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# Treating Body Dysmorphic Disorder with Medication: Evidence, Misconceptions, and a Suggested Approach

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## Abstract

Body dysmorphic disorder (BDD) is a relatively common and often disabling disorder with high morbidity and mortality. Both psychotropic medication and cognitive behavioral therapy (CBT) are considered first-line treatments for BDD, and medication treatment is often essential for more severely ill and suicidal patients. In this practical overview of the pharmacotherapy of BDD, we briefly describe BDD's clinical features, associated morbidity, and how to recognize and diagnose BDD. We describe the importance of forming a therapeutic alliance with the patient, the need for psychoeducation, and other essential groundwork for successful treatment of BDD. We review available pharmacotherapy research, with a focus on serotonin-reuptake inhibitors (SSRIs, or SRIs), which are currently considered the medication of choice for BDD. Many patients have substantial improvement in core BDD symptoms, psychosocial functioning, quality of life, suicidality, and other aspects of BDD when treated with appropriate pharmacotherapy that targets BDD symptoms. We also discuss practical issues such as dosing, length of treatment, and potential side effects associated with the use of SRIs. In addition, we discuss pharmacotherapy approaches that can be tried if SRI treatment alone is not adequately helpful. Finally, some misconceptions about pharmacotherapy, gaps in knowledge about BDD's treatment, and the need for additional research are discussed.

## Why Treat BDD With Medication?

Body dysmorphic disorder (BDD), a distressing or impairing preoccupation with an imagined or slight defect in one's physical appearance, is a relatively common disorder. An estimated 0.7%–1.7% of the general population has BDD (Bienvenu et al., 2000; Faravelli et al., 1997; Otto, Wilhelm, Cohen, & Harlow, 2001; Rief, Buhlmann, Wilhelm, Borkenhagen, & Brahler, 2006). BDD appears far more common than this in inpatient and outpatient settings (Phillips, 2005a). Currently, psychotropic medication and cognitive behavioral therapy (CBT) are considered the first-line treatments for BDD. Medication treatment is appropriate for individuals who meet full DSM-IV criteria for BDD, and, in our view, medication is essential for more severely ill and suicidal patients (Phillips, 2005a). Available data indicate that appropriate pharmacotherapy substantially improves core BDD symptoms, psychosocial

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functioning, quality of life, suicidality, and other aspects of BDD in a majority of patients (Phillips, 2005a).

Several lines of reasoning support the use of medication to treat BDD. First and foremost, available evidence, reviewed in this paper, indicates that certain medications are often efficacious for BDD. In addition, BDD's pathogenesis likely involves genetic/neurobiologic factors (Phillips, 2005a). BDD also has similarities to obsessive compulsive disorder (OCD), social phobia, and major depressive disorder – disorders for which the efficacy of pharmacotherapy is well established (Phillips, 2005a).

In our clinical experience, a major impediment to successful treatment is BDD's under-recognition and underdiagnosis. Indeed, BDD appears to usually go undiagnosed in clinical settings (Grant, Kim, & Crow, 2001; Phillips, Nierenberg, Brendel, & Fava, 1996; Phillips, McElroy, Keck, Pope, & Hudson, 1993; Zimmerman & Mattia, 1998). Therefore, we first present a brief overview of BDD's clinical features, associated morbidity, and how to recognize and diagnose BDD. After briefly discussing essential groundwork for treatment, we review available pharmacotherapy research, with a focus on serotonin-reuptake inhibitors (SSRIs, or SRIs), which are currently considered the medication of choice for BDD. We suggest strategies for successfully treating BDD with medication, and we discuss misconceptions about medication. Finally, we note some of the research needed to advance and improve the pharmacologic treatment of patients with this severe illness.

## Clinical Features of BDD

### Appearance Preoccupations, Poor Insight, and Compulsive Behaviors

**Appearance preoccupations—**People with BDD are preoccupied with the idea that some aspect(s) of their appearance is ugly, unattractive, deformed, defective, or flawed in some way (Hollander, Cohen, & Simeon, 1993; Phillips, 1991; Phillips et al., 1993; Veale et al., 1996). Concerns may focus on any body area, with multiple areas of concern common. The face or head are most frequently disliked, most often the skin (for example, acne, scars, wrinkles, or pale skin), hair (for example, hair thinning or excessive body or facial hair), and nose (for example, size or shape). The preoccupation usually focuses on specific areas but may involve overall appearance, as in muscle dysmorphia (a preoccupation with the idea that one's body is too small or is insufficiently lean or muscular) (Pope, Gruber, Choi, Olivardia, & Phillips, 1997).

