Mechanisms of Cognitive Behavioral Therapy Effects on Symptoms of Body Dysmorphic Disorder: A Network Intervention Analysis

Emily E. Bernstein[1], **Katharine A. Phillips**[2,3], **Jennifer L. Greenberg**[1], **Joshua Curtiss**[1], **Susanne S. Hoeppner**[1], **Sabine Wilhelm**[1]

[1]Massachusetts General Hospital and Harvard Medical School

[2]Rhode Island Hospital and Alpert Medical School of Brown University

[3]New York-Presbyterian Hospital and Weill Cornell Medical College

Abstract

BACKGROUND: Body dysmorphic disorder (BDD) is a severe and undertreated condition. Although cognitive behavioral therapy (CBT) is the first-line psychosocial treatment for this common disorder, how the intervention works is insufficiently understood. Specific pathways have been hypothesized, but only one small study has examined the precise nature of treatment effects of CBT, and no prior study has examined effects of supportive psychotherapy (SPT).

METHODS: This study re-examined a large trial (n=120) comparing CBT to SPT for BDD. Network intervention analyses were used to explore symptom-level data across time. We computed mixed graphical models at multiple time points to examine relative differences in direct and indirect effects of the two interventions.

RESULTS: In the resulting networks, CBT and SPT appeared to differentially target certain symptoms. Largest differences included CBT increasing efforts to disengage from and restructure

unhelpful thoughts and resist BDD rituals, while SPT was directly related to improvement in BDD-related insight. Additionally, the time course of differences aligned with the intended targets of CBT; cognitive effects emerged first and behavioral effects second, paralleling cognitive restructuring in earlier sessions and the emphasis on exposure and ritual prevention in later sessions. Differences in favor of CBT were most consistent for behavioral targets.

CONCLUSIONS: CBT and SPT primarily affected different symptoms. To improve patient care, the field needs a better understanding of how and when BDD treatments and treatment components succeed. Considering patient experiences at the symptom level and over time can aid in refining or reorganizing treatments to better fit patient needs.

Keywords

body dysmorphic disorder; cognitive behavioral therapy; supportive psychotherapy; network analysis; mechanisms

Body dysmorphic disorder (BDD) has a current nationwide prevalence of 1.7–2.9% in the general population and is far more common than this in psychiatric inpatient, psychiatric outpatient, and other medical settings (e.g., dermatology, cosmetic surgery) (Buhlmann et al., 2010; Schieber, Kollei, de Zwaan, & Martin, 2015; Veale, Gledhill, Christodoulou, & Hodsoll, 2016). Preoccupations with nonexistent or slight appearance flaws and related, compulsive rituals are usually accompanied by impaired social, occupational, or other role functioning, reduced quality of life, and elevated rates of psychiatric comorbidities and psychiatric hospitalizations (Phillips, 2000; Phillips, Menard, Fay, & Weisberg, 2005). More than three quarters of individuals with BDD report lifetime suicidal ideation and more than one quarter suicide attempts (Phillips, Coles, et al., 2005). Despite this serious public health concern, BDD remains underrecognized and undertreated (Phillips, Pagano, & Menard, 2006).

The recommended psychosocial treatment for BDD is cognitive behavioral therapy (CBT; Rasmussen, Gomez, & Wilhelm, 2017). Multiple clinical trials have demonstrated the efficacy of CBT for BDD (Harrison, Fernández de la Cruz, Enander, Radua, & Mataix-Cols, 2016; Wilhelm et al., 2019). However, not all patients respond to treatment, let alone experience full remission (Krebs et al., 2017; Veale, Miles, & Anson, 2015; Weingarden et al,. 2021). Furthermore, a majority of patients do not receive CBT (Phillips, Menard, Quinn, Didie, & Stout, 2013). Although less well studied, supportive psychotherapy (SPT) is the most common therapy for this condition (Phillips, Menard, Quinn, Didie, & Stout, 2013). Yet to our knowledge, only one paper has examined mechanisms of CBT's effects (Fang et al., 2020), and no prior study has examined mechanisms of SPT's impact. Thus, more work is needed to understand *how* treatments impact BDD symptoms in order to improve treatment delivery and outcomes.

Although precise mechanisms of treatment effects remain unknown, there are quite specific hypothesized pathways for symptom change in response to CBT (Wilhelm, Phillips, Fama, Greenberg, & Steketee, 2011). Briefly, the CBT model outlines that individuals with BDD selectively attend to facets of their appearance, fueling exaggerated, inaccurate, and intrusive negative beliefs and corresponding emotions. Efforts to neutralize these responses with

ritualistic behaviors (such as excessive grooming) or with avoidance (for example, of social situations) ultimately reinforce selective attention to perceived appearance flaws, distressing thoughts, and strong negative emotions (Feusner, Neziroglu, Wilhelm, Mancusi, & Bohon, 2010; Veale, 2004). Each CBT skill is intended to modify very specific targets, or components of this model, at the time it is introduced (e.g., cognitive restructuring should change responses to and frequency of maladaptive thoughts). As noted above, however, only one paper has tested such hypotheses. Fang and colleagues examined cognitive and behavioral factors theorized to maintain BDD psychopathology in a small sample of patients (n=45) undergoing CBT (Fang et al., 2020). Within-subject changes in maladaptive thoughts and related behaviors mediated symptom improvement. The authors concluded that treatments targeting specific thoughts, rituals, and avoidance symptoms—understood to be core symptoms maintaining BDD—may yield the strongest overall outcomes.

From a clinical perspective, it seems intuitive that BDD symptoms interact to activate, exacerbate, or maintain one another. For example, reducing time spent examining one's appearance would be expected to lead to reductions in appearance-related distress, or increases in appearance-related distress would be expected to lead to increases in ritualizing, such as checking one's appearance in the mirror or seeking reassurance from others. Thus, it should be useful to formally consider BDD from a network perspective, whereby the disorder is not some entity productive of symptoms, but rather symptoms and their interrelationships constitute the disorder (Schmittmann et al., 2013; Summers et al., 2020). In other words, it is the activated web of co-occurring, interrelated, dysregulated processes that precipitates harmful outcomes. It seems logical that targeting theoretically central processes—or those exhibiting the most influence on other symptoms or the larger system— could facilitate broader recovery or that a failure to do so would inhibit it. This has yet to be tested in BDD. Such an examination is necessary because, for example, it is possible that CBT does not actually act on these specific symptoms. It is possible that CBT might benefit patients in non-specific or variable ways, similar to how SPT benefits patients with other disorders (e.g., via common factors); expectations for treatment, psychoeducation about BDD, and acceptance and empathy from a clinician might alone have powerful effects on symptoms (Grencavage & Norcross, 1990; Wampold, 2015). As such, focusing on particular treatment modules or skills might not generate better results. We aim to build on this work to further understand whether CBT has specific effects on BDD symptoms.

