) is a heterogeneous disorder characterized by unwanted intrusive thoughts (i.e., obsessions) that provoke distress, and in turn, are resisted with rituals and avoidance (American Psychiatric Association, 2013). OCD can be highly impairing and result in significant disability and reduced quality of life (e.g., Albert et al., 2018; Meier et al., 2016; Schwartzman et al., 2017). Moreover, OCD impacts approximately 2 % of adults globally, translating to upwards of 150 million individuals (Ruscio et al., 2010). There is a clear need to understand factors that complicate the course and severity of OCD in order to mitigate its widely experienced, deleterious effects.

A historical barrier to understanding the phenomenology of OCD has been the heterogeneity observed in its presentation. Accordingly, over the past two decades, research has sought to identify OCD subtypes and

the clinical correlates of specific symptom dimensions (e.g., Cervin et al., 2021; Katerberg et al., 2010; Leckman et al., 2010; Williams et al., 2013b). While various models exist, one prevailing approach includes four core dimensions of OCD symptoms, with obsessions centered on: (a) contamination and germs, (b) personal responsibility for causing harm, (c) repugnant or taboo thoughts, and (d) the need for symmetry (e.g., McKay et al., 2004; Schulze et al., 2018). Along each of these obsessional themes, specific compulsions and avoidance behaviors often present, including (a) washing and cleaning, (b) checking and reassurance-seeking, (c) efforts to suppress or avoid unwanted thoughts, and (d) arranging, counting, and ordering rituals, respectively. Accumulating research supports the utility of examining OCD using a dimensional framework (e.g., Leckman et al., 2010; Schulze et al., 2018; Schwartzman et al., 2017; Williams et al., 2013b), with symptom dimensions demonstrating unique relationships to risk and maintaining factors, as

well as clinical outcomes.

Complicating the clinical picture, the majority of individuals with OCD present with co-occurring psychological conditions (e.g., Lochner et al., 2014; Torres et al., 2016). Depression is most commonly observed, with major depressive disorder (MDD) affecting more than a third of individuals with OCD (Quarantini et al., 2011). In addition to its prevalence, severe depression has been shown to predict poorer prognosis in OCD (e.g., Jakubovski et al., 2013). Notably, in regard to the symptom dimensions described above, depressive symptoms have consistently been shown to correlate most strongly with repugnant thoughts (e.g., Torres et al., 2011, 2016; Torresan et al., 2013), with weaker to absent correlations observed between depression and other symptom dimensions of OCD.

Taken together, these findings have elicited a strong rationale for understanding the phenomenology of comorbid depression and OCD. Accordingly, a number of investigators have sought to identify shared biopsychosocial risk factors for both conditions, conceptualizing the co-occurrence of OCD and depression as the result of shared genetic, physiological, psychological, or social factors (Abramowitz et al., 2007; Cardoner et al., 2007; Labad et al., 2018; Miegel et al., 2019; Yap et al., 2012). Yet, these conceptualizations have failed to fully account for the relationship between OCD and depression. Several studies suggest OCD often predates the onset of MDD (e.g., Bolhuis et al., 2014; Rickelt et al., 2016). Moreover, effective treatment for OCD symptoms can also produce significant reductions in depressive symptom severity (Zandberg et al., 2015). In line with these findings, researchers have hypothesized that functional relationships among OCD and depressive symptoms may also contribute to their co-occurrence (McNally et al., 2017). For example, obsessions may lead to significant distress and avoidance, thereby reducing opportunities for social reward and increasing risk for depression.

This latter conceptualization aligns with the *network theory of psychopathology*, which proposes that comorbidity results from functional relationships among symptoms that span diagnostic boundaries (Borsboom, 2017; Borsboom and Cramer, 2013; Cramer et al., 2010). In the case of OCD and depression, symptom-level relations are not clearly captured by overlapping diagnostic criteria. Still, potential links are evident. For example, obsessions and compulsions have long demonstrated associations with negative affect (Purdon and Clark, 2001; Ricciardi and McNally, 1995; Rowa et al., 2005; Sutherland et al., 1982). Additionally, there is evidence for a bidirectional relationship between OCD symptoms and sleep disturbance, with compulsions linked to delayed bedtimes and poor sleep to difficulties with repetitive negative thoughts (e.g., Cox et al., 2018a, 2018b; Hellberg et al., 2019; Nota et al., 2015a, 2015b). Further, cognitive difficulties (e.g., memory impairments) can be exacerbated by intrusions, anxiety, and checking behaviors (e.g., Hermans et al., 2008; Radomsky et al., 2006), and the impairment caused by OCD symptoms may decrease interest or engagement in valued activities (e.g., Blakey et al., 2019; Wheaton and Gallina, 2019).

With such relationships in mind, four studies have applied a network approach to examine the symptom-level associations that link OCD and depressive symptoms (Cervin et al., 2020; Jones et al., 2018; Klein et al., 2020; McNally et al., 2017). Findings to date suggest that distress and interference related to obsessions are most closely associated with the severity of depression. In addition, McNally et al. (2017) demonstrated that the link between obsessional distress and negative affectivity in particular facilitated downstream activation of other depressive symptomatology. In addition, Klein and colleagues found that low energy and resistance of compulsions contributed to the coherence of OCD and depressive symptoms. Building on these findings, Jones et al. (2018) found that concentration difficulties were central to the links among depressive and OCD symptoms in adolescents. Most recently, Cervin et al. (2020) investigated the link between OCD, anxiety and depressive symptoms in children and adolescents. They reported an association between the frequency and lack of control over obsessions with feelings of perceived failure related to depressive symptomatology. Interestingly, the authors examined not only item-level relations, but also the relationships among OCD symptom subscales (e.g., washing, checking) and overall depression and anxiety (e.g., panic, social) severity. In this regard, the authors found that obsessing, ordering, and doubting/checking were particularly related to depressive symptomatology, and that anxiety severity mediated the link between OCD and depressive symptoms.