BDD preoccupations are time consuming (occurring an average of 3–8 hours a day) and usually difficult to resist or control (Phillips, 2005a). They are distressing and associated with low self-esteem, shame, rejection sensitivity, and high levels of neuroticism, introversion, depressed mood, anxiety, anger-hostility, and perceived stress (Phillips, 2005a). Patients often believe that they are inadequate and unacceptable – e.g., worthless, inadequate, unlovable, and an object of ridicule and rejection (Veale et al., 1996).

**Insight/delusionality and referential thinking—**Most individuals with BDD are largely or completely convinced that their view of their appearance defects is accurate and undistorted. Studies have found that 27%–39% of patients are currently delusional (Phillips, 2004; Phillips, Menard, Pagano, Fay, & Stout, 2006). Most do not recognize that their belief is due to a mental illness or has a psychological/psychiatric cause (Phillips, 2004). Several studies have found that insight is poorer in BDD than in OCD (Eisen, Phillips, Coles, & Rasmussen, 2004; McKay, Neziroglu, & Yaryura-Tobias, 1997; Phillips et al., 2007)

In addition, a majority have ideas or delusions of reference, believing that other people take special notice of the supposed defects--for example, stare at them, laugh at them, or recoil in horror because they look so bad (Phillips, 2004).

**Compulsive and safety behaviors—**Nearly all individuals perform BDD-related compulsive or safety behaviors, which aim to diminish the distress caused by thoughts about the perceived flaws (Phillips, Menard, Fay, & Weisberg, 2005). Common behaviors include compulsively comparing the disliked body areas with the same areas on other people, camouflaging the disliked areas (for example, with a hat, hair, sunglasses, posture, makeup), compulsive checking of mirrors and other reflective surfaces, excessive grooming (for example, makeup application, hair styling, shaving, hair plucking), skin picking, reassurance seeking, and excessive exercise or weightlifting. These behaviors are time consuming (usually occurring for many hours a day) and are difficult to resist or control. More recently identified behaviors include tanning (for example, to darken "pale" skin or cover perceived scars or acne), excessive clothes changing, and compulsive shopping (for example, for beauty products, acne or hair-loss remedies, or clothes), which can cause substantial debt (Phillips, 2005a).

### Comorbidity

Comorbidity is common (Gunstad & Phillips, 2003; Phillips et al., 2005). Major depressive disorder is most frequently comorbid, occurring in about 75% of individuals over their lifetime. Social phobia, OCD, and substance use disorders are also common.

## Psychosocial Functioning and Quality of Life

Studies using standard measures with well-established norms have found that individuals with BDD have markedly poor psychosocial functioning and quality of life. For example, in two studies that used the SF-36, mental health-related quality of life was markedly poorer than for the general U.S. population and even poorer than for patients with clinical depression or a chronic or acute medical condition (e.g., type II diabetes or acute myocardial infarction) (Phillips, 2000; Phillips, Menard, Fay, & Pagano, 2005). Scores on other standard measures similarly reflect very poor functioning and quality of life (Phillips, Menard, Fay, & Pagano, 2005).

Patients usually experience problems in intimate relationships and social functioning because they are embarrassed and ashamed by their supposed ugliness, are anxious around others as a result, and fear being rejected because of how they look. Impairment in academic or occupational functioning is also common (Phillips, 2005a; Phillips et al., 2005). In a broadly ascertained BDD sample (*n*=200), 36% of individuals were not currently working, and 32% were not able to currently be in school or do school work, because of psychopathology (BDD was the primary diagnosis for most) (Phillips et al., 2005).

## Suicidality

Suicidal ideation and suicide attempts appear common in individuals with BDD. Reported lifetime rates of suicidal ideation range from 78%–81% and lifetime rates of suicide attempts from 24%–28% (Phillips & Diaz, 1997; Phillips et al., 2005; Veale et al., 1996). In a study of adolescent inpatients, those with BDD had significantly greater suicide risk on a standard measure than those without BDD (Dyl, Kittler, Phillips, & Hunt, 2006). While very preliminary, the rate of completed suicide appears markedly high, with an annual rate of 0.35% (Phillips & Menard, 2006). This rate (adjusted for age, gender, and geographic region) is approximately 45 times higher than for the U.S. population, and higher than that reported for most other mental disorders.

## How To Diagnose BDD

The morbidity and mortality associated with BDD, as well as its prevalence, underscore the importance of recognizing and diagnosing this disorder. Studies have found, however, that BDD usually goes undiagnosed in clinical practice (Grant et al., 2001; Phillips et al., 1993; Zimmerman & Mattia, 1998). Of particular relevance to this paper, in a study in which 110 BDD subjects had received psychotropic medication, only 41% of subjects had revealed their BDD symptoms to their pharmacotherapist (Phillips, Pagano, & Menard, 2006). Patients may not disclose BDD symptoms to their clinician because they are too embarrassed and ashamed, or fear they will be misunderstood and negatively judged (for example, as being vain). It is important to diagnose BDD when present, because its pharmacologic treatment may differ somewhat from that of disorders that may be more readily diagnosed, such as major depressive disorder, OCD, or social phobia. In addition, in a particular patient, comorbid disorders may improve with a particular medication but BDD may not – or vice versa (Phillips, Dwight, & McElroy, 1998). Thus, it is important to specifically target BDD symptoms when providing pharmacotherapy.