One limitation of existing work is the level of analysis typically employed in clinical trials. By reducing symptoms to a binary variable (presence of a diagnosis) or severity index (sum score), or by focusing only on post-treatment outcomes, we may be missing critical mechanisms of change or stagnation. For example, there are many meaningfully different ways to obtain similar scores on a severity measure (e.g., low levels of all symptoms, high levels of a few symptoms, similar levels but for different combinations of symptoms). Additionally, because CBT involves multiple skills modules, symptoms might change at different points in treatment. And, some symptoms may not change at all. It is also possible that CBT effectively targets only certain BDD symptoms and thus only benefits patients whose pathology is driven by such symptoms.

The present report addresses two questions: (1) Does CBT meaningfully target specific symptoms, compared to SPT? (2) Do CBT-driven changes in BDD symptoms occur in the order predicted by the course of CBT treatment for BDD? We consider unique, direct connections between received treatment and specific symptom changes as well as through indirect symptom-to-symptom pathways. To this end, we examine patient experiences across multiple time points during treatment. In recent years, the advent of network analytic approaches in clinical science has provided a new lens for considering such symptom-level relationships rather than relying on sum scores (Borsboom & Cramer, 2013). This approach emphasizes the multidimensionality of psychopathology and the interplay of observable components (e.g., symptoms) (McNally, 2016). In this report, by considering treatment as a factor influencing the network of interacting symptoms, we can trace plausible direct and indirect therapeutic mechanisms (Blanken et al., 2019). This novel perspective on symptom change in clinical trials could help to explain differential responses to treatment, inform treatment refinement and development, and guide more personalized, precision medicine. To improve patient care, we need to better understand how, when, and for whom different treatments and treatment components work.

This is the first report to use network intervention analysis to examine mechanisms of change in BDD. We use data from the only clinical trial comparing therapist-delivered CBT to SPT for BDD (Wilhelm et al., 2019). Comparing CBT to an active, alternative psychotherapy enables us to better isolate unique effects of CBT rather than common factors of psychotherapy. Briefly, as previously reported, this trial found that CBT was significantly more effective than SPT at one treatment site but not the other; response rates at 24 weeks were 84.6% at one site and 83.3% at the second site for CBT, and 69.2% and 45.5% for SPT, respectively (Wilhelm et al., 2019). A network approach may provide additional nuance in understanding this outcome; the paths to recovery *should* look different with each treatment, given hypothesized differences in what the two treatments specifically target. For example, although CBT and SPT groups may not significantly differ in overall BDD severity at treatment midpoint, the different treatments may have been enacting meaningful changes in different symptoms, which may have set off distinct cascades of changes thereafter. More specifically, we would expect CBT to directly impact cognitive targets (willingness and ability to disengage from and restructure BDD-related thoughts) early in treatment and behavioral targets (reducing BDD repetitive and avoidant behaviors) later in treatment, in parallel with the order of CBT modules used in this study. In contrast, symptoms which are not as explicitly targeted by CBT skills, such as distress, may be more indirectly impacted by changes in other symptoms. Because SPT uses a nondirective approach, encouraging self-esteem and offering coping skills in response to patients' unique and ongoing stressors, it is challenging to predict the timing or nature of its impact. We therefore did not have a priori hypotheses about SPT mechanisms. Understanding *how* treatments differentially influence specific symptoms may help future clinicians better tailor interventions to individual presentations.

Method

Sample

The sample included 120 adult participants, the majority of whom identified as white and female (mean age = 34.0, SD = 13.1, range = 18–77). Demographics of the sample are included in Table 1. Eligible participants had a primary diagnosis of BDD (DSM-IV) with at least moderate severity (BDD-YBOCS score 24), did not express current active suicidality or other severe symptoms meriting a higher level of care per screening procedures and clinician judgment, did not evidence significant cognitive impairment (IQ 80, WASI), and did not present with a current substance use disorder, mania, psychosis, borderline personality disorder, or other illness that would interfere with participation. Participants had not previously had 10 or more sessions of CBT for BDD and were not currently receiving psychotherapy. Within the final sample, only four participants endorsed prior sessions of CBT (range= 3–8).[1] Participants could continue taking psychotropic medications if doses were stable for at least two months prior to study entry and remained so for the study duration; they could not begin taking new psychotropic medication during the study.

Procedure

CBT and SPT were administered as 22 weekly sessions over 24 weeks. The final two sessions were biweekly. All study procedures were approved by the institutional review boards at Massachusetts General Hospital and Rhode Island Hospital. All patients provided informed consent. Core CBT components included psychoeducation about BDD, goal setting, cognitive restructuring, advanced cognitive strategies to broaden the basis of self-worth and self-compassion, exposure and ritual prevention, mindfulness and attentional/visual retraining, and relapse prevention (Wilhelm, Phillips, & Steketee, 2013). SPT was enhanced, in that it also included psychoeducation about BDD; it was then nondirective, emphasized the therapeutic relationship, and focused on self-esteem, coping skills, and processing current life issues (Pinsker, 1997). Assessments occurred at nine time points: baseline (week 0), monthly during treatment (weeks 4, 8, 12, 16, 20, 24), and at 3- and 6-month follow-up visits (weeks 37 and 50). Of the 120 randomized participants, 92 individuals (76.7%) completed treatment.

The present report re-examines the trial's primary outcome variable, BDD severity, as measured by the Yale-Brown Obsessive Compulsive Scale Modified for BDD (BDD-YBOCS; Phillips et al., 1997). The BDD-YBOCS is a 12-item rater-administered semi-structured scale with strong reliability and sensitivity to change (Phillips, Hart, & Menard, 2014). Items 1–5 assess appearance-related thoughts: *time occupied, interference in functioning due to BDD thoughts, distress due to thoughts*, *resistance* against thoughts (efforts to disregard, challenge, or turn attention from the thoughts), and *control* over thoughts (ability to successfully disengage from appearance-related thoughts). Items 6–10 assess BDD-related repetitive behaviors (rituals, compulsions), such as excessive mirror checking, skin picking, comparing with others, and excessive grooming, and are similar

---

[1]The exact number of prior CBT sessions for one participant was not unknown; however, it was confirmed to be less than 10 per the exclusion criteria.

to items 1–5 (*time spent* performing the behaviors, *interference in functioning* due to the behaviors, *distress* experienced if the behaviors are/were prevented, and *resistance* of and *control* over the behaviors). Item 11 assesses BDD-related *insight*, and item 12 assesses overall BDD-related *avoidance* of activities, such as work or social activities. Higher scores reflect greater symptom severity (e.g., worse insight, more avoidance). Further details about the clinical trial are available in the primary outcomes paper (Wilhelm et al., 2019).

