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A Network Perspective on Body Dysmorphic Disorder and Major Depressive Disorder

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Abstract

Background.—Body dysmorphic disorder (BDD) is a highly debilitating mental disorder associated with notable psychosocial impairment and high rates of suicidality. This study investigated BDD from a network perspective, which conceptualizes mental disorders as systems of symptoms that cause and exacerbate one another (e.g., preoccupation with perceived appearance defect triggering compulsive checking in the mirror).

Methods.—In a sample of BDD patients ($N = 148$), we used cross-sectional network models to explore the network structure of 1) BDD symptoms and 2) BDD symptoms and major depressive disorder (MDD) symptoms, and tested which symptoms were most central (i.e., most strongly associated to other symptoms).

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Authors Statement

Dr. Berta Summers and George Aalbers worked together to design the study. Dr. Berta Summers procured the data and wrote much of the background and methods, while Mr. Aalbers conducted the analyses. Payton Jones and Dr. Richard McNally contributed their expertise in Network analysis and assisted with study analyses and interpretation of results. Drs. Wilhelm and Phillips contributed the data for the current project as well as their expertise on body dysmorphic disorder. The discussion was a collaborative effort across authors. All authors contributed to and have approved the final manuscript.

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Results.—Interference in functioning due to appearance-related compulsions (BDD), feelings of worthlessness (MDD), and loss of pleasure (MDD) were most central.

Conclusion.—These symptoms were most strongly predictive of other BDD and MDD symptoms and may be features of BDD that warrant prioritization in theory development and treatment. A limitation of our study is that the precision of these findings may be limited due to a small sample size relative to the number of parameters. Replication studies in larger samples of BDD patients are needed.

Keywords

body dysmorphic disorder; major depressive disorder; comorbidity; network analysis

Introduction

Individuals with body dysmorphic disorder (BDD) are preoccupied with non-existent or minor flaws in their physical appearance, and they engage in repetitive compulsive behaviours such as compulsively checking, trying to modify, or seeking reassurance about their physical appearance (American Psychiatric Association, 2013). BDD is common (approximately 2–3% of the general population; e.g., Buhlmann et al., 2010; Schieber et al., 2015), and frequently co-occurs with major depressive disorder (MDD; Gunstad & Phillips, 2003; Phillips, Menard, Fay, & Weisberg, 2005). BDD is associated with markedly poor quality of life (Phillips, 2000), high levels of perceived stress (DeMarco, Li, Phillips, & McElroy, 1998), and strikingly high risk for suicidal ideation, suicide attempts, and completed suicide (Phillips et al., 2005; Phillips & Menard, 2006).

In this study, we investigated BDD from a network perspective, which views mental disorders as causal systems of mutually reinforcing symptoms (Borsboom, 2017). It differs radically from traditional common cause models whereby symptoms reflect an underlying disease entity that causes symptom emergence and covariance (Borsboom & Cramer, 2013). Common cause models disallow interactions among symptoms – a clinically implausible assumption – whereas network models reveal patterns of interactions constitutive of an episode of mental disorder (Borsboom, 2017; McNally, 2016; van den Hout, 2014). For instance, in MDD, decreased appetite and weight loss are likely causally connected. Causal interactions between symptoms are also observed in BDD; for instance, excessively checking perceived appearance defects (behavioral symptom) tends to maintain and exacerbate appearance-related preoccupation (cognitive symptom).

In a network of symptoms, the activation of one symptom may propagate beyond the categorical boundaries of mental disorders – for instance, preoccupation and distress/interference associated with perceived flaws in physical appearance (BDD symptoms) may cause depressed mood or suicidality (MDD symptoms). Network theorists hold that causal interactions between symptoms of different disorders can illuminate the meaning of comorbidity (Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011). Studying networks comprising symptoms from different disorders may enable us to identify the key activating features relevant to the shared network (MDD and generalized anxiety disorder [GAD]; Cramer, Waldorp, Van der Maas, & Borsboom, 2010; MDD and obsessive-

compulsive disorder [OCD]; McNally, Mair, Mugno, & Riemann, 2017; Jones, Mair, Riemann, Mugno, & McNally, 2018). In the present study, we applied network analytic methods to discern functional relations among BDD symptoms, as well as the relationship between BDD and MDD symptoms, in patients seeking treatment for BDD.

We first investigated the cross-sectional network structure of BDD symptoms alone, given that this disorder has yet to be a focus of network analysis – a method likely to deepen our understanding of relationships between symptoms in ways that are likely to inform theories of disorder maintenance. Second, we investigated the cross-sectional network structure of BDD and MDD symptoms to clarify the interplay between symptoms of these highly comorbid conditions. Networks model relationships between symptoms and provide statistical suggestions for potential causal pathways among symptoms. In network science, variables are commonly represented by ‘nodes’ (e.g., circles), and associations are illustrated as ‘edges’ (i.e., red or dotted lines for negative associations, green or solid lines for positive associations). The networks in this study are undirected; that is, their associations have no orientation, which means that – if there is indeed a causal relation between symptom A and B – we are unable to discern whether symptom A leads to symptom B, vice versa, or if the relationship is bidirectional.

Network studies have shown that symptoms differ in how strongly they are associated with other symptoms (e.g., Robinaugh, Millner, & McNally, 2016). The network perspective hypothesizes that if a specific symptom is more strongly associated with others, it can be conceptualized as ‘central’ and, in turn, has influence over the rest of the network. That is, the activation (e.g., onset of suicidal ideation) or deactivation (e.g., no longer experiencing ideation) of a „central“ feature can have a ripple effect on other aspects of the disorder. Indeed, although centrality measures have limitations (Bringmann et al., under review; Rodebaugh et al., 2018), some studies show that reduction in the severity of high-centrality symptoms is predictive of improvement in other symptoms, underscoring the clinical relevance of this analytical approach (Elliott, Jones, & Schmidt, 2018; Rodebaugh et al., 2018). Thus, in the context of BDD and MDD, identifying central aspects of the shared network can offer data-driven insight into maintenance factors that require direct intervention. The current study represents an exploratory first look at this topic; we used two common centrality measures (i.e., strength and one-step expected influence) to identify the most important symptoms in the BDD and BDD-MDD networks.