Although these studies have afforded insights into factors that may influence the co-occurrence of OCD and depression, there remains room for refinement. For example, most prior network studies have used individual scale items (e.g., Yale Brown Obsessive-Compulsive Scale [YBOCS]) as focal variables (i.e., nodes). Scale items, such as the time occupied by obsessions, do not necessarily correspond to the symptom-level relationships central to network theory and clinical practice (Guyon et al., 2017). In this regard, recent work has suggested that integrating traditional psychometric techniques (e.g., factor analyses) into network research may aid in generating nodes that are more robust and interpretable (Epskamp et al., 2017b; Hallquist et al., 2019; Smith et al., 2018; Wright et al., 2015). In addition, there is a need to better capture symptom heterogeneity in OCD within the network literature. For example, repugnant thoughts are consistently associated with depression, while contamination is not (e.g., Buchholz et al., 2019). Work by Cervin et al. (2020) provided preliminary support for a dimensional, symptom-level approach; yet, the OCD dimensions used were different from the empirically supported subtypes discussed above (e.g., Abramowitz et al., 2010). Further, focal constructs aligned more closely with diagnostic categories than functional symptom-level processes (Borsboom, 2017).

The present study therefore aims to further our understanding of links between OCD and depressive symptoms by using a network approach in a large ($N = 463$) sample of treatment-seeking individuals with OCD. First, we will examine the associations among OCD and depressive symptoms using process-level factors (e.g., obsession severity), rather than an item-level approach (e.g., resistance associated with obsessions). We hypothesize that both obsessions and negative affect would emerge as central bridge symptoms in the network; in other words, we expect that these two symptoms are highly influential in linking the symptoms of OCD and depression. Second, we will investigate the associations among the four OCD symptom dimensions described above and depressive symptoms. We hypothesize that repugnant obsessions will be most closely associated with depressive symptomatology. Moreover, we expect that the association between repugnant thoughts and negative affect will emerge as a primary pathway through which OCD symptom dimensions connect to broader depressive symptoms.

2. Methods

2.1. Participants

Patients seeking treatment for OCD ($N = 463$) within a network of specialized treatment programs were included in the study if they were diagnosed with OCD and provided informed consent during the clinic intake process (see *Procedure*). The presence of a depressive disorder was not required for inclusion. Still, approximately 38.8 % ($n = 181$) of the sample met criteria for a current depressive episode ($QIDS > 13$), with 90.1 % indicating at least mild current depressive symptoms ($QIDS > 5$) and more than half reporting moderate to severe depressive symptoms ($QIDS > 10$; e.g., Lamoureux et al., 2010). Regarding current OCD severity, the majority of participants (53.2 %) indicated moderate symptoms, with 36.9 % reporting moderate-severe, 7.9 % reporting mild, and 1.9 % indicating severe symptoms (Storch et al., 2015).

Participants were on average 29.69 years old ($SD = 11.13$, $Range = 18–69$). The majority of the sample was single (65%), and evenly divided in terms of sex (51% male). Regarding race and ethnicity,

participants self-identified as: American Indian or Alaska Native (0.4%), Asian (2.2%), Black or African American (2.2%), Native Hawaiian or Pacific Islander (0.2%), or White (78%). In addition, 17 % (*n* = 79) did not provide information about their racial identity. Participants were of varying education levels: 0.2% completed less than high school, 2.6% completed some high school, 9.1 % had a high school diploma or equivalent (i.e., GED), 30.7 % completed some college, 4.5 % completed vocational training or an Associate's degree, 22.2 % completed a Bachelor's degree, 2.4 % completed a postgraduate degree, 2.8 % completed some graduate school coursework, 9.9 % completed a Master's degree, 1.1 % completed a Doctoral degree, and 14.5 % did not provide information on their educational background.

2.2. Procedure

During the intake process, all participants completed a clinical interview with a psychiatrist to determine the presence of OCD, and any other co-occurring diagnoses. The interviewer presented assessment data to another expert clinician (i.e., the site director or senior clinician). Formal interrater reliability was not assessed; still, only individuals for whom both interviewers agreed on OCD as a principal diagnosis were included. Participants who met criteria for OCD and provided informed consent to participate were included in the study and provided with a packet of self-report measures to complete prior to treatment initiation. All procedures were approved by the institutional ethics committees.

2.3. Measures

2.3.1. Quick inventory of depressive symptomology (QIDS)

The QIDS is a 16-item, self-reported assessment of depression severity (Rush et al., 2003). Items assess nine core symptoms of depression, including depressed mood, loss of interest, difficulty concentrating, guilt, suicidality, decreased energy, sleep difficulties, changes in weight and/or appetite, and psychomotor changes. Individuals report the severity of each symptom over the past week (*Range*: 0–3). Higher QIDS total scores indicate more severe depressive symptoms. Psychometric research has supported the reliability and validity of the QIDS (Trivedi et al., 2004).

2.3.2. Yale-Brown obsessive-compulsive scale – self report (YBOCS)

The YBOCS (Goodman et al., 1989) is a widely used, validated measure of OCD. It includes a 10-item symptom severity scale that addresses the time, interference, distress, resistance, and control associated with obsessions and compulsions over the past month. Items are summed to compute two subscale scores to reflect the severity of obsessions and compulsions. The self-report version demonstrates strong psychometric properties (du Mortier et al., 2019; Federici et al., 2010; Steketee et al., 1996).