Data Analysis

Nodes (i.e., variables), *edges* (i.e., pairwise connections between variables), and *location* (i.e., proximity of nodes in visual representations) are three core features of network analyses. In this report, nodes comprise BDD-YBOCS items and treatment allocation. Edges and location are defined below. All analyses were conducted in R.

Data preparation.—Before including any BDD-YBOCS items as unique nodes, we used the *goldbricker* function from the R package *networktools* (Bernstein, Heeren, & McNally, 2019; Jones, 2018) to search for "redundant" items. Specifically, this approach identifies pairs of items that are both highly intercorrelated (r >.50) and show highly similar patterns of relationships to other items (i.e., > 85% of correlations with other items do not significantly differ for a pair) using the Hittner method for comparing dependent correlations (Hittner, May, & Silver, 2003). We identified interference from repetitive behaviors (BDD-YBOCS item 7) and BDD-related avoidance of life activities (item 12) as highly correlated and thus statistically redundant at baseline. Per the *net_reduce* function, redundancy was resolved by retaining avoidance as the more unique variable. Additionally, variables were roughly normally distributed (skewness .5, with the exception of resistance of repetitive behaviors (item 9) = .71), and therefore no variable transformations were conducted.

Network intervention analyses.—Network intervention analysis allows for inspection of treatment-induced changes in specific symptoms over time and characterizes direct versus indirect treatment effects (Blanken et al., 2019). First pioneered by Blanken and colleagues to examine the impact of CBT-I on insomnia and depression (compared to no treatment), this method has since been used in four studies of treatments for pediatric anxiety (Cervin et al., 2020) and depression (Boschloo et al., 2019; Curtiss et al., 2021; Mullarkey, Stein, Pearson, & Beevers, 2020). In the current report, networks were constructed for each assessment point. BDD-YBOCS items were entered into models as continuous variables. Each network also included an additional binary treatment allocation node.

Analyses followed the methods outlined by Blanken and colleagues (Blanken et al., 2019). At each time point, we used the package *mgm* (Haslbeck & Waldorp, 2020) to compute mixed graphical models (MGMs), which depict pairwise associations among nodes and control for effects of all other nodes (Epskamp & Fried, 2018). Graphical LASSO (Least Absolute Shrinkage and Selection Operator) was used to regularize models (Friedman, Hastie, & Tibshirani, 2011). Following Blanken and colleagues, we used the data-driven cross-validation approach to select the tuning parameter and therefore to determine the extent of regularization. We used ten folds and mean squared error as goodness-of-fit metric. In the resulting networks, thicker edges correspond to larger partial correlations. To ease

comparison across networks, we adjusted all layouts to be similar. Therefore, the relative proximity of nodes in a given network should not be overinterpreted. Instead, the resulting layout reflects the average across all networks, so that nodes that are closest together are generally the most highly intercorrelated across all time points; however, they are not necessarily the most highly intercorrelated at any one time point. We also computed strength centrality indices to further approximate the relative importance of each node within the overall network. Values reflect the sum of the edge weights connected to each node. Higher values indicate greater centrality and thus greater potential importance.

Relative size of a node signifies differences in changes in symptom severity from baseline by treatment. We standardized item means for each time point using pooled baseline means and standard deviations. Consequently, negative values indicate declines in symptom severity, and positive values indicate increases in severity. We then computed differences between the treatment groups. Looking across time points allows us to observe the progression of treatment-induced changes.

To evaluate accuracy of edge weight estimates (i.e., magnitude of associations between nodes), we used the *resample* function from the *mgm* package. For each network, we ran 100 bootstrap samples and plotted resulting 5% and 95% quantiles of the distribution. The proportion of non-zero edge weight estimates were also reported. Regularization used during network estimation notably biases all sampling distributions towards zero. Consequently, 5% and 95% quantiles including zero are not concerning (Blanken et al., 2019).

Results

Network Estimation

Figure 1 shows the 9 regularized networks estimated for each assessment point. Circular nodes indicate items from the BDD-YBOCS, and the square node indicates treatment (CBT or SPT). Edges between nodes represent conditional dependence relations, or the unique associations among the variables controlling for all the other variables in the network.

In Figure 1, among circular nodes, green edges (lines) represent positive associations, and red edges represent negative associations. Edge thickness is proportional to the relative strength of the association. For example, the most consistent and largest positive associations across time points are between interference from thoughts (ThInt; BDD-YBOCS item 2) and avoidance of life activities (Avoid; item 12) as well as between time occupied by appearance-related thoughts (ThTim; item 1) and time occupied by BDD repetitive behaviors (BeTim; item 6). Moderate associations were consistently identified connecting resistance and control, both for thoughts and behaviors, and between control of thoughts and distress related to thoughts. Interestingly, connections emanating from poor insight (Insig; item 11) were highly variable in terms of strength and destination. With few exceptions, edges among BDD-YBOCS items were green, indicating positive associations.

For edges between the square treatment node and circular symptom nodes, green indicates that relative to SPT, CBT is associated with more severe symptoms, and red indicates that CBT is associated with less severe symptoms (see Figure 1). As would be expected,

treatment allocation was unrelated to symptoms at week 0 (pre-treatment). During active treatment, relative to SPT, CBT was associated with more thought resistance at week 4 (ThRes; item 4), more repetitive behavior resistance at week 16 (BeRes; item 9), and less time spent engaged in repetitive behaviors at week 20 and 24 (BeTim; item 6). SPT was associated with greater insight at weeks 4, 16, and 24 (Insig; item 11). During follow-up, relative to SPT, CBT was associated with less time engaged in BDD thoughts at week 37 (ThTim; item 1), and less distress and more resistance related to BDD repetitive behaviors at week 50 (BeDis and BeRes; items 8 and 9) (see Figure 1).

Strength centrality estimates for symptom nodes are depicted in Figure S1. Metrics support that early in treatment cognitive nodes are most central, later in treatment behavioral modes are most central, and after treatment control of thoughts and behaviors is most central. In contrast, insight exhibits inconsistent, and often low, centrality across time.