Method

Participants

Our study included 148 participants (62.8% female; age $M = 33.72$, $SD = 11.72$) whose data came from three treatment studies¹ for BDD conducted at Butler Hospital and subsequently Rhode Island Hospital, both affiliated with the Alpert Medical School of Brown University, and Massachusetts General Hospital, affiliated with Harvard Medical School. Participants

¹To assess potential participant and/or network differences between studies, we estimated a moderated network model, using the R-package mgn (Haslbeck & Waldorp, 2018). This model estimates if treatment group predicts individual symptoms, and if group moderates associations between variables. Results showed no group differences, suggesting that current study findings were not influenced by group/treatment-specific variables.

were recruited via advertisements and flyers posted around the respective community, as well as brochures mailed to local dermatologists, plastic surgeons, and mental health professionals. Two studies examined the efficacy of cognitive behavioural therapy for BDD ($n = 12$; Wilhelm, Phillips, Fama, Greenberg, & Steketee, 2011; $n = 36$; Wilhelm et al., 2014), and one study was a pharmacotherapy relapse prevention study in BDD ($n = 100$; Phillips et al., 2016). All data presented in the current manuscript were collected at the pre-treatment baseline assessment, and all procedures were approved by the Institutional Review Board responsible for overseeing research at each respective study site. Inclusion criteria across all three studies were: (a) at least 18 years of age, (b) diagnosed with primary BDD, with symptoms present for at least 6 months (diagnosis determined by Structured Clinical Interview for DSM-IV [SCID-P; First et al., 1995]), and (c) at least a „moderate” degree of BDD symptom severity endorsed (as indicated by a score of ≥ 24 on the Yale-Brown Obsessive-Compulsive Scale Modified for Body Dysmorphic Disorder (BDD-YBOCS; Phillips et al., 1997).

Primary exclusion criteria, which varied slightly across the three studies, were: (a) active or clinically significant suicidality, (b) presence of a psychotic disorder, bipolar disorder, or borderline personality disorder, (c) substance abuse or dependence within the past three months, (d) cognitive impairment, (e) self-reported brain damage or dementia, (f) primary body image (weight) concerns better accounted for by an eating disorder diagnosis, (g) concurrent psychotherapy, (h) previous CBT for BDD similar to that provided in the CBT studies, and (i) recent or concurrent medication changes. Further details of study inclusion/exclusion criteria and a thorough description of the samples appear in the original articles (Wilhelm et al., 2011; Wilhelm et al., 2014; Phillips et al., 2016). See Table 1 for clinical and demographic characteristics of participants.

Materials

Yale-Brown Obsessive-Compulsive Scale Modified for BDD (BDD-YBOCS; Phillips et al., 1997; Phillips et al., 2014).—The BDD-YBOCS is a 12-item semi-structured clinician-administered measure regarded as the gold standard measure for assessing the current severity of BDD symptoms. Items are anchored to the past week and probe obsessional preoccupations/thoughts about the perceived appearance flaw, BDD-related compulsions, insight regarding appearance beliefs, and BDD-related avoidance of situations and activities. Possible scores range from 0–48, with higher scores signifying more severe BDD. The BDD-YBOCS has good internal consistency ($\alpha = .80-.92$), high test-retest reliability ($r_s = 0.83-0.88$), and good convergent validity with other symptom assessments (e.g., the Body Dysmorphic Disorder Examination; $r_s = 0.55-0.82$; Phillips et al., 1997; Phillips et al., 2014). Consistent with previous research, in the present study, the BDD-YBOCS had adequate internal consistency ($\alpha = 0.74$).

Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996).—The BDI-II is a 21-item self-report questionnaire designed to assess depressive symptoms mapping on to the diagnostic criteria for MDD over the past two weeks. The BDI-II has excellent internal consistency ($\alpha = 0.92$; Beck, Steer, & Brown, 1996); scores range from 0–63, with higher scores indicative of more severe depression (suggested cut scores: 0–13 = “minimal,” 14–19

= “mild,” 20–28 = “moderate,” and 29–63 = “severe”). Consistent with previous research, in the present study, BDI-II had excellent internal consistency ($\alpha = 0.93$). In our sample, 35 individuals (23.64%) scored within the moderate range and 47 (31.76%) within the severe range of BDI-II. Of these individuals, 79 (53.34%) met DSM-IV diagnostic criteria for MDD.

Data analysis

Network estimation—In the open source R package *qgraph* (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012), we estimated and visualized Gaussian Graphical Models (GGMs) of a) BDD symptoms, and b) BDD and MDD symptoms. Given that this study represents an initial evaluation of this topic, analyses were largely exploratory and designed to enhance model stability. GGMs consist of nodes and edges, with nodes representing symptoms and edges representing Least Absolute Shrinkage and Selection Operator (LASSO)-regularized partial correlations between symptoms. Partial correlations represent the association between two symptoms after adjusting for their associations with all other symptoms. LASSO is used to set small partial correlations to exactly zero to reduce false positives. The LASSO regularization parameter was selected by using the extended Bayesian Information Criterion (EBIC; Foygel & Drton, 2010) with hyperparameter $\gamma = 0$. Hyperparameter γ controls the trade-off between sensitivity (i.e., probability of including associations that are non-zero in the true network) and specificity (i.e., probability of not including associations that are zero in the true network). A recent simulation (Williams & Rast, 2018) suggests that, in a small sample ($N = 150$), hyperparameter $\gamma = 0$ has higher sensitivity and lower specificity than *qgraph*'s default hyperparameter setting ($\gamma = 0.5$). To err on the side of discovery, we estimate both GGMs with hyperparameter $\gamma = 0$.

To increase the stability of our networks and their centrality indices, we calculated Spearman correlations instead of polychoric correlations. The edges of the Spearman and polychoric correlation networks correlated strongly ($r = 0.99$).

Centrality indices—We calculated strength and one-step expected influence for each individual symptom to evaluate the relative impact they might have on the rest of the network (Robinaugh, Millner, & McNally, 2016). Strength was calculated with the *qgraph* package (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012) and one-step expected influence with the *networktools* package (Jones, 2018). To quantify whether symptoms differed in how strongly they were associated with those of the other disorder, we also computed bridge one-step expected influence by using the *networktools* package.

