



Psychological treatment of social anxiety disorder improves body dysmorphic concerns



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ABSTRACT

Social anxiety disorder and body dysmorphic disorder are considered nosologically distinct disorders. In contrast, some cognitive models suggest that social anxiety disorder and body dysmorphic disorder share similar cognitive maintenance factors. The aim of this study was to examine the effects of psychological treatments for social anxiety disorder on body dysmorphic disorder concerns. In Study 1, we found that 12 weekly group sessions of cognitive-behavioral therapy led to significant decreases in body dysmorphic symptom severity. In Study 2, we found that an attention retraining intervention for social anxiety disorder was associated with a reduction in body dysmorphic concerns, compared to a placebo control condition. These findings support the notion that psychological treatments for individuals with primary social anxiety disorder improve co-occurring body dysmorphic disorder symptoms.

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1. Introduction

A growing body of research suggests that BDD shares some similarities with social anxiety disorder (SAD) in diagnostic features, demographic characteristics, course and onset, clinical characteristics, and treatment outcome (Fang & Hofmann, 2010; Fang et al., 2011; Kelly, Walters, & Phillips, 2010). Prevalence studies show that among individuals with SAD, 4.8–12% also meet criteria for BDD, and among individuals with BDD, 12–68.8% also meet criteria for SAD (Fang & Hofmann, 2010).

Historically, most of the research on body dysmorphic disorder (BDD) has emphasized its relationship to obsessive-compulsive disorder (OCD). Cognitive models of both OCD and BDD propose that maladaptive cognitions maintain and exacerbate these disorders (Rachman, 1997; Wilhelm & Neziroglu, 2002; Wilhelm & Steketee, 2006). A further discussion of the relationship between BDD and OCD, and the inclusion of BDD on the putative obsessive-compulsive spectrum is discussed elsewhere (for a review, see Phillips et al., 2010). The current paper will focus on the relationship between BDD and SAD.

Several models of BDD have strong theoretical overlap with models of SAD. For example, cognitive-behavioral models of BDD emphasize dysfunctional cognitive processes (e.g., negative appraisals of body image, self-focused attention, post-event

rumination) and maladaptive behaviors that maintain BDD (e.g., mirror checking, social avoidance, comparing appearance with others) (Veale, 2004; Wilhelm, Phillips, & Steketee, 2013), which are consistent with processes that are proposed to maintain SAD (Hofmann, 2007; Rapee & Heimberg, 1997). In particular, cognitive-behavioral models of BDD highlight the cognitive aspects of the disorder such as the view of oneself as an esthetic object, which contributes to distorted mental imagery from an observer perspective, self-focused attention, meta-cognitions about the importance of self-focused attention, and a loss of a self-serving bias (Neziroglu, Khemlani-Patel, & Veale, 2008). This literature shares strong similarities with cognitive behavioral models of SAD, which emphasize the view of the self as a social object and leads to hypervigilance of social threat cues (Clark & Wells, 1995; Hofmann, 2007; Rapee & Heimberg, 1997). For both BDD and SAD, it may be that the mental representation of the self is generated from both internal cues (e.g., physical symptoms) and external environmental cues (e.g., facial expressions).

In a study of BDD among individuals with anxiety disorders, Wilhelm, Otto, Zucker, and Pollack (1997) found that BDD was most common among individuals with SAD (12%) and less common among individuals with OCD (7.7%), generalized anxiety disorder (6.7%), and panic disorder (1.5%). Moreover, among all individuals with comorbid SAD and BDD in that study, the onset of SAD preceded that of BDD. This suggests that the presence of SAD may be related to the development of subsequent BDD concerns. Taken together, these findings suggest that BDD symptoms may be elevated among individuals with SAD and that SAD may be a risk factor for the development of BDD symptoms and full-blown BDD.

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The treatment outcome literature further suggests that cognitive-behavioral therapy (CBT) is an efficacious psychological treatment for both BDD (e.g., [Veale et al., 1996](#); [Wilhelm, Otto, Lohr, & Deckersbach, 1999](#)) and SAD (e.g., [Hofmann & Otto, 2008](#)). One study, which examined the effect of CBT for BDD and included SAD symptom outcome measures, found that compared to the wait list control group, individuals who received CBT had significantly less SAD symptoms at post-treatment ([Veale et al., 1996](#)). To our knowledge, no study has yet investigated the effect of CBT for SAD on BDD symptoms.

Furthermore, attentional mechanisms have been the subject of much research attention for both disorders. One study suggested that individuals with BDD selectively attended to appearance-related information and emotional appearance-unrelated information ([Buhlmann, McNally, Wilhelm, & Florin, 2002](#)). Similarly, studies suggest that individuals with SAD have an attentional bias, as demonstrated by faster detection of probes replacing social threat words than of those replacing neutral or positive words in a modified dot-probe paradigm ([Amir, Elias, Klumpp, & Przeworski, 2003](#)). As such, an emerging line of research has begun to evaluate the potential therapeutic benefit of modifying attentional biases in SAD using attention bias modification interventions ([Amir, Weber, Beard, Bomyea, & Taylor, 2008](#)). Studies suggest that attention bias modification interventions, or attention retraining, leads to significantly reduced attentional biases in individuals with SAD, and improves social anxiety symptom severity ([Amir et al., 2009, 2008](#); [Schmidt, Richey, Buckner, & Timpano, 2009](#)). Recent meta-analyses, however, suggest that there may be mixed evidence for the efficacy of cognitive bias modification, and that the effect size of attention retraining for anxiety disorders may be smaller than what other studies suggest ([Beard, Sawyer, & Hofmann, 2012](#); [Hallion & Ruscio, 2011](#)). Nevertheless, attention retraining is relevant to explore in BDD given the hypotheses set forth by cognitive-behavioral models of BDD that individuals with BDD may be particularly attentive to threatening cues with a socio-evaluative component (e.g., threatening faces), and in light of existing evidence showing an attentional bias to appearance-related information.