Group Differences in Symptom Change

Node size corresponds to relative difference in symptom severity change between CBT and SPT groups from baseline. Larger size indicates that the CBT group experienced greater improvement in a given symptom than the SPT group; smaller size indicates that the SPT group experienced greater improvement. Group differences are visualized in Figure 1 and summarized in Table 2. Average symptom levels by group and over time are visualized in Supplementary Figure S2. Although standardized group differences are small for a number of items, particularly in early weeks, a few notable trends stand out. First, as expected there is no clear pattern to the direction of group differences at week 0. However, as treatment progressed, with the exception of poor insight (particularly earlier in treatment), observed differences in symptom change consistently trend in favor of CBT, including during post-treatment weeks. Interestingly, node size for control of thoughts (item 5), time engaged in thoughts (item 1), control of repetitive behaviors (item 10), and time engaged in repetitive behaviors (item 6) grow most robustly across treatment, particularly in latter assessments, indicating that CBT was particularly efficacious for these symptoms relative to SPT.

Edge Weight Accuracy

Plots depicting 5% and 95% quantiles of the sampling distributions and proportion of estimates whose values are nonzero are included in Supplementary Figures S3–11. Keeping in mind the bias towards zero, edge weights surviving regularization and appearing in the estimated networks were adequate. With the present sample size and relatively narrow range of estimated edge weights, we do not overinterpret the ordering of edge weights (Burger et al., 2020).

Discussion

Analyses support that CBT and SPT differentially target specific BDD symptoms. The strongest observed differences were that CBT was directly associated with more adaptive efforts to relate to one's thoughts and behaviors (e.g., challenge or disengage from unhelpful thoughts, resist the urge to ritualize) than SPT, while SPT was directly associated with better BDD-related insight earlier in treatment than CBT. However, greater improvements

in insight began emerging later in treatment and during follow-up among participants in the CBT group compared to the SPT group (Table 2 and Figure S2).

The time course of these differences align with the intended targets of CBT. Specifically, cognitive effects emerged first and behavioral effects second, paralleling the use of cognitive restructuring in earlier CBT sessions and the emphasis on exposure and ritual prevention in later sessions. Furthermore, direct effects on efforts to resist repetitive behaviors emerged before reduced time spent engaged in such behaviors. In other words, compared to SPT, CBT could be eliciting greater awareness of maladaptive behavioral patterns (rituals) and willingness to reduce them; following this shift, patients receiving CBT could subsequently also be more likely to ultimately succeed in ritual reduction. Although more idiographic work is needed, these patterns lend support to the hypothesized mechanisms of CBT's therapeutic effects, namely that CBT changes two key maintenance factors: maladaptive BDD-related beliefs and BDD rituals (Fang et al., 2020; Wilhelm, Buhlmann, Hayward, Greenberg, & Dimaite, 2010).

Findings support including both cognitive and behavioral strategies when treating BDD. However, more work that isolates each individual treatment component further is merited. With current treatment protocols, it is difficult to capture the extent to which observed behavioral changes are only direct products of later behavioral modules versus are secondary to earlier cognitive changes. Moreover, the unique treatment effects of CBT versus SPT did not emerge as consistently or pervasively as one might have expected. Thus, we cannot overlook the meaningfulness of shared factors, such as validating patients' experiences and still encouraging them to reexamine their symptoms, with or without a theoretical model for doing so. More importantly, this should push researchers to examine for whom a given intervention is more likely to lead to positive change and consider more idiographic, process-focused approaches to mapping changes across treatment. This level of analysis could ideally uncover specific treatment strategies to target specific change processes and conditions under which they work best.

Still, results seem to highlight the unique impact of the behavioral components of CBT, compared to SPT, in reducing rituals. The observed treatment differences in favor of CBT were particularly strong for behavioral targets. In practice, formally specifying and countering repetitive behaviors is the most significant difference between CBT and SPT. Although SPT does not involve explicit instruction in cognitive models or skills, its emphasis on self-esteem and processing life issues could potentially, to some degree, resemble CBT skills of increasing cognitive flexibility and modifying core beliefs, which for many patients include beliefs about self-worth. It is worth noting, however, that high levels of treatment fidelity were attained during the study, indicating that therapists providing each treatment did not use strategies from the other treatment (Wilhelm et al., 2019). Results support that CBT and SPT provide distinct therapeutic benefits beyond any shared, or common, factors.

Interestingly, direct effects of treatment allocation (i.e., number and magnitude of edge weights between the treatment node and symptom nodes) were moderate, with some of the strongest outcomes favoring CBT emerging as indirect effects. Findings bolster our

confidence that CBT is effective in modifying patients' relationships to their thoughts and rituals, aligning with the suggestion that treatment-induced repeated practice (e.g., resisting rituals) is ultimately what leads to decreases in distress, increased control, self-efficacy, and functioning. It also highlights areas requiring more research, namely to replicate and further explain how these downstream cascades in symptom improvement occur. For example, more work is needed to understand how practicing stepping back from and evaluating unhelpful thoughts ultimately translates to the thoughts themselves being less distressing; in the present data, it is unclear to what extent thought content changes to be inherently less negative or whether patients have less negative or intense reactions to the same thoughts (e.g., understanding that the thoughts are less important or valid than previously believed, or cultivating greater acceptance or tolerance of painful thoughts without trying to modify their meaning), or both. These next steps would further inform specific targets for enhancing treatment outcomes.

Results are consistent with the main findings of the trial showing that CBT was significantly more effective than SPT for response rates at one of the two treatment sites (Wilhelm et al., 2019). Current results may also shed light on why SPT still conferred benefits for some patients and CBT may not have markedly benefited all patients. In this way, results could reveal important directions for trying to improving BDD treatments, including when and how to better directly address poor insight and do so earlier in treatment. A majority of individuals with BDD, including in this trial, display poor insight, and more than one third present with absent insight (i.e. delusional beliefs) about their appearance (Eisen, Phillips, Coles, & Rasmussen, 2004). More work is needed to fully understand treatment interference and malleability of poor insight. This report does not provide a clear picture. Some (but not all) past clinical trials have found that poorer BDD-related insight predicts worse outcomes (Greenberg, Phillips, Steketee, Hoeppner, & Wilhelm, 2019). Notably, CBT has been shown to improve BDD-related insight in prior studies (Enander et al., 2016; Wilhelm et al., 2014). And in the primary report from this study, patients in the CBT group did report improvements in insight overall (Wilhelm et al., 2019); in fact, later in the study and at post-treatment follow-up, these changes were trending in favor of the CBT group (Table 2 and Figure S2).