Strength represents the sum of absolute values of associations from symptom X to all other symptoms in the network. This metric does not distinguish between positive and negative associations, which do not pose a problem in networks exclusively connected by positive associations, as is the case for most, but not all, symptom networks.

Unlike strength, one-step expected influence distinguishes between positive and negative associations by taking the sum of all non-absolute values of associations from symptom X to the other symptoms. If networks contain both positive and negative associations, distinguishing between them is important, as positive associations could indicate activating

influence between symptoms, whereas negative associations could indicate deactivating influence. For example, if symptom A has 5 positive (i.e., activating) associations to the rest of the network, and symptom B has 5 equally strong negative (i.e., de-activating) associations to the rest of the network, these symptoms may have the same strength (i.e., 5) but a different one-step expected influence; that is, symptom A has an expected influence of 5, whereas symptom B has -5 . This would suggest that these symptoms have a different association pattern within the network; i.e., one is associated with activation of other nodes in the network and the other is associated with deactivation of other nodes in the network. Robinaugh, Millner and McNally (2016) have demonstrated that expected influence is a more appropriate measure than strength when there are negative associations in the network, as it accounts for valence of associations and is more strongly associated with observed influence when negative associations are present.

Bridge expected influence (one-step; Jones, 2018; Jones, Ma., & McNally, in press) is similar to one-step expected influence. It is calculated by summing all absolute associations between a symptom of one syndrome (e.g., BDD symptom A) and symptoms of another syndrome (MDD symptoms A through E).

Stability tests²—We used the R-package bootnet (Epskamp, Borsboom, & Fried, 2018) to assess the stability of the strength centrality index, and a modified version of bootnet (Jones, 2018) to assess the stability of one-step expected influence. Bootnet calculates the correlation stability (CS) coefficient, which represents the maximum proportion of participants that can be excluded from the network analysis, such that the correlation between original centrality indices (i.e., those based on the full sample) and centrality indices based on subsets of the sample is 0.7 or higher with 95% probability. A simulation study by Epskamp and colleagues (2018) suggests that the CS-coefficient should not be below 0.25, and preferably above 0.5, to interpret centrality differences. Stability tests suggested good stability for strength and one-step expected influence (CS-coefficients > 0.5) and just above the acceptable level of stability for bridge one-step expected influence (CS-coefficient = 0.27). This means that the bridge centrality values must be interpreted with caution.

Bootstrapped difference test—We used the bootstrapped difference test (Epskamp, Borsboom, & Fried, 2018) to compute whether any symptoms significantly differed in terms of strength and expected influence on the rest of the symptoms in the network.

²To examine if results were stable across different estimation methods, we estimated mixed graphical models (MGMs) and unregularized partial correlation (i.e., concentration) networks. The MGMs were very sparsely connected, which was likely due to our small sample size. Because we believe that this result reflects an unacceptably large proportion of false negative associations, we do not report these models in this article. Main results were similar for LASSO-regularized and unregularized partial correlation networks. However, stability tests suggested that strength and one-step expected influence had low stability (CS-coefficient < 0.5). Hence, we only report results from the LASSO-regularized networks.

Results

BDD network

As Figure 1 illustrates, individual BDD symptoms are interconnected by associations that differ in edge weight. Teal circles represent BDD-YBOCS items, green/solid lines indicate positive associations, and red/dotted lines indicate negative associations. The thickness of a line corresponds to the strength of the associations. For instance, interference in functioning due to appearance preoccupations (obsessions) and interference in functioning due to compulsions are strongly associated ($\rho = 0.38$ [0.24–0.52]), whereas interference in functioning due to compulsions and disorder-related avoidance are more weakly associated ($\rho = 0.14$ [0.01–0.28]). The strongest associations were between similarly worded items, such as time spent on compulsions and time spent on obsessions, as well as between interference due to compulsions and interference due to obsessions. However, though they are similarly worded, these items represent different constructs from a clinical perspective. Of note, running analyses in which similarly-worded items were combined via the *goldbricker* function in the networktools R package (Jones, 2018) did not alter the pattern or interpretation of results (see Supplemental Materials for *goldbricker* network figures). Furthermore, not all associations in the network are positive; for instance, interference in functioning due to appearance preoccupations and effort to resist compulsions are negatively associated ($\rho = -0.09$ [-0.2–0.03]). Bootstrapping results show that all confidence intervals of negative associations contain 0. Finally, some BDD symptoms are not directly connected, such as effort made to resist compulsions and lack of insight ($\rho = 0.00$ [-0.09–0.09]).

BDD centrality measures³

We illustrate this network's centrality measures in Figure 2. Individual BDD symptoms differ in one-step expected influence (i.e., sum of associations) and strength (i.e., sum of absolute value of associations) centrality. These centrality metrics flag „interference due to BDD compulsions' as the most central (i.e., most strongly connected) BDD symptom in this network. Bootstrapping tests showed that this symptom had greater strength than 75% of the BDD symptoms. Most other symptoms are neither highly central nor peripheral. One-step expected influence is lowest for lack of BDD-related insight, effort made to resist compulsions, and distress associated with compulsions. In measures of strength, lack of insight, distress associated with prevention of compulsions, and effort to resist thoughts rank among the least central symptoms. Strength and one-step expected influence had good stability (for both metrics, CS-coefficient = 0.57).

BDD/MDD network

Figure 3 illustrates the network of BDD and MDD symptoms. Teal circles represent BDD-YBOCS items, yellow circles represent BDI-II items. Green/solid lines indicate positive associations and red/dotted lines indicate negative associations. Thicker lines represent stronger associations. Figure 3 suggests that associations are strongest between symptoms of the same syndrome. On average, associations among BDD symptoms are 12.89 times

³To check if the differences in centrality values might be related to differential item variance, we correlated centrality estimates to each variable's standard deviation. We found that this correlation was non-significant across estimation methods.

stronger than the average association between BDD and MDD symptoms. Associations among MDD symptoms are, on average, 9.78 times stronger than the average association between BDD and MDD symptoms. However, nearly all BDD symptoms are directly associated with MDD symptoms, as indicated by the lines between them. Bootstrapping results show that the 95% confidence interval of nearly all these associations contains zero, indicating no statistical significance. The exceptions to this are two positive LASSO-regularized partial correlations: 1) between distress caused by thoughts about appearance and sadness ($p = 0.12$ [>0.00 – 0.23]), and 2) between interference due to appearance-related thoughts and loss of interest ($p = 0.11$ [0.01 – 0.21]).