We recommend that, ideally, BDD be screened for in patients seen in mental health, substance abuse, dermatology, and cosmetic surgery settings. BDD can be diagnosed using relatively straightforward questions which follow the DSM-IV diagnostic criteria. A more detailed discussion is provided elsewhere (Phillips, 2005a). In brief, clinicians should ask whether the patient is worried about their appearance in any way or is unhappy with how they look, whether these concerns are preoccupying (e.g., take at least an hour a day), and what effect the appearance preoccupations have on the person's life -- for example, whether they have caused a lot of distress or significantly interfered with their social life, relationships, school work, job, or any other activities. The appearance concerns should not be better accounted for by an eating disorder. However, BDD and eating disorders may co-occur, in which case both disorders should be diagnosed. While not required for the diagnosis, clues to BDD's presence include the compulsive and safety behaviors discussed above, referential thinking, avoidance of activities, and social anxiety.

It is recommended that clinicians *not* ask patients if they are concerned with an "imagined" defect in their appearance, as most patients have poor insight or no insight and do not consider their flaws to be imagined. We also suggest that initial screening/diagnostic questions not use words such as "deformed" or "disfigured," as terms like these may be too extreme for some patients to endorse. It is also preferable to avoid asking an excessively broad question, such as whether the person thinks something is wrong with their body, as this may be misinterpreted to refer only to physical or bodily functioning rather than appearance.

## Essential Groundwork for Treatment

It is critically important to lay some groundwork for treatment before reaching for a prescription pad. It can be difficult to engage BDD patients in treatment. Some welcome a BDD diagnosis, as they feel less isolated and are relieved to hear that they have a bona fide and treatable disorder. Many, however, resist the diagnosis, believing they truly are deformed and that surgery or dermatologic treatment is the solution to their appearance problems. Many do not recognize that their appearance concerns are due to a treatable mental illness (Eisen et al., 2004).

It is important to first try to engage the patient and establish an alliance so they are willing to try treatment. We suggest not trying to talk them out of their appearance beliefs, as this is usually ineffective. It is important to listen to their concerns, without trying to convince them that their views of their appearance are distorted, or, alternatively, without agreeing that they look abnormal. Instead, empathize with the patient's suffering, and focus on the potential for

psychiatric treatment to diminish their distress and preoccupation and to improve their functioning and quality of life. Motivational interviewing techniques may be helpful.

It is important to provide psychoeducation about BDD and its treatment and to recommend reading on BDD that provides accurate information. The clinician needs to provide information about medication, the rationale for its use, and expected benefits (see below). It can be helpful to explain that SRIs are usually well tolerated, are not habit forming, appear to normalize the brain (and do not cause brain damage), and often diminish depressive symptoms and suicidal thinking in people with BDD. Information should be provided about potential side effects and how they can be managed or made more tolerable should they occur, the expected time course of improvement, and other aspects of medication treatment.

## Pharmacologic Approaches

The treatment of BDD with medication is described in more detail elsewhere, including in a guideline from the United Kingdom's National Institute of Clinical Excellence (NICE) on the treatment of OCD and BDD (Phillips, 2005a; National Collaborating Centre for Mental Health). Serotonin-reuptake inhibitors (SRIs, or SSRIs) are currently considered the medication of choice for BDD. While more research is needed, available data consistently indicate that a majority of patients improve with SRI treatment that is appropriately tailored to BDD symptoms.

Currently, no medications are FDA approved for BDD; not enough studies designed to obtain such approval have been done. It is important to emphasize that what follows is a review of the pharmacotherapy literature and suggested treatment approaches based on currently available research evidence, as well as clinical experience where indicated. These suggestions and guidelines are likely to change as future research is done. These suggestions cannot always be followed in a rigid or "cookbook" fashion. Individual patients may meaningfully differ – for example, in terms of comorbidity, symptoms such as agitation, past treatment, tolerance of side effects, access to mental health treatment, and access to certain medications. Thus, the general guidelines below may need to be modified, based on clinical judgment, to treat an individual patient. In addition, first-line approaches, such as use of an SRI, are more strongly supported by empirical evidence than are subsequent strategies that may be needed (for example, to augment a partial SRI response), as research-based data for choices such as the latter are far more limited.