2.3.3. Dimensional obsessive-compulsive scale (DOCS)

The DOCS is a 20-item self-report measure that assess the four, empirically derived OCD symptom dimensions: (a) contamination, (b) responsibility for harm (c) repugnant thoughts, and (d) symmetry and ordering (Abramowitz et al., 2010). Respondents read examples of characteristic obsessions and compulsions for each dimension, and then rate the time occupied by these symptoms, severity of avoidance behavior, distress, functional interference, and difficulty controlling and resisting compulsions. Items are summed to compute four subscale scores, which address the four symptom dimensions of OCD outlined above. The DOCS evidences excellent psychometric properties (Abramowitz et al., 2010).

2.4. Data analytic plan

Data were cleaned and descriptive statistics computed for demographic variables and study measures. All analyses were conducted in

R, version 3.6.3. Procedures were consistent with recent published tutorials (Epskamp et al., 2017a; Epskamp and Fried, 2018) and empirical papers (Levinson et al., 2018; McNally et al., 2017).

2.4.1. Factor reduction

An exploratory factor analysis (EFA) was conducted to reduce the QIDS, given limited data on its factor structure in samples of patients with OCD. The R package *EFAtools* was used to conduct factor analyses (Steiner and Grieder, 2020). First, to determine the number of factors to use, the scree plot was examined. Various methods are available to facilitate factor extraction (e.g., Auerswald and Moshagen, 2019). Using *EFAtools*, the number of recommended factors to retain can be examined across multiple methods (e.g., Kaiser-Guttman Criterion [KGC], sequential χ2 model tests [SMT], the Hull method, Empirical Kaiser Criterion [EKC]). Given prior findings by Auerswald and Moshagen (2019), the factor loading recommended by sequential χ2 model tests (SMT) was prioritized over other factor solutions. Across approaches, the recommended number of factors to retain ranged from 1 to 7. The lower bound of root mean square error of approximation (RMSEA) 90 % confidence interval from SMT suggested retaining 4 factors, which converged with the Kaiser-Guttman criterion with Principal Components Analysis (PCA) recommendation. Only components with eigenvalues over 1 were included in the final model. Factor loadings were then estimated using principal axis factoring and Promax rotation. Eigenvalues <0.3 were not excluded. The factor solution was examined to ensure conceptual clarity before proceeding with further analysis. Confirmatory factor analysis was conducted on the YBOCS and DOCS subscales to ensure adequate fit in the present sample, given the factor structure of these measures has been extensively examined in patient samples with OCD.

2.4.2. Graphical LASSO

For additional information on network estimation see Epskamp and Fried, 2018. Graphical LASSO models were generated with the R package *qgraph* (Epskamp et al., 2012, 2021) using *EBICglasso* (Friedman et al., 2008, 2014). The graphical LASSO algorithm maps an undirected Gaussian graphical model. Nodes represent variables in the model, and edges between nodes reflect regularized partial correlations between nodes. Likely trivial effects are reduced to 0 and removed from the final network structure. Two networks were computed using these methods: (1) a network to examine obsessive-compulsive (YBOCS) and depressive symptom (QIDS) relationships, and (2) a network of OCD dimensions (DOCS) and depressive symptoms (QIDS).

2.4.3. Centrality statistics

Centrality parameters for each network were calculated (Epskamp et al., 2017a; Epskamp and Fried, 2018). Centrality metrices indicate the relative importance of symptoms within the network. Conceptually, this can identify factors of chief importance to the coherence of symptoms. Three metrics are often calculated: strength, betweenness, and closeness. Strength represents the aggregate sum of partial correlations (i.e., edges) connected to a given node. Betweenness represents the number of instances in which a node provides the shortest path between two other nodes. Finally, closeness reflects the average of the shortest paths between a node and all others in the network.

2.4.4. Network stability

Recent literature suggests examination of the stability of networks is critical (Epskamp et al., 2017a). The R package *bootnet* was used to calculate stability estimates (Epskamp et al., 2015). A cut-off of 0.50 or high has been suggested for assuming adequate stability. Lower stability estimates suggest network parameter estimates are sensitive to sampling changes.

2.4.5. Bridge statistics

Of particular relevance to the present study were "bridge" symptoms,

which play a central role in linking subsets of symptoms (Jones et al., 2019). Bridge centrality indices were computed using the *NetworkTools* package (Jones and Jones, 2020). Building on centrality indices described above, bridge statistics quantify relationships among symptoms within a community framework. Communities of symptoms can be defined empirically (i.e., by the data) or conceptually (i.e., based upon diagnoses). In the present study, we defined communities conceptually to reflect OCD and depressive symptoms.

3. Results

3.1. Factor reduction

The KMO statistic for the EFA for the QIDS suggested adequate sampling (0.81) and the Bartlett's test was significant ($\chi^2(120) = 1564.27, p < .001$), supporting the appropriateness of factor analysis. As noted above, a four-factor solution was used (See Supplemental Table 1). The item assessing hypersomnia (#4) failed to adequately load onto any factor; thus, the four-factor solution consisted of 15 items. Factors were named to characterize key symptom dimensions, including: (a) Cognitive/Somatic Changes; (b) Negative Affect, (c) Appetite/Weight Changes, and (d) Insomnia. Items were summed to compute subscale scores. Confirmatory factor analysis was conducted for the YBOCS and DOCS. Findings supported standard scoring procedures (Abramowitz et al., 2010; Goodman et al., 1989). Descriptive statistics are reported in Supplemental Table 2.