BDD/MDD network centrality measures

Figure 4 shows that the following symptoms have the greatest strength and one-step expected-influence in the BDD and MDD symptom network: interference in functioning due to compulsions, feelings of worthlessness, and loss of pleasure, which have significantly greater strength than 43.8%, 40.6 %, and 53.1% of the other symptoms in the network, respectively (see Supplemental Materials for bootstrapped significance test results). Lack of insight has significantly lower strength than 59.3% of the other symptoms in the network. Strength and one-step expected influence had good stability (CS-coefficient for strength = 0.53, for expected influence = 0.57). Figure 5 shows that bridge expected influence is greatest for time spent on obsessions, distress due to obsessions, lack of insight, avoidance, and punishment feelings. Bridge expected influence had acceptable stability (CS-coefficient = 0.27).

Discussion

The current study represents an initial, exploratory examination of the network structure of BDD and MDD symptoms in a clinical BDD sample. In the BDD-only network, „interference in functioning due to compulsions’ emerged as the most central item, meaning that it was most strongly connected to the rest of the BDD symptoms and most strongly predictive of other BDD symptoms. Central symptoms may not only be more predictive than non-central symptoms, but may also be more causally influential (e.g., Robinaugh, Millner, & McNally, 2016). However, because our data are cross-sectional, our findings cannot confirm causality and whether interference due to appearance-related compulsions strongly activates other symptoms, *vice versa*, or there is a bi-directional relationship. Of note, without engagement in compulsions, it would be impossible to experience interference due to compulsions. Hence, it is conceivable that „interference in functioning due to compulsions’ is a symptom that occurs at the end of the causal chain rather than a symptom that activates other BDD symptoms. In light of the BDD network structure, it appears that distress due to compulsions might drive the interference in psychosocial functioning caused by appearance-related compulsions.

In the BDD/MDD network, ‘interference in functioning due to compulsions’, ‘feelings of worthlessness’, and ‘loss of pleasure’ were most strongly associated with other symptoms. Closer inspection of their connectivity revealed that these symptoms were primarily associated with symptoms of the same syndrome (e.g., feelings of worthlessness with other

MDD symptoms). This could suggest they do not bridge between BDD and MDD, but rather are causally related to symptoms of their respective syndromes. Activation of central symptoms in one cluster/disorder can potentially influence activation of symptoms in the other cluster/disorder. To investigate which symptoms do potentially ‘bridge’ between BDD and MDD, we computed each symptom’s bridge centrality, which was greatest for time spent on obsessions, distress due to obsessions, lack of insight, avoidance, and punishment feelings. However, these values should be interpreted with caution as bridge centrality met barely acceptable levels of stability. One potential explanation for this is that our sample size might have been too small to accurately estimate the BDD/MDD symptom network, attenuating the reliability of the bridge centrality estimates. Second, the modest stability of this metric may indicate that BDD and MDD symptoms do not vary in terms of bridge centrality. We recommend that future studies test the robustness of this result in a larger sample of BDD patients. It is also possible that the most important ‘bridging’ elements between these two conditions may not have been captured in our data. For instance, transdiagnostic processes, such as maladaptive cognitive processing or attentional styles may explain the comorbidity between MDD and BDD, rather than core symptoms assessed via the BDD-YBOCS and the BDI-II. Phenomenological studies examining age of onset suggest that BDD tends to develop prior to MDD in individuals who meet criteria for both conditions (e.g., Gunstad & Phillips, 2003); however, given the cross-sectional nature of our data, we cannot draw conclusions about directionality of influence. Thus, future research into possible causal relationships between core BDD and MDD symptoms, as well as clinically and theoretically relevant non-symptoms, is warranted.

Although several studies have applied cross-sectional network analysis to uncover the connections among symptoms of different mental disorders, this is the first to do so for BDD and MDD. The present study is theoretically and clinically relevant in several ways. First, our findings may strengthen empirical support for BDD as a disorder on both the affective and obsessive-compulsive spectrum (Phillips et al., 1995; Phillips & Stout, 2006). Relevant to the affective spectrum, although the symptoms of each disorder seemed to cluster together within their respective syndrome, edges between symptoms of the two disorders were overwhelmingly positive, rather than negative. These findings corroborate clinical observations that these conditions represent interconnected, but clinically distinct, phenomena. Similar to the current study, two recent OCD/MDD comorbidity network studies also identified loss of pleasure as a central symptom in the shared network (McNally, Mair, Mugno, & Riemann, 2017; Jones, Mair, Riemann, Mugno, & McNally, 2018). These studies, along with the present study, further found high strength centrality for interference in functioning due to compulsions. The observed overlap in findings from BDD and OCD network studies underscores the relative importance of compulsions to other elements of the networks and further bolsters research showing etiological and phenomenological similarities between these disorders (Phillips & Stout, 2006).

The observed high centrality of feelings of worthlessness is noteworthy, as many BDD patients base their self-worth on how they look, endorsing the belief: “If my appearance is defective, then I am worthless” (Veale et al., 1996). As BDD patients perceive their appearance to be defective (in accordance with DSM-5 diagnostic criteria; APA, 2013), it is conceivable that their feelings of worthlessness could be attributable to these exaggerated

and erroneous beliefs about their unsatisfactory appearance. However, as our study did not include a measure of appearance-related beliefs (e.g., beliefs about the importance of appearance and perceived consequences of imperfection), future studies should investigate how these beliefs might contribute to feelings of worthlessness in BDD patients.