Poor insight was strongly, although inconsistently, connected to other symptoms throughout the study. Consequently, there are not well-defined hypotheses for how poor insight maintains BDD or how and when it is best to intervene based on the present results. One possibility is that poor insight interferes with motivation or general engagement with learning skills, or leads to greater "white knuckling" (tensely pushing through an exposure) or other ineffective ways of resisting BDD rituals (Abramowitz, 2006; Foa et al., 1999). It seems plausible that if all therapists could more directly and effectively address insight earlier in treatment more robust, reliable outcomes could emerge (Eisen et al., 2004). Enhancing attentional/perceptual retraining may be promising, as visual processing aberrations likely contribute to poor/absent insight in BDD (Li et al., 2015; Phillips, 2017). Drawing from the OCD literature, more tailored exposure strategies and integration of motivational interviewing techniques should also be tested (Maltby & Tolin, 2005; Middleton & Hezel, 2019). Furthermore, understanding how SPT achieves earlier shifts in insight could provide meaningful guidance, such as adding a supportive, insight-focused

module to accompany psychoeducation. For example, SPT's early emphasis on coping (e.g., social support) and enhancing self-esteem, which is typically low in BDD (Phillips, Pinto, & Jain, 2004), might have led to sooner shifts in insight.

Although attempting to change insight is not a major focus of the CBT used in this study, the psychoeducational, motivational, cognitive restructuring, behavioral experiments, and attentional/perceptual retraining strategies are expected to have positive effects on BDD-related insight. However, present results suggest that this improvement later in treatment and post-treatment may have been less a direct consequence of treatment, evidenced by the absence of a positive direct effect of CBT treatment allocation, and instead be better accounted for by larger shifts in other symptoms or overall severity, similar to patterns observed in OCD (Eisen, Rasmussen, Phillips, et al., 2001). For example, more avoidance behavior and less willingness to engage in exposure and ritual prevention tend to strongly correlate with low insight; through behavioral experiments and ritual prevention, however, patients directly approach feared situations and with repetition often realize that their beliefs are unreliable or ineffective (Foa, Abramowitz, Franklin, & Kozak, 1999; Selles et al., 2020).

Based on our results, we hypothesize that CBT yielded more consistent positive outcomes because the symptoms it directly targets are more widely and consistently connected to others. For example, treatment allocation appeared to most directly impact resistance of BDD thoughts and behaviors, signified by the direct edges between the treatment node and the two resistance nodes. Indirect effects, or ostensible benefits of CBT via the above direct connections, then emerged particularly towards the end of treatment; CBT increasingly outweighed SPT in positive changes in control over thoughts and behaviors and reduced time spent on BDD-related thoughts and rituals. Results suggest that CBT could lead to these later sustained advantages for control and time through its earlier impact on efforts to resist (Wilhelm et al., 2013). In this way, positive downstream cascades (e.g., from awareness of rituals to successful control and less time engaged in them) and thus broader recovery could be more likely, in line with the CBT model. No such evidence of specific cascading effects was evident for SPT or insight. Strength centrality estimates further align with this interpretation as cognitive processes were most central early in the trial and behavioral processes later in the trial.

Regarding limitations, it is unlikely that the BDD symptom variables selected in this report perfectly capture all variables relevant to understanding how CBT or SPT benefits some patients more than others. The BDD-YBOCS largely assesses appearance-related beliefs and repetitive behaviors, which could bias current results to illuminating the positive effects of receiving CBT. For example, other variables of interest for future study could include attention, visuospatial, or other related performance metrics (Buhlmann, McNally, Wilhelm, & Florin, 2002); neurobiological factors (Grace, Labuschagne, Kaplan, & Rossell, 2017); or self-esteem (Buhlmann, Teachman, Gerbershagen, Kikul, & Rief, 2008). Also, the diversity of the sample was limited in terms of race, ethnicity, and gender. Future studies are needed to understand whether the current findings generalize beyond the present group. Additionally, as we favored robust effects and minimizing false positives, it is possible that models missed some small treatment effects or associations between nodes. Thus,

edges should be interpreted as compelling pathways to be followed up on rather than the only possible pathways. We hope this work will motivate further, more targeted study of individual components of treatment. A strength of this work is the inclusion of longitudinal data. However, because this statistical approach is quite young, we have yet to determine the optimal timescale at which to examine treatment effects.

In conclusion, this approach sheds light on how two very different psychotherapeutic approaches affect BDD outcomes. Although additional research designed for network analyses is required to replicate findings and resolve remaining questions and limitations from this initial examination, this approach is promising. Ultimately, understanding *how* treatment effects unfold over time can help clinicians set clearer expectations for patients and enhance treatment credibility. This breakdown could aid in refining or reorganizing modular treatments to better fit patient needs, as we could be more confident regarding what factors treatment components are directly influencing. By considering patient experiences at the symptom level and at multiple intervals, we could ideally identify patterns of symptoms that predict better responses to one treatment over the other. This line of work could support more precision or personalized medicine.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

References

Abramowitz JS (2006). The psychological treatment of obsessive—compulsive disorder. The Canadian Journal of Psychiatry, 51, 407–416. [PubMed: 16838822]

Bernstein EE, Heeren A, & McNally RJ (2019). Reexamining trait rumination as a system of repetitive negative thoughts: A network analysis. Journal of Behavior Therapy and Experimental Psychiatry, 63, 21–27. 10.1016/j.jbtep.2018.12.005 [PubMed: 30590225]

Blanken TF, Van Der Zweerde T, Van Straten A, Van Someren EJW, Borsboom D, & Lancee J (2019). Introducing Network Intervention Analysis to Investigate Sequential, Symptom-Specific Treatment Effects: A Demonstration in Co-Occurring Insomnia and Depression. Psychotherapy and Psychosomatics, 88, 52–54. 10.1159/000495045 [PubMed: 30625483]

Borsboom D, & Cramer AOJ (2013). Network Analysis: An Integrative Approach to the Structure of Psychopathology. Annual Review of Clinical Psychology, 9(1), 91–121. 10.1146/annurev-clinpsy-050212-185608

Boschloo L, Cuijpers P, Karyotaki E, Berger T, Moritz S, Meyer B, & Klein JP (2019). Symptom-specific effectiveness of an internet-based intervention in the treatment of mild to moderate depressive symptomatology: The potential of network estimation techniques. Behaviour Research and Therapy, 122, 103440. 10.1016/j.brat.2019.103440 [PubMed: 31542565]

Buhlmann U, Glaesmer H, Mewes R, Fama JM, Wilhelm S, Brähler E, & Rief W (2010). Updates on the prevalence of body dysmorphic disorder: A population-based survey. Psychiatry Research, 178, 171–175. 10.1016/j.psychres.2009.05.002 [PubMed: 20452057]

Buhlmann U, Teachman BA, Gerbershagen A, Kikul J, & Rief W (2008). Implicit and explicit self-esteem and attractiveness beliefs among individuals with body dysmorphic disorder. Cognitive Therapy and Research, 32, 213–225.