### SRIs Are the Medication of Choice for BDD

SRIs are antidepressant medications that also diminish obsessional thinking and compulsive behaviors. They are the medication of choice for OCD and certain other psychiatric disorders. SRIs are widely used to treat a broad range of disorders such as major depressive disorder, panic disorder, social phobia, post-traumatic stress disorder, hypochondriasis, bulimia, and binge eating disorder. SRIs may also be helpful for symptoms such impulsivity and anxiety, and sometimes also for aggression or pain. SRIs currently available in the United States are citalopram (Celexa), escitalopram (Lexapro), fluoxetine (Prozac), sertraline (Zoloft), fluvoxamine (Luvox), paroxetine (Paxil), and clomipramine (Anafranil).

The SRIs are the best-studied medications for BDD. All SRI studies to date have found that a majority of patients with BDD improve with these medications. A number of early case reports suggested that fluoxetine and clomipramine improved BDD symptoms (Brady, Austin, & Lydiard, 1990; Hollander, Liebowitz, Winchel, Klumker, & Klein, 1989; Phillips, 1991). These reports led to larger clinical series and subsequently to more methodologically rigorous open-label studies, which used standardized outcome measures, specific dosing titration schedules, assessment at specified intervals, and other standard clinical trial methodology. Two controlled

studies have also been done. Table 1 provides a summary of published open-label and controlled SRI studies. All studies but one used the Yale-Brown Obsessive-Compulsive Scale (BDD-YBOCS), a reliable and valid scale (Phillips et al., 1997), as the primary outcome measure. In all SRI studies published to date, BDD symptoms significantly improved with SRI treatment, with 53% to 73% of participants considered SRI responders. These response rates are based on intention-to-treat data analyses, which included all subjects who were randomized to treatment (in controlled trials) or began taking study medication (in open-label trials). In studies that reported the response rate in subjects who completed the study, completer response rates were higher than intention-to-treat response rates.

In a controlled double-blind cross-over trial that compared the SRI clomipramine to the non-SRI antidepressant desipramine, 29 subjects were randomized to 8 weeks of treatment with each medication (Hollander et al., 1999). Clomipramine was superior to desipramine for BDD symptoms and functional disability. Treatment efficacy was independent of the presence or severity of comorbid OCD, depression, or social phobia. This study suggests that SRIs may be more efficacious for BDD than non-SRI antidepressants (or at least, a non-SRI tricyclic antidepressant). This finding is consistent with retrospective data, described below, which similarly suggest that SRIs may be more efficacious for BDD than other types of antidepressant medications. This finding highlights the importance of specifically treating BDD symptoms – not simply depressive symptoms – in patients with BDD.

In a placebo-controlled study, 67 subjects were randomized to 12 weeks of treatment with fluoxetine versus placebo in a double-blind parallel-group trial (Phillips, Albertini & Rasmussen, 2002). Fluoxetine was significantly more efficacious than placebo for BDD symptoms, with statistically significant separation from placebo beginning at week 8. In the fluoxetine group, BDD-YBOCS scores decreased from 31.5 ± 5.6 at baseline to 21.0 ± 9.8 at endpoint, whereas in the placebo group scores decreased from 30.8 ± 5.8 to 26.9 ± 9.5 ($p<.001$). 53% of subjects responded to fluoxetine compared to 18% to placebo. Treatment efficacy was independent of the presence of major depression or OCD. In addition, response of BDD was not predicted by BDD severity, BDD duration, or the presence of a personality disorder. Improvement in psychosocial functioning was also significantly greater with fluoxetine than with placebo (Phillips & Rasmussen, 2004).

As shown in Table 1, four systematic open-label SRI studies have been done. In a 16-week open-label fluvoxamine study ($n=30$), BDD-YBOCS scores significantly decreased, from 31.1 ± 5.4 at baseline to 16.9 ± 11.8 at termination ($p<.001$) (Phillips et al., 1998). Of the 30 subjects, 63% responded to fluvoxamine. Fluvoxamine response was not related to illness severity. In a 10-week open-label fluvoxamine study of 15 patients, 10 of 15 patients were much or very much improved on the Clinical Global Impressions Scale (CGI) (Perugi et al., 1996). Ten of the 12 patients who completed the study were responders.

In an open-label study of citalopram in 15 patients, BDD-YBOCS scores decreased from 30.7 ± 4.9 at baseline to 15.3 ± 10.6 at termination ($p<0.001$), with 73.3% ($n=11$) of subjects responding (Phillips & Najjar, 2003). Psychosocial functioning and mental health-related quality of life also significantly improved. Considering only those subjects who completed all 12 weeks of the study, 81.8% were responders. Similar results were obtained in an open-label study of escitalopram ($n=15$), with significant improvement in BDD symptoms, functioning, and quality life. In an intention-to-treat analysis, 73% of subjects responded (Phillips, 2006).