Our data also highlight potentially important intervention targets that are worth examining in future research, such as feelings of worthlessness and interference in psychosocial functioning due to appearance-related compulsions. In fact, the empirically supported and manualized CBT treatment commonly used with BDD patients (Wilhelm, Phillips, & Steketee, 2013; Wilhelm, et al., 2019) does target maladaptive core beliefs via cognitive restructuring techniques, which may include feelings of worthlessness. However, our findings suggest that even greater emphasis on worthlessness and other key depressive symptoms identified in this report may be fruitful. For instance, it may be useful to explicitly identify and emphasize tracking of these feelings early in treatment and dedicate full sessions to challenging and remediating these thoughts over the course of therapy. In addition, our findings underscore the importance of addressing interference in functioning due to BDD compulsions; namely, reducing avoidance behaviours and fading out rituals that directly interfere with role functioning (e.g., work, school, relationships) via exposure and response prevention. Although these findings largely map on to current treatment interventions, follow-up studies (with larger sample sizes) should be conducted to test if the high centrality of these symptoms is a robust phenomenon and whether there are other symptoms in the network that deserve more (or less) emphasis in treatment.

Without supporting theoretical underpinnings and empirical work, centrality metrics alone can suggest, but not confirm, the most important clinical targets. Thus, it is important to note that in addition to the BDD network mapping on to existing gold-standard treatment approaches, our MDD/BDD network data also converge with previous empirical findings. For instance, we found associations from feelings of worthlessness to suicidality. Previous research in MDD patients demonstrates that, compared to other MDD symptoms, feelings of worthlessness are more strongly associated with lifetime suicide attempts (Jeon et al., 2014). Additionally, research in MDD patients found that feelings of worthlessness and suicidality positively predict each other across time (e.g., stronger feelings of worthlessness in the previous week predicted more severe suicidal ideation in the following week; Bringmann, Lemmens, Huibers, Borsboom, & Tuerlinckx, 2015). As BDD is associated with high risk for suicidality (Phillips, Menard, Fay, & Weisberg, 2005; Phillips & Menard, 2006), our study underscores the importance of future research into the causal link from feelings of worthlessness to suicidality in this population, as well as the factors that contribute to the development of these feelings.

This is the first study to apply the network approach to BDD and MDD. One strength of our study is that we used state-of-the-art methods to conduct cross-sectional network analysis. Furthermore, we used a gold-standard structured clinical interview to diagnose BDD symptoms – an approach that is especially valuable as many patients lack insight into their condition, which complicates the use of self-report diagnostic measures (Phillips, 2004). Moreover, although the present study had a relatively small sample size in proportion to the number of variables, stability analyses show that centrality indices ranged from acceptable to

good, which suggests that centrality differences between symptoms are interpretable (Epskamp, Borsboom, & Fried, 2018) and thus offer a meaningful contribution to the currently limited literature.

Our study also has limitations that should be considered in future research. First, our data were cross-sectional; thus, current study findings offer tentative hypotheses about possible causal relationships between symptoms that must be more directly tested. To investigate directionality of associations between symptoms, we recommend 1) experience-sampling methodology to investigate how individual BDD and MDD symptoms predict each other across time (Bringmann et al., 2013), and 2) experimental work that manipulates specific BDD sequelae, such as symptoms (e.g., appearance-related compulsions) and mechanisms (e.g., interpretation biases).

Another limitation inherent to cross-sectional data is that our results are not readily translatable to the individual. The within-subjects structure of BDD and MDD symptoms may be heterogeneous across individuals; thus, the between-subjects structure utilized in the current study may not represent all patients. Recent advances in the analysis of high-intensive time-series data now enable researchers to study the contemporaneous and temporal associations of symptoms within individuals (Epskamp, Waldorp, Möttus, & Borsboom, 2018), and research into clinical applications of such intra-individual network models is currently in progress (e.g., Bak, Drukker, Hasmi, & van Os, 2016). Furthermore, our sample was fairly homogenous with respect to race and ethnicity, which may also limit generalizability of findings. Future research should seek to recruit more diverse samples, as BDD affects individuals of all ethnic and cultural backgrounds.

Although our sample size is comparable to other novel network analytical explorations of comorbidity (e.g., Jones, Mair, Riemann, Mugno, & McNally, 2018; Levinson et al., 2017), it is worth noting that the present sample was relatively small given the number of variables in the network. Stability analyses suggested that centrality indices were stable enough to be interpreted, although it is possible they were driven primarily by strong associations between symptoms within the same syndrome rather than those between syndromes. This is likely a natural consequence of syndromic clustering; however, the current study represents a first step in understanding the shared BDD-MDD network, and future replication efforts with larger samples are needed to clarify the stability of the observed clusters. Future studies may also use non-treatment-seeking samples to explore whether the pattern of findings is consistent with those observed in the current sample.

Furthermore, MDD symptoms were assessed via a self-report questionnaire (BDI-II) rather than a clinical interview. However, given the sound psychometric properties of the BDI-II (Beck, Steer, & Brown, 1996), we do not expect this to have significantly altered results. Additionally, given that different assessment methodologies (i.e., self-report and clinical interview) were utilized to measure BDD and MDD symptoms, associations were likely not artificially inflated due to common method variance. However, this difference in assessment modality could have contributed to the low stability of bridge centrality. Future work should investigate whether this is a robust phenomenon. Use of different depression measures (for an overview, see Fried, 2017) in further iterations of this research could reveal a different

pattern of findings and/or provide more information about relationships observed in the current study. Relatedly, future studies may consider testing the network structure of different measures of BDD sequelae such as the self-report Body Dysmorphic Disorder Symptom Scale (BDD-SS; Wilhelm, Greenberg, Rosenfield, Kasarskis, & Blashill, 2016). An important step for future research will be the development of a computational model that posits specific causal relationships among BDD symptoms (for an example in panic disorder research, see Robinaugh et al., 2019). Such a model would integrate current BDD theories and empirical evidence into a system of mathematical equations, representing theorized and experimentally tested causal relationships among BDD-related sequelae (e.g., symptoms, cognitive mechanisms). This model would be useful to assess what current disorder conceptualizations in the literature can and cannot explain about BDD and has the potential to generate new questions to be tested via experimental work. Experimental designs are critical for drawing causal conclusions about relations between network elements.