Cervin M, Storch EA, Piacentini J, Birmaher B, Compton SN, Albano AM, … Kendall PC (2020). Symptom-specific effects of cognitive-behavioral therapy, sertraline, and their combination in a large randomized controlled trial of pediatric anxiety disorders. Journal of Child Psychology and Psychiatry, 61, 492–502. 10.1111/jcpp.13124 [PubMed: 31471911]

Curtiss JE, Wallace B, Fisher LB, Nyer M, Jain F, Cusin C, & Pedrelli P (2021). Change Processes in Cognitive Behavioral Therapy and Motivational Interviewing for Depression and Heavy Alcohol Use: A Network Approach. Journal of Affective Disorders Reports, 3, 100040. 10.1016/j.jadr.2020.100040

Eisen JL, Phillips KA, Coles ME, & Rasmussen SA (2004). Insight in Obsessive Compulsive Disorder and Body Dysmorphic Disorder. Comprehensive Psychiatry, 45, 10–15. 10.1016/j.comppsych.2003.09.010 [PubMed: 14671731]

Eisen JL, Rasmussen SA, Phillips KA, Price LH, Davidson J, Lydiard RB, ... & Piggott T (2001). Insight and treatment outcome in obsessive-compulsive disorder. Comprehensive Psychiatry, 42, 494–497. [PubMed: 11704942]

Enander J, Andersson E, Mataix-Cols D, Lichtenstein L, Alström K, Andersson G, … Rück C (2016). Therapist guided internet based cognitive behavioural therapy for body dysmorphic disorder: Single blind randomised controlled trial. BMJ, 4, e005923. 10.1136/bmj.i241

Epskamp S, & Fried EI (2016). A tutorial on regularized partial correlation networks. ArXiv Preprint ArXiv:1607.01367.

Fang A, Steketee G, Keshaviah A, Didie E, Phillips KA, & Wilhelm S (2020). Mechanisms of Change in Cognitive Behavioral Therapy for Body Dysmorphic Disorder. Cognitive Therapy and Research, 44, 596–610.

Fang A, & Wilhelm S (2015). Clinical features, cognitive biases, and treatment of body dysmorphic disorder. Annual Review of Clinical Psychology, 11, 187–212. 10.1146/annurev-clinpsy-032814-112849.

Foa EB, Abramowitz JS, Franklin ME, & Kozak MJ (1999). Feared consequences, fixity of belief, and treatment outcome in patients with obsessive-compulsive disorder. Behavior therapy, 30(4), 717–724.

Friedman J, Hastie T, & Tibshirani R (2011). glasso: Graphical lasso-estimation of Gaussian graphical models.

Grant JE, & Phillips KA (2005). Recognizing and treating body dysmorphic disorder. Annals of Clinical Psychiatry, 17. 10.1080/10401230500295313

Greenberg JL, Phillips KA, Steketee G, Hoeppner SS, & Wilhelm S (2019). Predictors of Response to Cognitive-Behavioral Therapy for Body Dysmorphic Disorder. Behavior Therapy, 50, 839–849. 10.1016/j.beth.2018.12.008 [PubMed: 31208692]

Grencavage LM, & Norcross JC (1990). Where Are the Commonalities Among the Therapeutic Common Factors? Professional Psychology: Research and Practice, 21, 372–378. 10.1037/0735-7028.21.5.372

Harrison A, Fernández de la Cruz L, Enander J, Radua J, & Mataix-Cols D (2016). Cognitive-behavioral therapy for body dysmorphic disorder: A systematic review and meta-analysis of randomized controlled trials. Clinical Psychology Review, 48, 43–51. 10.1016/j.cpr.2016.05.007 [PubMed: 27393916]

Haslbeck JM, & Waldorp LJ (2018). How well do network models predict observations? On the importance of predictability in network models. Behavior Research Methods, 50(2), 853–861. 10.3758/s13428-017-0910-x. [PubMed: 28718088]

Haslbeck JMB, & Waldorp LJ (2020). MGM: Estimating time-varying mixed graphical models in high-dimensional data. Journal of Statistical Software. 10.18637/jss.v093.i08

Hittner JB, May K, & Silver NC (2003). A monte carlo evaluation of tests for comparing dependent correlations. Journal of General Psychology. 10.1080/00221300309601282

Jones P (2018). Networktools: Tools for Identifying Important Nodes in Networks. R Package Version 1.1.1. https://CRAN.R-project.org/package=networktools.

Krebs G, de la Cruz LF, Monzani B, Bowyer L, Anson M, Cadman J, … Mataix-Cols D (2017). Long-Term Outcomes of Cognitive-Behavioral Therapy for Adolescent Body Dysmorphic Disorder. Behavior Therapy, 48, 462–473. 10.1016/j.beth.2017.01.001 [PubMed: 28577583]

Li W, Lai TM, Bohon C, Loo SK, McCurdy D, Strober M, ... & Feusner J (2015). Anorexia nervosa and body dysmorphic disorder are associated with abnormalities in processing visual information. Psychological Medicine, 45(10), 2111. [PubMed: 25652023]

Maltby N, & Tolin DF (2005). A brief motivational intervention for treatment-refusing OCD patients. Cognitive Behaviour Therapy, 34, 176–184. [PubMed: 16195056]

McNally RJ (2016). Can network analysis transform psychopathology? Behaviour Research and Therapy, 86, 95–104. 10.1016/j.brat.2016.06.006 [PubMed: 27424882]

Middleton RR, & Hezel DM (2019). Utilizing exposure and response prevention to address poor insight in obsessive-compulsive disorder. Journal of Cognitive Psychotherapy, 33(3), 213–227. [PubMed: 32746428]

Mullarkey MC, Stein AT, Pearson R, & Beevers CG (2020). Network analyses reveal which symptoms improve (or not) following an Internet intervention (Deprexis) for depression. Depression and Anxiety, 37, 115–124. 10.1002/da.22972 [PubMed: 31710772]

Phillips KA (2000). Quality of life for patients with body dysmorphic disorder. Journal of Nervous and Mental Disease, 188, 170–175. 10.1097/00005053-200003000-00007 [PubMed: 10749282]

Phillips KA, Coles ME, Menard W, Yen S, Fay C, & Weisberg RB (2005). Suicidal ideation and suicide attempts in body dysmorphic disorder. Journal of Clinical Psychiatry, 66, 717–725. 10.4088/JCP.v66n0607 [PubMed: 15960564]

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. 10.1016/j.jocrd.2014.04.004