3.2. Network stability

Stability of edge weights were good (*coefficients* > 0.67) with moderate confidence intervals across estimates (see Appendix). Centrality stability coefficients ranged from unacceptable to good in Network 1 (*Closeness* = 0.67, *Strength* = 0.75, *Betweenness* = 0.05) and Network 2 (*Closeness* = 0.05, *Strength* = 0.67, *Betweenness* = 0.05). Strength emerged as the most stable metric across networks. For Network 1, the stability of bridge strength (*coefficient* = 0.67) and betweenness

(*coefficient* = 0.51) estimates were acceptable, while the closeness estimate was not (*coefficient* = 0.05). For network 2, only bridge betweenness estimates were adequately stable (*coefficient* = 0.75).

3.3. Symptom networks

3.3.1. Network 1: QIDS and YBOCS

The first network examined interrelations between the YBOCS and QIDS subscales (see Fig. 1). In the network depicted, green lines (i.e., edges) represent significant (*p*'s < .05), positive partial correlations among nodes. The thickness of an edge reflects the magnitude of the association. Several significant interrelationships were detected. A strong and significant association between YBOCS-Obsessions and YBOCS-Compulsions was observed, as well as between the QIDS-Cognitive/Somatic Changes and QIDS-Negative Affect. YBOCS-Obsessions was positively and significantly associated with QIDS-Negative Affect as well as QIDS-Cognitive/Somatic Changes. YBOCS-Compulsions was weakly, albeit significantly, associated with QIDS-Cognitive/Somatic Changes.

Symptoms with the highest strength centrality were: YBOCS-Obsessions, QIDS-Negative Affect, and QIDS-Cognitive/Somatic Changes (see Fig. 2). Significant difference testing revealed the strength of did not significantly differ across these symptoms; however, they were more central to the network than most others in the model (*p*'s < .05; See Appendix A). The links between YBOCS-Obsessions and YBOCS-Compulsions, as well as that between QIDS-Negative Affect and QIDS-Cognitive/Somatic Changes, were significantly stronger than all others in the network. Finally, the association between YBOCS-Obsessions and QIDS-Negative Affect was significantly stronger than any other associations in the network. Similarly, the bridge symptoms with strongest centrality were: YBOCS-Obsessions (*Strength* = 0.36), QIDS-Negative Affect (*Strength* = 0.24), and QIDS-Cognitive/Somatic Changes (*Strength* = 0.22; see Appendix).

3.3.2. Network 2: QIDS factors and DOCS dimensions

Next, a network was generated for the QIDS and DOCS subscales. As

Depression
- Ins: Insomnia
- NgA: Negative Affect
- W/A: Weight/Appetite
- C/S: Cognitive/Somatic