SRIs have not been directly compared to one another in a prospective study. However, in a chart-review study of 90 patients treated in the first author's clinical practice, response rates were similar for each type of SRI (these data did not include citalopram or escitalopram, which came on the market later than other SRIs) (Phillips, Albertini, Siniscalchi, Khan, & Robinson,

2001). Overall, 63% (*n*=55) of adequate SRI trials led to clinically significant improvement (much or very much improvement on the Clinical Global Impressions Scale for BDD). Most clinicians would start treatment with an SSRI rather than clomipramine, given the greater likelihood of side effects with clomipramine and its toxicity in overdose.

Earlier studies, which contained largely retrospective data, similarly suggested that SRIs are often efficacious for BDD and that other psychotropic medications may not be (see below). In two cases, intravenous, pulse-loaded clomipramine was reported to be effective for BDD (Pallanti & Koran, 1996).

SRIs also appear efficacious for children and adolescents with BDD, although data are far more limited than for adults. SRI efficacy has been reported in case reports and in a series of 33 children and adolescents with BDD, in which 19 (53%) subjects treated with an SRI had clinically significant improvement in BDD symptoms (Albertini & Phillips, 1999; Albertini, Phillips & Guvermont, 1996; El Khatib & Dickie, 1995; Heimann, 1997; Sondheimer, 1988). Considering individual SRI treatments (rather than patients), 45% of 22 SRI treatments led to significant improvement in the case series (Albertini & Phillips, 1999). Thirteen of the 22 SRI treatments were provided by the authors (rather than other clinicians); of these 13 treatments, 62% led to improvement in BDD (the authors tended to use higher SRI doses than other clinicians did). In this study, no non-SRI medications (haloperidol, perphenazine, imipramine, carbamazepine, clonidine, lithium, methylphenidate, and dextroamphetamine; *n*=8 trials) were effective for BDD (Albertini & Phillips, 1999).

## What Improves with an SRI

Patients who get better with an SRI usually experience improvement on all items of the BDD-YBOCS. This includes less frequent obsessions, decreased urges to perform compulsive/safety behaviors, and better control over BDD obsessions and compulsions. BDD-related distress also usually improves. Some pharmacotherapy studies have found that insight regarding the perceived appearance flaws also improves (Phillips, 2005a). In the studies conducted by Phillips and colleagues, insight was evaluated with the Brown Assessment of Beliefs Scale (Eisen et al., 1998), which assesses components of delusionality/insight such as conviction that beliefs about appearance are accurate and recognition that the beliefs have a psychiatric/psychological explanation. The study conducted by Hollander and colleagues assessed improvement in fixity of beliefs using the BDD modification of the Fixity of Beliefs Questionnaire, a direct modification of the Fixity of Beliefs Questionnaire for OCD (Hollander et al, 1999). Other areas of improvement usually include depression, anxiety, anger/hostility, somatization, suicidal ideation, and psychosocial functioning (Hollander et al., 1999; Phillips, 2006; Phillips et al., 2002; Phillips & Rasmussen, 2004; Phillips, Siniscalchi, & McElroy, 2004). Clinicians can easily track improvement by focusing on the first three BDD-YBOCS items (time preoccupied with appearance preoccupations, associated distress, and impairment in psychosocial functioning), which map onto DSM-IV's criteria for BDD. Other relevant symptoms, such as those noted above, and those relevant to a particular patient (e.g., related to comorbidity), should also be followed.

## SRI Dosing

No studies have compared different SRI doses for BDD; such studies are greatly needed. In our clinical experience, BDD often appears to require relatively high SRI doses which are higher than those typically used for depression. This underscores the importance of targeting BDD symptoms in treatment. If the clinician focuses only on depressive symptoms, the SRI dose may be too low to effectively treat BDD.

The following mean doses are those used by the first author in her clinical practice: 29 ± 12 mg/day of escitalopram, 66 ± 36 mg/day of citalopram, 67 ± 24 mg/day of fluoxetine, 308 ± 49 mg/day of fluvoxamine, 55 ± 13 mg/day of paroxetine, 202 ± 46 mg/day of sertraline, and 203 ± 53 mg/day of clomipramine (Phillips, 2005a). Some patients may benefit from SSRI doses that exceed the maximum dose recommended by the pharmaceutical company – for example, 50 mg/day of escitalopram, 80–100 mg/day of citalopram, 400 mg/day of fluvoxamine, or 300 mg/day of sertraline. These higher doses are best suited to patients who have only partially responded to the highest dose recommended by the pharmaceutical company and who are tolerating the medication well. This approach is also more appealing for patients who have not responded to several previous SRIs or SRI augmentation trials, as remaining medication options are becoming more limited for such patients. Clomipramine doses, however, should *not* exceed 250 mg/day. Thus, we suggest using the maximum SRI dose recommended by the pharmaceutical company, if tolerated, unless a lower dose is effective. For some patients it can be helpful to exceed this dose.