Phillips KA, Hollander E, Rasmussen SA, Aronowitz BR, DeCaria C, & 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, Menard W, Fay C, & Weisberg R (2005). Demographic characteristics, phenomenology, comorbidity, and family history in 200 individuals with body dysmorphic disorder. Psychosomatics, 46, 317–325. 10.1176/appi.psy.46.4.317 [PubMed: 16000674]

Phillips KA, Pagano ME, & Menard W (2006). Pharmacotherapy for body dysmorphic disorder: Treatment received and illness severity. Annals of Clinical Psychiatry, 18, 251–257. 10.1080/10401230600948464 [PubMed: 17162625]

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. 10.1016/j.jpsychires.2005.10.001 [PubMed: 16309706]

Phillips KA, Menard W, Quinn E, Didie ER, & Stout RL (2013). A four-year prospective observational follow-up study of course and predictors of course in body dysmorphic disorder. Psychological Medicine, 43, 1109–1117. 10.1017/S0033291712001730 [PubMed: 23171833]

Phillips KA. (2017). Insight and delusional beliefs in body dysmorphic disorder. In Phillips KA (Ed.), Body Dysmorphic Disorder: Research and Clinical Advances. New York, NY: Oxford University Press.

Pinsker H (1997). A Primer of Supportive Psychotherapy. Hillsdale, N.J: The Analytic Press.

Rasmussen J, Gomez AF, & Wilhelm S (2017). Cognitive-behavioral therapy for body dysmorphic disorder. In Phillips KA (Ed.), Body Dysmorphic Disorder: Research and Clinical Advances. New York, NY: Oxford University Press.

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. 10.1016/j.jpsychores.2015.01.002 [PubMed: 25595027]

Summers BJ, Aalbers G, Jones PJ, McNally RJ, Phillips KA, & Wilhelm S (2020). A network perspective on body dysmorphic disorder and major depressive disorder. Journal of Affective Disorders, 262, 165–173. 10.1016/j.jad.2019.11.011 [PubMed: 31733461]

Veale D, Gledhill LJ, Christodoulou P, & Hodsoll J (2016). Body dysmorphic disorder in different settings: A systematic review and estimated weighted prevalence. Body Image, 18, 168–186. 10.1016/j.bodyim.2016.07.003 [PubMed: 27498379]

Veale D, Miles S, & Anson M (2015). Long-Term Outcome of Cognitive Behavior Therapy for Body Dysmorphic Disorder: A Naturalistic Case Series of 1 to 4 Years After a Controlled Trial. Behavior Therapy, 46, 775–785. 10.1016/j.beth.2015.06.003 [PubMed: 26520220]

Weingarden H, Hoeppner SS, Snorrason I, Greenberg JL, Phillips KA, & Wilhelm S (2021). Rates of remission, sustained remission, and recurrence in a randomized controlled trial of cognitive behavioral therapy versus supportive psychotherapy for body dysmorphic disorder. Depression and Anxiety.

Wilhelm S, Phillips KA, Fama JM, Greenberg JL, & Steketee G (2011). Modular cognitive–behavioral therapy for body dysmorphic disorder. Behavior Therapy, 42(4), 624–633. [PubMed: 22035991]

Wilhelm S, Phillips K, & Steketee G (2013). A cognitive behavioral treatment manual for body dysmorphic disorder. New York: Guilford Press.

Wilhelm S, Buhlmann U, Hayward LC, Greenberg JL, & Dimaite R (2010). A Cognitive-Behavioral Treatment Approach for Body Dysmorphic Disorder. Cognitive and Behavioral Practice, 17, 241–247. 10.1016/j.cbpra.2010.02.001

Wilhelm S, Phillips KA, Didie E, Buhlmann U, Greenberg JL, Fama JM, … Steketee G (2014). Modular cognitive-behavioral therapy for body dysmorphic disorder: A randomized controlled trial. Behavior Therapy, 45, 314–327. 10.1016/j.beth.2013.12.007 [PubMed: 24680228]

Wilhelm S, Phillips KA, Greenberg JL, O'Keefe SM, Hoeppner SS, Keshaviah A, … 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. 10.1001/jamapsychiatry.2018.4156 [PubMed: 30785624]
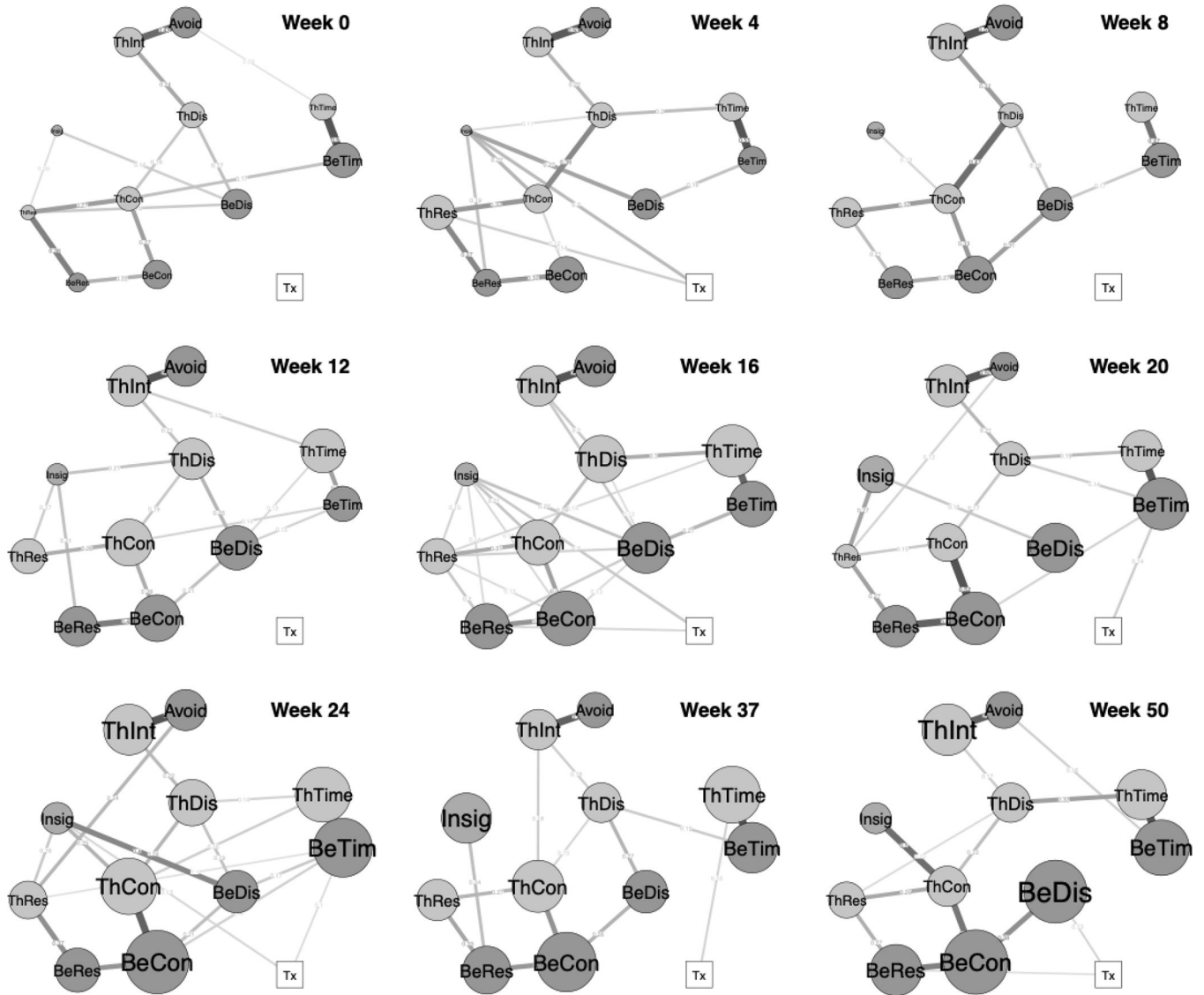
Figure 1.