OCS
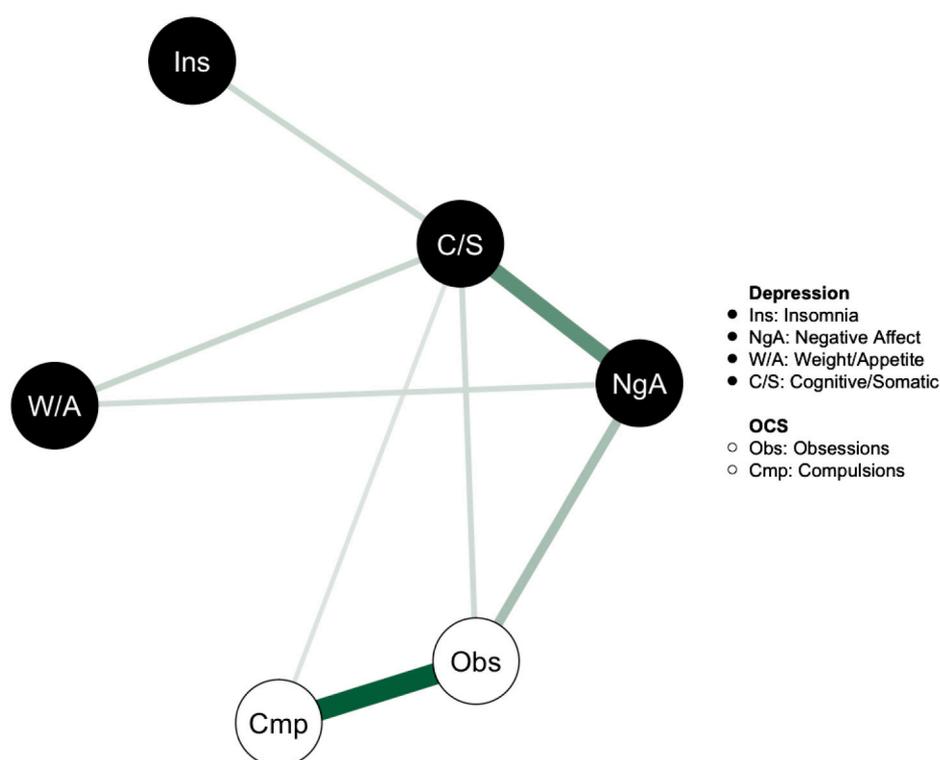- Obs: Obsessions
- Cmp: Compulsions

Fig. 1. Network 1: inter-symptom relationships among YBOCS and QIDS factors.
Note. Network plot shows the edges (i.e., associations) among nodes (i.e., symptoms) for depression and obsessive-compulsive disorder symptoms in a large sample (*N* = 463) of treatment-seeking patients. Green lines indicate positive associations among factors, while red lines indicate negative associations. The thickness of the line corresponds to the magnitude of the association between nodes (i.e., symptoms), with thicker lines representing stronger associations. Only associations that met the significance threshold are included in the plot. Depression subscales were derived from the Quick Inventory of Depressive Symptoms – 16-item version (QIDS). Obsessive-compulsive symptom subscales were derived from the Yale-Brown Obsessive-Compulsive Scale (YBOCS). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
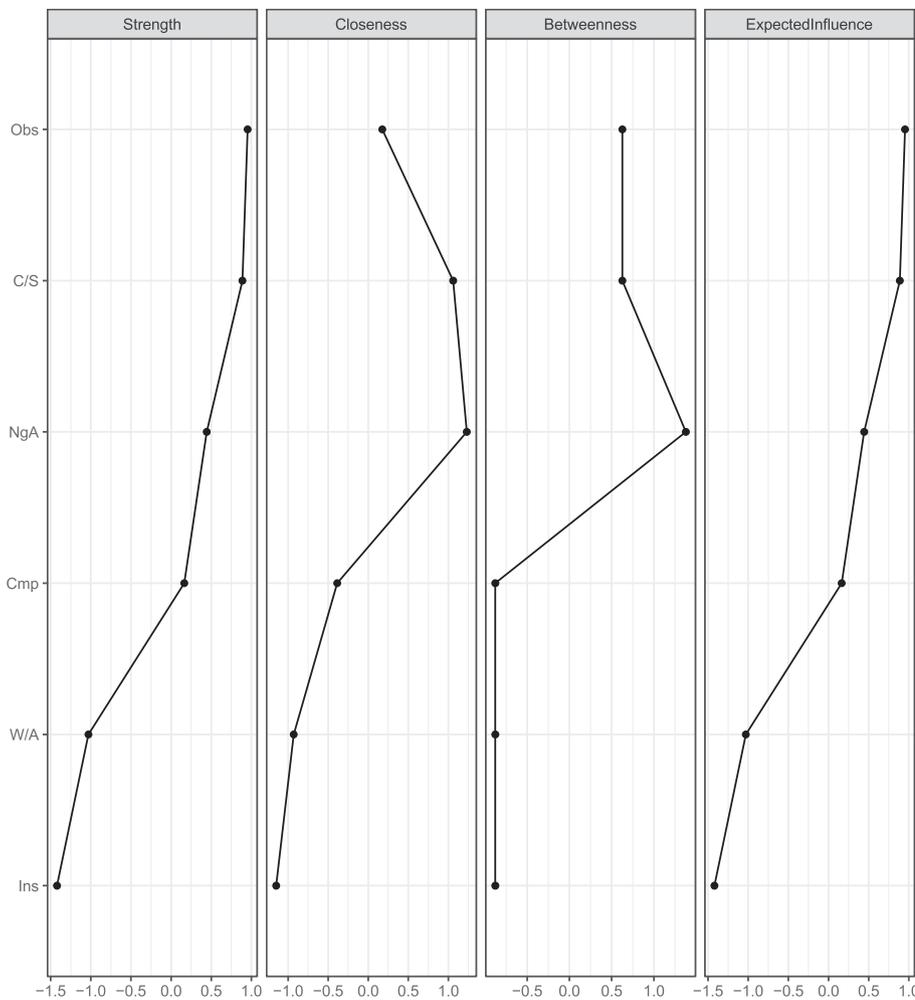
Fig. 2. Network 1: centrality plot.
Note. Centrality estimates are depicted for Network 1, examining the associations between the YBOCS and QIDS factors. Centrality scores are standardized (range = −1 to 1, mean = 0). Higher scores indicated greater centrality in the network. YBOCS = Yale-Brown Obsessive Compulsive Scale, Obs = YBOCS Obsessions, Cmp = YBOCS compulsions, C/S = QIDS Cognitive Somatic Changes, NgA = QIDS Negative Affective, W/A = QIDS Weight/Appetite Changes, Ins = QIDS Insomnia. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
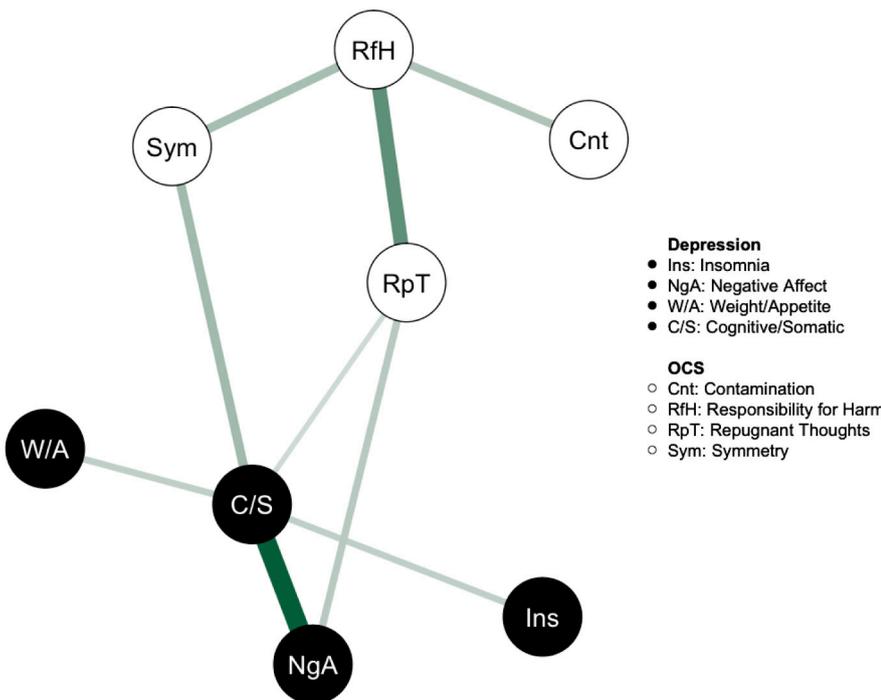