How rapidly the dose is raised will depend on several factors. We generally suggest quicker titration for very ill and suicidal patients, but titration will also depend on how well the medication is tolerated, the frequency of office visits/patient monitoring, and patient preference. Generally, we suggest attempting to reach the maximum SRI dose recommended by the pharmaceutical company by week 5 to 9 of treatment, if tolerated. Titrating the dose fairly rapidly, as described here, may make it more difficult to determine whether a lower dose might be effective for a particular patient (because they may not stay on a lower dose long enough to determine whether response might occur at that dose). However, a slower titration has the potential disadvantage of an unnecessarily protracted treatment trial. SRI titration, however, needs to be tailored to each patient depending on the clinical circumstances.

## SRI Trial Duration

Response to an SRI usually develops gradually and may not be evident for 12 or, occasionally, even 14–16 weeks. The above studies of fluvoxamine and fluoxetine reported a mean time to BDD response of 6 to 9 weeks, whereas citalopram and escitalopram studies reported a mean time to response of 4.6 ± 2.6 weeks and 4.7 ± 3.7 weeks, respectively. These studies used a fairly rapid titration schedule, so an even longer time to response might be expected with a slower dose increase.

It is possible that SRI trials longer than 12–16 weeks would result in a higher response rate, but longer treatment durations have not been studied. In the authors' clinical experience, it is unlikely for patients to first evidence a medication response (at least 30% improvement on the BDD-YBOCS) after 12–16 weeks of treatment, unless the dose titration has been slow.

## Continuation and Maintenance SRI Treatment

No published continuation or maintenance studies have been done. To our knowledge, the only available empirical data are from a chart-review study in which 25 patients who responded to an SRI continued SRI treatment for 6 additional months (Jain, Grant, Menard, Cerasoli, & Phillips, 2004). Over the next 6 months, 40% of these patients had further improvement in BDD symptoms, and only 8% relapsed. Relapse of BDD was defined as worsening on the BDD-Psychiatric Symptom Rating scale by at least 2 points plus meeting full DSM-IV criteria for BDD; improvement was defined as at least 1 point improvement on this scale between the beginning and end of the 6-month continuation phase. This finding is consistent with our clinical experience in suggesting that relapse with continued SRI treatment is rare and that many patients who respond to an SRI by 12 to 16 weeks of acute treatment continue to experience further gradual improvement with continued SRI treatment.

Based on clinical experience, we recommend that an effective SRI be continued for several years, if not longer. There appear to be no major risks of continuing an SRI over years. However, the duration of SRI treatment needs to be tailored to each patient and based on clinical judgment. For example, patients who have previously relapsed when discontinuing an SRI are good candidates for even longer-term SRI treatment. In our view, it may be best for severely ill patients – especially those who have made numerous or potentially lethal suicide attempts or have a history of violence due to BDD (e.g., property damage or aggression toward others) – to continue an effective SRI for life.

## Discontinuation of an Effective SRI

Only limited data are available on the important issue of relapse risk after discontinuation of an effective SRI. The only data available, to our knowledge, are from the above-noted chart-review study. In this study, allowing for censoring, life table analysis estimated that 87% ($n$=20) of patients who discontinued an effective SRI relapsed within the next 6 months, compared to 8% ($n$=2) in the group who continued an effective SRI ($p$<.0001) (Jain et al., 2004). The median time to relapse in the SRI discontinuation group was approximately 75 days. While preliminary, these data suggest that caution should be used if an SRI is discontinued, as relapse may be likely. We are aware of suicides that have occurred following discontinuation of an effective SRI.

Patients may wish or need to discontinue effective medication for a variety of reasons, such as a desire to be medication free, side effects, cost, or lack of access to medication. If an effective SRI is discontinued, clinical experience suggests that this should ideally be done when the patient is not highly stressed and seems able to tolerate a relapse, if it were to occur. In addition, it is probably better to slowly taper the SRI (e.g., over many months) rather than abruptly discontinuing it. It should not be assumed that receiving CBT while taking an SRI will reduce the relapse risk if the SRI is discontinued. This important question has not been studied. In our experience, if patients relapse after SRI discontinuation and decide to restart the SRI, they are likely to respond again, although occasional patients do not respond as robustly as in their first trial.