A recent study showed that manipulating certain BDD symptoms (i.e., appearance-related behaviours such as mirror checking, grooming, reassurance-seeking) in a subclinical sample of female undergraduates yielded changes in symptoms of BDD and common comorbid conditions (i.e., symptoms of depression, social anxiety, bulimia nervosa) as well as clinically relevant non-symptoms (e.g., interpretive biases, beliefs about the importance of appearance; Summers & Cogle, 2018). This finding corroborates those of the current study by highlighting compulsions (interference due to compulsions) as a central component of the BDD network. These findings also provide preliminary evidence against the latent variable hypothesis that all BDD symptoms are merely epiphenomena of an underlying disease. Future work might seek to replicate and extend this work in a heterogeneous sample of individuals with a BDD diagnosis and examine nuanced network changes following experimental manipulations of individual elements. Experimental work (combined with computational modelling, see e.g., Robinaugh et al., 2019) represents a critical step for developing and testing a network theory of BDD that could enhance the efficacy of BDD treatment by guiding scientist-practitioners to the most influential intervention targets.

Taken together, this study suggests that different BDD and MDD symptoms are not equally central and thus may be differentially influential. Although we cannot draw causal conclusions from these cross-sectional analyses, we believe that they provide a start for future research into this issue. Specifically, follow-up research is needed to develop and quantitatively test an integrative computational network model of BDD. As argued in previous publications, a network theory of mental disorders can be complete only when all causally relevant elements are included (Fried & Cramer, 2017; Jones et al., 2017). Hence, future network studies on BDD should include relevant symptoms (i.e., of BDD and common comorbid disorders such as MDD and social anxiety disorder), as well as non-symptoms that may be driving transdiagnostic vulnerabilities, such as maladaptive beliefs or interpretation biases (Wilhelm et al., 2013).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub.
- Bak M, Drukker M, Hasmi L, & van Os J (2016). An n=1 clinical network analysis of symptoms and treatment in psychosis. *PloS one*, 11, e0162811. [PubMed: 27643994]
- Beck AT, Steer RA, & Brown GK (1996). Beck Depression Inventory-II. San Antonio, 78, 490–498.
- Bringmann LF, Elmer T, Epskamp S, Krause RW, Schoch D, Wichers M, ... & Snippe E (under review). What do centrality measures measure in psychological networks?.
- Bringmann LF, Vissers N, Wichers M, Geschwind N, Kuppens P, Peeters F, ... & Tuerlinckx F (2013). A network approach to psychopathology: new insights into clinical longitudinal data. *PloS one*, 8, e60188. [PubMed: 23593171]
- Bringmann LF, Lemmens LHJM, Huibers MJH, Borsboom D, & Tuerlinckx F (2015). Revealing the dynamic network structure of the Beck Depression Inventory-II. *Psychological Medicine*, 45, 747–757. [PubMed: 25191855]
- Borsboom D (2008). Psychometric perspectives on diagnostic systems. *Journal of Clinical Psychology*, 64, 1089–1108. [PubMed: 18683856]
- Borsboom D, Cramer AO, Schmittmann VD, Epskamp S, & Waldorp LJ (2011). The small world of psychopathology. *PloS one*, 6, e27407. [PubMed: 22114671]
- Borsboom D, & Cramer AO (2013). Network analysis: An integrative approach to the structure of psychopathology. *Annual Review of Clinical Psychology*, 9, 91–121.
- Borsboom D (2017). A network theory of mental disorders. *World Psychiatry*, 16, 5–13. [PubMed: 28127906]
- Buhlmann U, Glaesmer H, Mewes R, Fama JM, Wilhelm S, Brahler E, et al. (2010). Updates on the prevalence of body dysmorphic disorder: A population-based survey. *Psychiatry Research*, 178, 171–175. [PubMed: 20452057]
- Cramer AO, Waldorp LJ, Van der Maas HL, & Borsboom D (2010). Comorbidity: A network perspective. *Behavioral and Brain Sciences*, 33, 137–150 [PubMed: 20584369]
- DeMarco LM, Li LC, Phillips KA, & McElroy SL (1998). Perceived stress in body dysmorphic disorder. *The Journal of Nervous and Mental Disease*, 186, 724–726. [PubMed: 9824177]
- Elliott H, Jones PJ, & Schmidt U (2018). Central symptoms predict post-treatment outcomes and clinical impairment in anorexia nervosa: A network analysis in a randomized-controlled trial. Retrieved from <https://psyarxiv.com/hw2dz/>
- Epskamp S, Borsboom D, & Fried EI (2018). Estimating psychological networks and their accuracy: A tutorial paper. *Behavior Research Methods*, 50, 195–212. [PubMed: 28342071]
- Epskamp S, Cramer AO, Waldorp LJ, Schmittmann VD, & Borsboom D (2012). qgraph: Network visualizations of relationships in psychometric data. *Journal of Statistical Software*, 48, 1–18.
- Epskamp S, Waldorp LJ, Möttus R, & Borsboom D (2018). The Gaussian Graphical Model in Cross-Sectional and Time-Series Data. *Multivariate Behavioral Research*, 53, 453–480. [PubMed: 29658809]
- First MB, Spitzer RL, Gibbon M, & Williams JB (1995). Structured clinical interview for DSM-IV Axis I disorders-patient edition (SCID-I/P, version 2.0). New York: Biometrics Research Department, New York State Psychiatric Institute.
- Fried EI, & Cramer AO (2017). Moving forward: challenges and directions for psychopathological network theory and methodology. *Perspectives on Psychological Science*, 12, 999–1020. [PubMed: 28873325]