Fig. 3. Network 2: inter-symptom relationships among DOCS and QIDS factors.
Note. Network plot shows the edges (i.e., associations) among nodes (i.e., symptoms) for depression and obsessive-compulsive symptom dimensions in a large sample (*N* = 463) of treatment-seeking patients. Green lines indicate positive associations among factors, while red lines indicate negative associations. The thickness of the line corresponds to the magnitude of the association between nodes (i.e., symptoms), with thicker lines representing stronger associations. Only associations that met the significance threshold are included in the plot. Depression subscales were derived from the Quick Inventory of Depressive Symptoms – 16-item version (QIDS). Obsessive-compulsive symptom dimensions were derived from the Dimensional Obsessive-Compulsive Scale (DOCS). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

can be seen in Fig. 3, results again revealed several significant associations. The DOCS-Responsibility for Harm and DOCS-Repugnant Thoughts subscales were strongly and positively associated. The relationship between QIDS-Cognitive/Somatic Changes and QIDS-Negative Affect remained positive and significant. Positive associations also were observed between: (a) DOCS-Symmetry and QIDS-Cognitive/Somatic Changes, (b) DOCS-Repugnant Thoughts and QIDS-Cognitive/Somatic Changes, and (c) DOCS-Repugnant Thoughts and QIDS-Negative Affect. No direct association was found between DOCS-Responsibility for Harm and any QIDS factors. DOCS-Responsibility for Harm was primarily connected to QIDS factors through its association with DOCS-Repugnant Thoughts.

Symptoms with the highest strength centrality were: QIDS-Cognitive/Somatic Changes, DOCS-Responsibility for Harm, QIDS-Negative Affect, and DOCS-Repugnant Thoughts (see Fig. 4). Significant difference testing revealed that the strength estimates between these symptoms did not significantly differ from one another (See Appendix B). The associations between (1) QIDS-Negative Affect and QIDS-Cognitive/Somatic Changes, and (2) DOCS-Responsibility for Harm and

DOCS-Repugnant Thoughts, were significantly stronger than all other edges in the network. Regarding bridge symptoms, the association between DOCS-Symmetry and QIDS-Cognitive/Somatic Changes, and between DOCS-Repugnant Thoughts and QIDS-Negative Affect, were stronger than others in the network (See Appendix B). Accordingly, the symptoms with the strongest bridge strength centrality were: QIDS-Cognitive/Somatic Changes (*Strength* = 0.31), DOCS-Repugnant Thoughts (*Strength* = 0.27), DOCS-Symmetry (*Strength* = 0.20), and QIDS-Negative Affect (*Strength* = 0.15).

4. Discussion

Individuals with OCD frequently experience co-occurring depressive symptoms, and this comorbidity has been associated with more severe symptomatology, functional impairment, and an attenuated response to treatment. Research is needed to better understand this symptom pattern in order to inform our conceptual models and refine treatment for this patient population. Network theory offers a novel opportunity to expand our understanding of the mechanisms of co-occurring
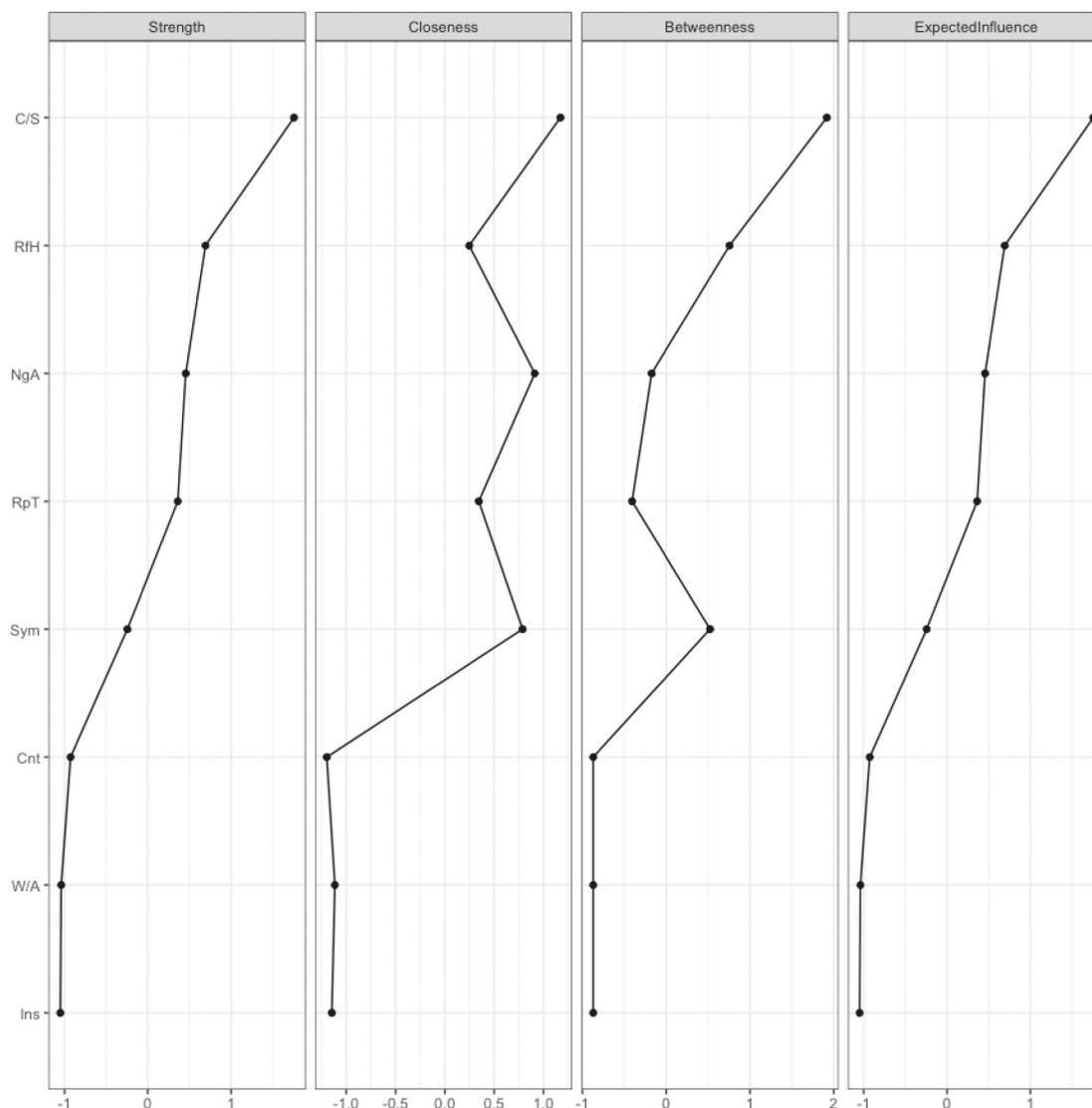