## SRI Side Effects

The side effects associated with the use of SRIs in BDD are similar to those found in other disorders treated with SRIs, such as major depression or OCD, although since the dosages of SRIs used in BDD may be higher than in other disorders, the side effects may be proportionally dose related. SRI side effects are not uncommon, but they are often tolerable and often resolve over time. For this reason, it is very helpful to tell the patient when an SRI is first prescribed what the possible side effects may be; that they are usually tolerable, if they occur at all; and that subtle dose adjustments or taking the medication at a different time of day can potentially ameliorate side effects. Advance discussion of possible side effects, education regarding side effects, and a strong therapeutic alliance are helpful in enhancing treatment adherence, which is essential for successful pharmacotherapy. SRI-associated side effects may include sedation or activation, insomnia, gastrointestinal symptoms such as nausea, delayed orgasm, and symptoms such as vivid dreams or dizziness upon discontinuation. For such reasons, it is always a good idea to slowly titrate up or down when adjusting the dose.

## Suicidality and SRIs

There has been recent media attention regarding the possibility of suicidal thoughts or behaviors associated with the use of SRIs in youth and young adults (up to age 24) in short-term studies of major depressive disorder and other psychiatric disorders. This association has not been found in adults beyond age 24. In the package insert of antidepressant medications, including SRIs, there is a “black box” warning about this possible increased risk. Certain

analyses, which had methodologic limitations, found that the average risk of such events in patients receiving antidepressants was 4% compared to 2% with placebo. It is important to emphasize that no increase in the risk of completed suicide has been associated with SRI use; suicidal thoughts and behaviors are not the same as completed suicide. A more recent report that included additional studies concluded that the benefits of antidepressants in youth appear to be much greater than risks from suicidal ideation/suicide attempts (Bridge et al., 2007). Additional recent studies indicate that antidepressant medications are associated with a decrease, rather than an increase, in suicidal behavior. For example, suicide attempt rates have been shown to be higher before antidepressant treatment than after starting antidepressant treatment, including in adolescents and young adults (Gibbons et al., 2007a; Simon & Savarino, 2007). Suicide attempt rates are lower among depressed adults treated with antidepressants, including SRIs, than among untreated depressed patients (Gibbons et al., 2007a). In addition, higher SRI prescription rates are associated with lower rates of death by suicide in children and adolescents, and as SRI prescriptions for youth have decreased in recent years, completed suicides rates have increased (Gibbons, Hur, Bhaumik, & Mann, 2006; Gibbons et al., 2007b)

Another important consideration is that illnesses such as depression and BDD themselves are associated with high rates of suicidal thinking, attempts, and suicide. Thus, the substantial risks posed by inadequate treatment of these serious illnesses must be weighed against the very small risk of an increase in suicidal thinking that has been reported during the early phases of SRI treatment. As discussed below, BDD studies have found a decrease in suicidal thinking in patients treated with SRIs. Nonetheless, from a clinical standpoint, it is essential to closely monitor patients for an increase in suicidal thoughts, urges, or behaviors on initiating SRI treatment, and when adjusting the dose.

Several BDD SRI studies have examined suicidality as an outcome measure; for safety reasons, these studies were careful to exclude more highly suicidal patients from the study. In the fluoxetine study, 6% of fluoxetine-treated subjects worsened on the HAM-D suicidal ideation item between baseline and endpoint compared to 30% of subjects on placebo ($p$=.001) (no completed suicides occurred) (Phillips et al., 2002). In an escitalopram study, scores on the HAM-D suicidal ideation item significantly decreased (p<.001) (Phillips, 2006).

## Efficacy of SRIs for Delusional BDD

Available data indicates that SRIs are currently the medication of choice for patients with delusional BDD beliefs, as well as those who have some insight regarding their appearance defects (Hollander et al., 1999; Phillips, McElroy, Dwight, Eisen, & Rasmussen, 2001; Phillips, McElroy, Keck, Pope, & Hudson, 1994; Phillips et al., 2002;). Although delusional symptoms in other disorders are typically treated with antipsychotics, every BDD study that has examined this issue has had the same finding: delusional BDD patients are as likely to respond to SRI monotherapy as nondelusional patients. In the placebo-controlled fluoxetine study, fluoxetine was as efficacious for those with delusional BDD as for those with nondelusional BDD, with a response rate of 50% and 55%, respectively (Phillips, Albertini, et al., 2002). In the desipramine/clomipramine study, clomipramine was more efficacious than desipramine regardless of whether patients had insight or held their BDD beliefs with delusional intensity (Hollander et al., 1999). In fact, clomipramine was even more effective for delusional patients than for nondelusional patients. These findings are consistent with earlier findings from case reports (El-Khatib & Dickey, 1995; Hollander et al., 1989; Sondheimer, 1988), clinical series (Albertini & Phillips, 1999; Phillips et al., 1994), and open-label trials (Phillips, McElroy, et al., 2001), which also indicated that delusional patients have a high response rate to SRIs alone (i.e., without an antipsychotic). In contrast, available data suggest that greater delusionality/

overvalued ideation in BDD may be associated with a poorer response to CBT (Neziroglu, Stevens, McKay, & Yaryura-Tobias, 2001).