- Gunstad J, & Phillips KA (2003). Axis I comorbidity in body dysmorphic disorder. *Comprehensive Psychiatry*, 44, 270–276. [PubMed: 12923704]
- Jeon HJ, Park JI, Fava M, Mischoulon D, Sohn JH, Seong S, ... & Cho MJ, (2014). Feelings of worthlessness, traumatic experience, and their comorbidity in relation to lifetime suicide attempt in community adults with major depressive disorder. *Journal of Affective Disorders*, 166, 206–212. [PubMed: 25012433]
- Jones PJ (2018). networktools: Tools for Identifying Important Nodes in Networks. R package version 1.1.1. <https://CRAN.R-project.org/package=networktools>
- Jones PJ, Heeren A, & McNally RJ (2017). Commentary: A network theory of mental disorders. *Frontiers in Psychology*, 8, 1305. [PubMed: 28824490]
- Jones PJ, Ma R, & McNally RJ (in press). Bridge centrality: A network approach to understanding comorbidity. *Multivariate Behavioral Research*.
- Jones PJ, Mair P, Riemann BC, Mugno BL, & McNally RJ (2018). A network perspective on comorbid depression in adolescents with obsessive-compulsive disorder. *Journal of Anxiety Disorders*, 53, 1–8. [PubMed: 29125957]
- Levinson CA, Zerwas S, Calebs B, Forbush K, Kordy H, Watson H, ... Bulik CM (2017). The core symptoms of bulimia nervosa, anxiety, and depression: A network analysis. *Journal of Abnormal Psychology*, 126, 340–354. [PubMed: 28277735]
- McNally RJ (2016). Can network analysis transform psychopathology? *Behaviour Research and Therapy*, 86, 95–104. [PubMed: 27424882]
- McNally RJ, Mair P, Mugno BL, & Riemann BC (2017). Co-morbid obsessive-compulsive disorder and depression: A Bayesian network approach. *Psychological Medicine*, 47, 1204–1214. [PubMed: 28052778]
- Phillips KA (2000). Quality of life for patients with body dysmorphic disorder. *The Journal of Nervous and Mental Disease*, 188, 170–175. [PubMed: 10749282]
- Phillips KA (2004). Psychosis in body dysmorphic disorder. *Journal of Psychiatric Research*, 38, 63–72. [PubMed: 14690771]
- Phillips KA, Coles ME, Menard W, Yen S, Fay C, Weisberg RB (2005). Suicidal ideation and suicide attempts in body dysmorphic disorder. *The Journal of Clinical Psychiatry*, 66, 717–725. [PubMed: 15960564]
- Phillips KA, Hollander EE, Rasmussen SA, Aronowitz BR, DeCaria CC, & Goodman WK (1997). A severity rating scale for body dysmorphic disorder: Development, reliability, and validity of a modified version of the Yale-Brown Obsessive Compulsive Scale. *Psychopharmacology Bulletin*, 33, 17–22. [PubMed: 9133747]
- Phillips KA, Keshaviah A, Dougherty DD, Stout RL, Menard W, & Wilhelm S (2016). Pharmacotherapy relapse prevention in body dysmorphic disorder: A double blind, placebo-controlled trial. *American Journal of Psychiatry*, 173, 887–895. [PubMed: 27056606]
- Phillips KA, & Menard W (2006). Suicidality in body dysmorphic disorder: A prospective study. *American Journal of Psychiatry*, 163, 1280–1282. [PubMed: 16816236]
- Phillips KA, Menard W, Fay C, & Weisberg R (2005). Demographic characteristics, phenomenology, comorbidity, and family history in 200 individuals with body dysmorphic disorder. *Psychosomatics*, 46, 317–332. [PubMed: 16000674]
- Phillips KA, & Stout RL (2006). Associations in the longitudinal course of body dysmorphic disorder with major depression, obsessive-compulsive disorder, and social phobia. *Journal of Psychiatric Research*, 40, 360–369. [PubMed: 16309706]
- Phillips KA, McElroy SL, Hudson JI, & Pope JH (1995). Body dysmorphic disorder: an obsessive-compulsive spectrum disorder, a form of affective spectrum disorder, or both? *The Journal of Clinical Psychiatry*, 56, 41–51.
- Phillips KA, Hart AS, & Menard W (2014). Psychometric evaluation of the Yale Brown Obsessive-Compulsive Scale modified for body dysmorphic disorder (BDD-YBOCS). *Journal of Obsessive-Compulsive and Related Disorders*, 3, 205–208.
- Robinaugh DJ, Millner AJ, & McNally RJ (2016). Identifying highly influential nodes in the complicated grief network. *Journal of Abnormal Psychology*, 125, 747–757. [PubMed: 27505622]

- Robinaugh D, Haslbeck JMB, Waldorp L, Kossakowski JJ, Fried EI, Millner A, ... Borsboom D (2019). Advancing the Network Theory of Mental Disorders: A Computational Model of Panic Disorder. doi: 10.31234/osf.io/km37w
- Rodebaugh TL, Tonge NA, Piccirillo ML, Fried E, Horenstein A, Morrison AS, ... & Blanco C (2018). Does centrality in a cross-sectional network suggest intervention targets for social anxiety disorder?. *Journal of Consulting and Clinical Psychology*, 86, 831–844. [PubMed: 30265042]
- Schieber K, Kollei I, de Zwaan M, Martin A (2015). Classification of body dysmorphic disorder--What is the advantage of the new DSM-5 criteria? *Journal of Psychosomatic Research*, 78, 223–227. [PubMed: 25595027]
- van den Hout M (2014). Psychiatric symptoms as pathogens. *Clinical Neuropsychiatry: Journal of Treatment Evaluation*, 11, 153–159.
- Veale D (2004). Advances in a cognitive behavioural model of body dysmorphic disorder. *Body Image*, 1, 113–125. [PubMed: 18089145]
- Veale D, Boocock A, Gournay K, Dryden W, Shah F, Willson R, & Walburn J (1996). Body dysmorphic disorder: A survey of fifty cases. *The British Journal of Psychiatry*, 169, 196–201. [PubMed: 8871796]
- Veale D, & Riley S (2001). Mirror, mirror on the wall, who is the ugliest of them all? The psychopathology of mirror gazing in body dysmorphic disorder. *Behaviour Research and Therapy*, 39, 1381–1393 [PubMed: 11758697]
- Wilhelm S, Greenberg JL, Rosenfield E, Kasarskis I, & Blashill AJ (2016). The Body Dysmorphic Disorder Symptom Scale: Development and preliminary validation of a self-report scale of symptom specific dysfunction. *Body Image*, 17, 82–87. [PubMed: 26971118]
- Wilhelm S, Phillips KA, Fama JM, Greenberg JL, & Steketee G (2011). Modular cognitive-behavioral therapy for body dysmorphic disorder. *Behavior Therapy*, 42, 624–633. [PubMed: 22035991]
- Wilhelm S, Phillips KA, & Steketee G (2012). *Cognitive-Behavioral Therapy for Body Dysmorphic Disorder: A Treatment Manual*. New York, NY: Guilford Press.
- Wilhelm S, Phillips KA, Didie E, Buhlmann U, Greenberg JL, Fama JM, Keshaviah A, & Steketee G (2014). Modular cognitive-behavioral therapy for body dysmorphic disorder: A randomized controlled trial. *Behavior Therapy*, 45, 314–327. [PubMed: 24680228]
- Wilhelm S, Phillips KA, Greenberg JL, O'Keefe SM, Hoepfner SS, Keshaviah A, Sarvode-Mothi S, Schoenfeld DA (2019). Efficacy and posttreatment effects of therapist-delivered cognitive behavioral therapy vs supportive psychotherapy for adults with body dysmorphic disorder: A randomized clinical trial. *JAMA Psychiatry*, 76, 363–373. [PubMed: 30785624]