Fig. 4. Network 2: centrality plot.
Note. Centrality estimates are depicted for Network 2, examining the associations between the YBOCS and DOCS factors. Centrality scores are standardized (range = −1 to 1, mean = 0). Higher scores indicated greater centrality in the network. DOCS = Dimensional Obsessive-Compulsive Scale, RfH = DOCS Responsibility for Harm, RpT = DOCS Repugnant Thoughts, Sym = DOCS Symmetry/Ordering, Cnt = DOCS Contamination, C/S = QIDS Cognitive Somatic Changes, NgA = QIDS Negative Affective, W/A = QIDS Weight/Appetite Changes, Ins = QIDS Insomnia.

psychological syndromes. Many studies leveraging network tools, including the present investigation, have relied on cross-sectional data (Cervin et al., 2020; Jones et al., 2018; Levinson et al., 2018; McNally et al., 2017). While the limitations of this approach must be considered (see: Robinaugh et al., 2020), the insights afforded by cross-sectional networks can inform the refinement of our conceptual models and the design of subsequent studies, including longitudinal designs to further examine the causal nature of implicated processes.

In this context, the present study extends the network literature by using factor analysis to derive conceptually meaningful OCD and depressive symptom dimensions. As a result of this approach, the processes included in our analyses mirrored core functional symptoms of OCD (e.g., obsessions, compulsions) and depression (e.g., negative affect, sleep disturbance). In addition, we incorporated a measure of theme-based OCD symptom dimensions (e.g., contamination, responsibility for harm, repugnant thoughts, symmetry), given evidence that the associations among OCD symptoms and depression vary systematically depending on the thematic content of intrusions.

Our results were generally consistent with our hypotheses. First, we found that the link between obsessions and negative affect was central to the relationship between OCD and depressive symptoms; in addition, cognitive-somatic changes were influential to the network. These results bring together findings previously observed by McNally et al. (2017) and Klein et al. (2020). For example, McNally and colleagues found a central link between obsessional distress and sadness, while Klein and colleagues observed a central association between obsessional distress and low energy. In our case, these specific symptoms were subsumed within the broader nodes of *negative affect* and *cognitive-somatic changes*. The use of item-level associations in prior studies may have obscured the simultaneous detection of these effects, due to the decreased power and potential for conceptual redundancy across scale items. Taken together, our findings suggest both of these pathways are necessary to consider in future investigations of co-occurring OCD and depression. Moreover, results suggest that the integration of factor analytic methods into network analysis may afford more parsimonious solutions, compared to often used item-level approaches (Guyon et al., 2017).

Importantly, the directional nature of these associations necessitates further testing. For example, it is possible that the distressing, ego-dystonic nature of obsessions triggers low mood, guilt, and shame (Moulding et al., 2014; Rowa et al., 2005), and thus, increased obsession severity leads to increased negative affect. However, there is also evidence that negative affective states can lead to more frequent, distressing, or difficult to dismiss intrusions (Cranston, 2013; Reynolds and Salkovskis, 1992; Sutherland et al., 1982). Moreover, individual differences warrant consideration; that is, the direction of this relationship may vary across individuals, as has been shown in network research in related areas (Fisher et al., 2017; Reeves and Fisher, 2020). Accordingly, further efforts to expand this work with intensive longitudinal designs may offer more precise, person-centered insights (e.g., Piccirillo et al., 2019). In turn, this line of work may afford new insights that can guide personalized approaches to treatment matching and selection (Rubel et al., 2018). For example, individual differences in the centrality of obsessions, negative affectivity, and cognitive-somatic changes may be used to inform the sequencing of effective therapeutic techniques (e.g., exposure, behavioral activation, cognitive defusion) in line with emerging process-based approaches to psychotherapy (e.g., Hayes et al., 2020).

The present study is the first to use network analysis to examine associations between depressive symptoms and these four theme-based OCD symptom dimensions. Consistent with our second hypothesis, repugnant obsessions were most closely linked to depressive symptoms, with particularly close associations observed between these symptoms and both negative affect and psychomotor changes. These results add more specificity to prior findings that, among the array of OCD symptom themes, repugnant obsessions are most strongly associated with depression. The prominent role of negative affect observed aligns with

hypotheses that the link between depression and this dimension of OCD is driven by the highly distressing and ego-dystonic nature of repugnant thoughts (Brakoulias et al., 2013; Moulding et al., 2014; Rowa et al., 2005). Yet, we also observed a strong association between repugnant thoughts and cognitive-somatic changes. Prior research has shown that individuals with violent, sexual, or taboo intrusions frequently engage in covert mental strategies to manage distress (Brakoulias et al., 2013). The excess use of such neutralization strategies may result in pronounced cognitive interference, and in turn, explain the strong association between this symptom dimension and cognitive-somatic changes (e.g., difficulty concentrating). Moreover, given the autogenous nature of repugnant obsessions (Lee and Kwon, 2003), individuals with repugnant obsessions often struggle to avoid fear cues. As such, the uncontrollable occurrence of these intrusions may lead to more prolonged psychomotor effects. These hypotheses necessitate direct testing in experimental and longitudinal designs.