The purpose of the current paper was to examine the effect of CBT and attention retraining for SAD on BDD-related cognitions and symptoms in individuals with a primary diagnosis of SAD. In Studies 1 and 2, we examined the effect of group CBT for SAD (Study 1) and an attention retraining intervention for SAD (Study 2) on BDD symptoms. We hypothesized that treatment in both studies would lead to a significant reduction of overall BDD symptoms in patients with primary SAD and co-occurring subclinical symptoms of BDD. In both studies, BDD symptoms were measured using the Body Dysmorphic Disorder Symptom Scale (BDD-SS; [Wilhelm, 2006](#); [Wilhelm et al., 2013](#)), which provides an overall severity score, as well as scores in seven different symptom domains. In particular, we hypothesized that the symptom domain reflecting BDD-related cognitions, or the “beliefs about appearance” subscale, would be significantly reduced following treatment in both studies.

2. Study 1

2.1. Participants

The initial sample consisted of 85 adult patients (18 years of age or older) who were participating in a multi-site clinical trial examining the efficacy of d-cycloserine (DCS) augmentation of cognitive-behavioral therapy for SAD. In this trial, participants were randomly assigned to receive cognitive-behavioral therapy augmented with either DCS or pill placebo. The main results of this trial are reported elsewhere ([Hofmann et al., 2013](#)). There were no differences between DCS and Placebo in any demographic

variables, except that more males received DCS-augmented CBT than pill placebo-controlled CBT ([Hofmann et al., 2013](#)). Furthermore, the groups did not differ in BDD symptoms at pre-treatment, $t(25) = -.89, p = .38$. There was also no effect of DCS on BDD symptoms between pre- and post-treatment (Wilks' lambda = .97, $F(1, 25) = .661, p = .42$). A greater proportion of participants in the current study were randomized to receive DCS (63%) than Placebo (37%), but the groups did not differ by gender ($\chi^2(1, 27) = 1.56, p = .21$).

Participants were treatment-seeking individuals presenting to an outpatient clinic specializing in anxiety disorders, or were recruited through online advertisements and flyers distributed in the community. Diagnostic status for participants was determined via administration of the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV-L) or the Mini-Anxiety Disorders Interview Schedule for DSM-IV (Mini-ADIS; [Brown, DiNardo, & Barlow, 1994](#)) at the time of their baseline visit for the study. Diagnostic interviews were conducted by masters-level clinicians. Reliability and integrity of the diagnostic interviews were observed by providing clinicians with weekly supervision and feedback about approximately 20% of audiotaped interviews. All participants met diagnostic criteria for SAD, generalized subtype, as the principal diagnosis, which was defined as the disorder that was most distressing or interfering to the patient. Only one participant met full criteria for a comorbid diagnosis of BDD. The rest of the sample did not meet full diagnostic status for BDD and indicated only subclinical symptoms of BDD. Participants also met a severity cut-off of 60 or greater on the Liebowitz Social Anxiety Scale (LSAS; [Liebowitz, 1987](#)) and had no clinically significant abnormalities based on a physical examination, electrocardiogram, and laboratory findings.

Exclusion criteria included: lifetime history of bipolar disorder, schizophrenia, psychosis, delusional disorders or obsessive-compulsive disorder; eating disorder or posttraumatic stress disorder in the past six months; organic brain syndrome, mental retardation or other cognitive dysfunction that could interfere with the ability to engage in therapy; history of substance abuse or dependence (except for nicotine and caffeine); significant suicidal ideation; concurrent psychotropic medication (e.g., antidepressants, anxiolytics, beta blockers) for at least two weeks prior to study baseline visit or concurrent use of isoniazid; significant personality dysfunction likely to interfere with study participation; serious medical illness or instability for which hospitalization was likely the following year; history of seizures; pregnancy; concurrent psychotherapy initiated within three months of baseline, or ongoing psychotherapy directed toward treatment of SAD; previous non-response to exposure therapy; and, history of head trauma. The final sample consisted of 27 patients. The remaining subjects were excluded from analyses due to insufficient data on BDD symptoms before and after CBT.

2.2. Measures

Participants completed pre- and post-treatment questionnaires assessing social anxiety symptoms using the LSAS and body dysmorphic disorder symptoms using the Body Dysmorphic Disorder Symptom Scale (BDD-SS; [Wilhelm, 2006](#); [Wilhelm et al., 2013](#)).

The LSAS is a clinician-administered 24-item scale that assesses fear and avoidance of social interaction and performance situations in the past week. The measure yields two subscales (fear and avoidance) as well as a total symptom severity score. The LSAS has demonstrated good psychometric properties with regard to internal consistency (Cronbach's alphas ranging from .82 to .92) and convergent validity with other social anxiety measures such as the Social Interaction Anxiety Scale and Social Phobia Scale ([Clark et al., 1997](#); [Heimberg et al., 1999](#)). Example items include “speaking up at a meeting,” “participating in small groups,” and

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