Highlights

- This is the first study to examine BDD and MDD symptoms from a network perspective.
- Interference due to rituals emerged as the most central BDD symptom.
- Feelings of worthlessness and loss of pleasure were the most central MDD symptoms.
- Central symptoms were most strongly associated with symptoms in the same syndrome.
- Findings warrant research into a BDD network theory, which could benefit treatment.

Figure 1.

LASSO-regularized partial correlation network of BDD-YBOCS items. Circles represent the 12 BDD-YBOCS items (past-week BDD symptoms). Items starting with “O” represent the obsession items anchored to appearance-preoccupations. Items starting with “C” represent the compulsive items anchored to appearance-rituals. The remaining two items assess participants’ insight into their condition and avoidance anchored to symptoms. Green/solid (red/dotted) lines indicate positive (negative) associations between BDD-YBOCS items, and thicker (thinner) lines represent stronger (weaker) associations.

Figure 2.

One-step expected influence, and strength centrality measures for each of the 12 BDD-YBOCS items in the LASSO-regularized partial correlation network (shown in Figure 1). Items starting with “O” represent the obsession items anchored to appearance-preoccupations (i.e., time spent engaged in thoughts, interference due to thoughts, distress due to thoughts, effort made to resist thoughts, and actual control over thoughts). Items starting with “C” represent the compulsive items anchored to appearance-rituals (i.e., time spent engaged in compulsions, interference due to compulsions, distress due to compulsions, effort made to resist compulsions, and actual control over compulsions). The remaining two items assess participants’ insight into their condition and avoidance anchored to symptoms.

Figure 3.

LASSO-regularized partial correlation network of BDD-YBOCS and BDI-II items. Teal circles represent the 12 BDD-YBOCS items, yellow circles represent the 21 BDI-II items. Green/solid (red/dotted) lines indicate positive (negative) LASSO-regularized partial correlations. Thicker (thinner) lines represent stronger (weaker) correlations. For BDD symptoms, items starting with “O” represent the obsession items anchored to appearance-preoccupations, while items starting with “C” represent the compulsive items anchored to appearance-rituals. The remaining two items assess participants’ insight into their condition and avoidance anchored to symptoms.

Figure 4.

One-step expected influence and strength centrality measures for each BDD-YBOCS and BDI-II item in the LASSO-regularized partial correlation network (shown in Figure 3). For BDD symptoms, items starting with “O” represent the obsession items anchored to appearance-preoccupations, while items starting with “C” represent the compulsive items anchored to appearance-rituals. The remaining two items assess participants’ insight into their condition and avoidance anchored to symptoms. MDD symptoms are depicted as squares.

Figure 5.

Bridge one-step expected influence for each BDD-YBOCS and BDI-II item in the LASSO-regularized partial correlation network (shown in Figure 3). For BDD symptoms, items starting with “O” represent the obsession items anchored to appearance-preoccupations, while items starting with “C” represent the compulsive items anchored to appearance-rituals. The remaining two items assess participants’ insight into their condition and avoidance anchored to symptoms. MDD symptoms are depicted as squares.

Table 1.

Clinical and demographic characteristics of participants.

		%
Age in years	<i>M</i> = 33.72 (<i>SD</i> = 11.72)	
Gender	Female (<i>n</i> = 93)	62.84
	Male (<i>n</i> = 55)	37.16
Ethnic background	Caucasian (<i>n</i> = 120)	81.08
	Hispanic (<i>n</i> = 12)	8.11
	African American (<i>n</i> = 10)	6.76
	Asian (<i>n</i> = 2)	1.35
	Alaskan (<i>n</i> = 2)	1.35
	American Indian (<i>n</i> = 1)	0.68
	Other (<i>n</i> = 1)	0.68
Comorbid Axis I disorders	OCD (<i>n</i> = 16)	10.81
	Trichotillomania (<i>n</i> = 2)	1.35
	Anorexia Nervosa (<i>n</i> = 1)	0.68
	Binge Eating Disorder (<i>n</i> = 5)	3.38
	Bulimia Nervosa (<i>n</i> = 1)	0.68
	Eating Disorder NOS (<i>n</i> = 5)	3.38
	Bipolar I Disorder (<i>n</i> = 1)	0.68
	Major Depressive Disorder (<i>n</i> = 79)	53.38
	Dysthymia (<i>n</i> = 11)	7.43
	Generalized Anxiety Disorder (<i>n</i> = 16)	10.81
	Agoraphobia (<i>n</i> = 8)	5.41
	Panic Disorder (<i>n</i> = 5)	3.38
	Post-Traumatic Stress Disorder (<i>n</i> = 2)	1.35
	Social Anxiety Disorder (<i>n</i> = 38)	25.68
	Specific Phobia (<i>n</i> = 17)	11.49
	Any substance use disorder (<i>n</i> = 9)	6.08
Comorbid personality disorders	Avoidant Personality Disorder (<i>n</i> = 9)	6.08
	Obsessive-Compulsive Personality Disorder (<i>n</i> = 7)	4.73
	Dependent Personality Disorder (<i>n</i> = 2)	1.35
	Passive Aggressive Personality Disorder (<i>n</i> = 2)	1.35
	Other (<i>n</i> = 2)	1.35
	Paranoid Personality Disorder (= 1)	0.68

Note. Descriptive statistics shown were collected at the baseline assessment prior to participants taking part in one of three separate treatment studies. For comprehensive breakdown of descriptives by study, please reference the respective clinical trials (i.e., Wilhelm, Phillips, Fama, Greenberg, & Steketee, 2011; Wilhelm et al., 2014; Phillips et al., 2016).