Unexpectedly, we found that symmetry-related OCD symptoms were also associated with cognitive-somatic changes. While we did not hypothesize this result, it is consistent with the finding that, relative to other presentations of OCD, symmetry-related symptoms are associated with reduced neuropsychological performance (Bragdon et al., 2018). Moreover, Vellozo et al. (2021) found that the symmetry dimension of OCD was associated with more pervasive and severe sensory phenomena (e.g., tactile, muscular, skeletal-visceral sensations) and more severe depressive symptoms. It is possible that the phenomena of not-just-right experiences (NJREs), which is commonly associated with symmetry-related OCD, also contributes to greater sensitivity to or severity of somatic changes underlying this relationship (Ben-Sasson et al., 2017; Coles et al., 2005). Research in this area is sparce and data are needed to better understand mechanisms that tie symmetry OCD to depressive symptoms.

In contrast to our hypotheses, OCD symptoms concerning responsibility for harm were not directly associated with depressive symptoms. Instead, findings suggested the relationship between these symptoms was facilitated by a shared association with repugnant thoughts. These effects were surprising, given prior research has consistently shown a significant association between harm-related OCD and depression (e.g., Jacoby et al., 2014; Yap et al., 2018). Interestingly, however, prior studies have typically examined the independent associations among OCD symptom dimensions and depression. In contrast, one study examined the unique predictive utility of these dimensions in explaining concurrent depression in a sample of patients with OCD (Buchholz et al., 2019). The results suggested that harm-related OCD severity did not significantly predict depression severity, after accounting for the effects of repugnant OCD symptoms. Similarly, our study considered OCD symptom dimensions simultaneously. Thus, our results suggest the unacceptable nature of mental content may be an important factor that links harm-related OCD to depression. Relatedly, obsessive beliefs related to responsibility have been shown to be driven, in part, by high personal values regarding morality, competence, and social acceptability (Doron et al., 2007). As such, some harm-related fears (e.g., concerns about preventing mistakes or rituals used to protect loved ones) may feel aligned with one's values and, thus, not significantly activate depressive symptoms. Yet, when harm-related intrusions involve repugnant thought content (e.g., doubts about having caused serious harm to someone), they may then more powerfully activate depressive symptoms. Further examination of this model is warranted.

The present study has several strengths. First, we used a dimensional, symptom-level approach to examine the relationship between OCD and depressive symptoms. Second, factor analysis was used to derive the nodes included in the network, and this approach afforded findings that more closely aligned with network theory, compared to prior item-level examinations. As a result, our results are reported in units of analysis (e.g., obsessions, negative affectivity) that converge with conceptual models of OCD, depression, and their treatment. Additional work is

needed to examine whether this approach improves the replicability of findings across settings and samples. Finally, our sample was large and included treatment-seeking sample of individuals with OCD. Thus, our findings are likely to generalize to relevant clinical samples and settings for patients with OCD and comorbid depressive symptomatology.

Our findings should also be interpreted in light of several limitations. First, data were cross-sectional. As a result, links among symptoms reflect patterns of covariation rather than causal relations. Moreover, nomothetic (i.e., group-level) findings may not generalize to the idiographic (i.e., individual) level. It is possible the relationships observed vary significantly between individuals, as well as within individuals over time. Intensive longitudinal investigations are needed to probe these person-specific and temporal dynamics. Additionally, our sample lacked racial and ethnic diversity. Moreover, comprehensive sociodemographic information (e.g., gender identity, socioeconomic status) was not captured. As data were collected through a network of treatment centers, sociodemographic barriers to receiving treatment (i.e., cost of services; Marques et al., 2010) and broader factors that perpetuate inequities in psychological research (e.g., mistrust due to historical harms; Schmotzer, 2012) likely contributed to the underrepresentation of minority participants. Ensuring equity and inclusion in network analytic studies is critical to supporting the generalizability of findings and addressing historical gaps. It is recommended that future studies consult relevant guidelines to improve the recruitment of minority populations in network research (Williams et al., 2013a). Additionally, participants in the sample were required to have OCD and were seeking treatment. Thus, our findings may not generalize to other populations and settings in which OCD and depression cohere (e.g., the community). Finally, the OCD factor structure used represents one of several dimensional conceptualizations in the literature (e.g., Leckman et al., 2010). Recent evidence suggests network-based approaches may be useful for clarifying heterogeneity in OCD (Cervin et al., 2021). Accordingly, replications of work using competing dimensional models would be useful.

5. Conclusion

Using a dimensional, network analytic approach, the present study showed that obsessions, particularly repugnant thoughts, were closely associated with negative affect and cognitive-somatic changes associated with depression, suggesting these symptoms may influence the presentation of co-occurring depressive symptoms in OCD. Understanding these functional relationships is key to improving clinical care for the large numbers of patients adversely affected by OCD and comorbid depressive symptomatology. Further research utilizing prospective designs is greatly needed to better understand causal mechanisms and individual differences in this symptom presentation.

Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation, and with the Helsinki Declaration of 1975, as revised in 2008.

CRediT authorship contribution statement

Samantha Hellberg (Conceptualization, Methodology, Data Curation, Formal analysis, Writing – Original Draft, Writing – Review & Editing), Jonathan Abramowitz (Conceptualization, Methodology,

Formal Analysis, Resources, Writing - Review & Editing, Supervision), Heidi Ojalehto (Writing – Review & Editing), Megan Butcher (Writing – Review & Editing), Bradley Riemann (Resources, Writing - Review & Editing, Project Administration).

Conflict of interest

None to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2022.08.101.

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