Regularized networks by assessment point

Note. Pre-treatment=week 0 (n = 120). Treatment=weeks 4 (n = 107), 8 (n = 97), 12 (n = 89), 16 (n = 88), 20 (n = 87), and 24 (n = 92). Post-treatment=weeks 37 (n = 84) and 50 (n = 74). **Node colors** (online only) are as follows: yellow=thoughts, blue=behaviors, pink=insight, white=treatment. **Nodes** reflect treatment condition and BDD-YBOCS items: Tx=Treatment assignment. ThTim=time occupied by thoughts. ThInt=interference due to thoughts. ThDis=distress due to thoughts. ThRes=resistance of thoughts. ThCon=degree of control over thoughts. BeTim=time spent in repetitive behaviors (i.e., rituals, compulsions) related to body defects. BeDis=distress if repetitive behaviors are/were prevented. BeRes=resistance of repetitive behaviors. BeCon=degree of control over repetitive behaviors. Avoid=avoidance of life activities due to BDD. Insig= BDD-related insight. Higher scores indicate more severe symptoms (e.g., more avoidance, worse insight). Note that items 7 (interference in functioning due to repetitive behaviors) and

12 (avoidance) were collapsed as statistically redundant nodes. **Edges (lines)** represent pairwise associations. Green edges between symptom (circular) nodes=positive associations. Red edges between symptom nodes=negative associations. Green edges between the binary treatment (Tx; square) node and symptom nodes=CBT associated with more severe symptom than SPT. Red edges between the binary treatment (Tx; square) node and symptom nodes=CBT associated with less severe symptom than SPT. **Node size** reflects relative difference between treatments in change since baseline. Bigger size=CBT associated with greater symptom improvement than SPT. Smaller size=CBT associated with less symptom improvement than SPT. The size of node ThTim at week 1 reflects negligible difference between groups.

Table 1.

Demographic characteristics of the sample

Measure	n (%)
Gender	
Male	28 (23.3)
Female	92 (76.7)
Race/ethnicity	
White, non-Hispanic	100 (83.3)
White, Hispanic	4 (3.33)
Black	2 (1.7)
Asian/Pacific Islander	7 (5.8)
Other	7 (5.8)
Marital Status	
Single, never married	73 (60.8)
Married	23 (19.2)
Other	24 (20.0)
Employment	
Full-time	46 (38.3)
Part-time	27 (22.5)
Student	14 (11.7)
Unemployed	22 (18.3)
Other	11 (9.2)
Education	
High school graduate	17 (14.2)
Technical school/some college	32 (26.7)
College graduate	38 (31.7)
Graduate or professional school	33 (27.5)
	M ± SD (Range)
Age	34.0 ± 13.1 (18–77)

Table 2.

Group differences in changes in BDD-YBOCS item severity over time

	ThTim	ThInt	ThDis	ThRes	ThCon	BeTim	BeDis	BeRes	BeCon	Avoid	Insig
Week 0	0.01	-0.07	0.02	0.30	0.06	-0.21	-0.09	0.19	-0.05	-0.10	0.40
Week 4	-0.04	-0.09	0.03	-0.20	-0.05	-0.02	-0.10	-0.01	-0.24	-0.09	0.41
Week 8	-0.10	-0.27	0.05	-0.09	-0.07	-0.21	-0.17	-0.15	-0.26	-0.22	0.23
Week 12	-0.44	-0.36	-0.38	-0.22	-0.52	-0.22	-0.45	-0.33	-0.51	-0.35	0.12
Week 16	-0.65	-0.36	-0.53	-0.23	-0.48	-0.48	-0.62	-0.48	-0.70	-0.36	0.09
Week 20	-0.37	-0.38	-0.20	0.07	-0.29	-0.64	-0.58	-0.38	-0.66	-0.04	-0.26
Week 24	-0.72	-0.61	-0.50	-0.28	-0.76	-0.82	-0.40	-0.44	-0.94	-0.37	-0.13
Week 37	-0.77	-0.35	-0.34	-0.38	-0.65	-0.61	-0.41	-0.54	-0.83	-0.22	-0.60
Week 50	-0.59	-0.63	-0.33	-0.24	-0.35	-0.73	-0.92	-0.63	-0.96	-0.25	-0.12

Note. Standardized change scores were computed at each week (e.g., week 0 to week 4 change in time occupied by BDD-related thoughts in the CBT and SPT groups respectively). Differences in changes between the two groups were then calculated. **Values** indicate the magnitude of *difference* between the groups, rather than the magnitude of change for any individual group. A value of 0 indicates that the CBT and SPT group exhibited the same degree of change. **Positive values** indicate that the SPT group reported larger declines in symptom severity from baseline than the CBT group. **Negative values** indicate that the CBT group reported larger declines in symptom severity from baseline than the SPT group. ThInt=interference due to thoughts. ThDis=distress due to thoughts. ThTim=time occupied by thoughts. ThCon=degree of control over thoughts. ThRes=resistance of thoughts. BeDis=distress if repetitive behaviors are/were prevented. BeTim=time spent in repetitive behaviors related to body defects. BeRes=resistance of repetitive behaviors. BeCon=degree of control over repetitive behavior. Insig=insight. Avoid=avoidance behavior/interference. Item 7 (interference in functioning due to repetitive behaviors) is not included given its redundancy with avoidance behavior.