The only data on the use of antipsychotics as monotherapy for BDD come from retrospective studies (Phillips et al., 1994). These data suggest that antipsychotics alone are only rarely effective for delusional BDD. Thus, we currently recommend treating delusional BDD with an SRI and not with an antipsychotic alone. However, studies of antipsychotic monotherapy for delusional BDD are greatly needed.

### If an SRI Isn't Adequately Effective: Switching to Another SRI or Augmenting the SRI

An SRI should be tried for 12–16 weeks before drawing conclusions about its effectiveness (Phillips, 2005a). Ideally, by this time the highest dose recommended by the pharmaceutical company (if necessary) or tolerated by the patient will have been tried for at least 3 weeks of the 12–16 weeks. If tolerated, higher doses than this can be cautiously tried to obtain or optimize a response (excluding clomipramine). If this approach is not adequately effective, switching to another SRI or augmenting the SRI with another medication is indicated at this time.

Switching from one SRI to another is a good option. In the above-noted chart-review study, 43% of patients who did not adequately respond to an initial adequate SRI trial responded to at least one subsequent adequate SRI trial, and 43.5% of subsequent adequate SRI trials received by these patients were effective (Phillips, Albertini, et al., 2001). A somewhat different question is how likely *responders* to an SRI are to respond to a different SRI. This issue may arise if side effects are problematic or if a particular SRI is no longer affordable or not completely effective. In the chart-review study, among responders to an initial SRI, 92% of subsequent SRI trials also resulted in response (Phillips, Albertini, et al., 2001).

Only one controlled study has examined SRI augmentation in BDD. This small double-blind randomized study ($n$=28) examined the addition of pimozide versus the addition of placebo to ongoing fluoxetine treatment, after subjects had received an adequate fluoxetine trial (Phillips, 2005b). Pimozide was not more efficacious than placebo; the effect size was small, and the response rate to pimozide was 18.2% versus 17.6% with placebo. There was no significant effect of baseline delusionality on endpoint BDD severity (i.e., more delusional patients did not have a better response).

Augmentation with buspirone, a $5HT1_A$ partial agonist, is appealing because this medication is usually so well tolerated. In a small open study, 13 patients with BDD who had not responded or had only partially responded to an adequate SRI trial had buspirone added to the SRI (Phillips, 1996a). Six subjects (46%) improved. In the above-noted chart-review study, 33.3% ($n$=12) of SRI augmentation trials with buspirone led to significant improvement, with a large effect size (Phillips, Albertini, et al., 2001). The mean buspirone dose was 56.5 ± 15.2 mg/day.

Adding clomipramine to an SSRI, or vice versa, is another option. In the chart-review study, this approach resulted in a response in 44.4% ($n$=4) of cases, with a small to medium effect size (Phillips, Albertini, et al., 2001). However, relatively few patients were treated with this combination, limiting the conclusions that can be drawn. While this approach is generally well tolerated, it should be used with caution, because SSRIs have the potential to unpredictably and sometimes dramatically increase clomipramine blood levels, which has a low therapeutic index. Thus, clomipramine levels should be monitored, starting at a clomipramine dose of only 25 – 50 mg/day (when adding clomipramine to an SSRI). We suggest that clomipramine generally not be combined with an SSRI until an attempt has been made to first optimize a trial with only one of them (Phillips, 2005a).

Retrospective data and the above-noted pimozide study suggest that augmentation with typical (first generation) antipsychotics may not be particularly helpful for BDD. In the chart-review study, only 15% (*n*=2) of antipsychotic SRI augmentation trials led to a response, although the effect size for atypical antipsychotics was large. However, data are still very limited, and additional studies of SRI augmentation with antipsychotics are needed. In our view, adding an antipsychotic to an SRI may be worth trying, especially for patients who are delusional, have prominent delusions of reference, are very agitated, or appear at risk for suicidal or violent behavior. In our clinical experience, an atypical (or second-generation) antipsychotic may be more helpful than a typical antipsychotic, and ziprasodone seems particularly promising. In our view, patients should receive an antipsychotic for BDD only in combination with an SRI.

Clinical experience suggests that occasional patients respond well to SRI augmentation with lithium or methylphenidate (Phillips, Albertini, et al., 2001). These approaches may also improve depressive symptoms, suicidality, or anergia. Venlafaxine augmentation of SSRIs is also promising.

All of the above augmentation approaches need to be studied. No methodologically rigorous studies have compared one augmentation agent to another. In addition, it is unclear what an optimal duration is for an augmentation trial, although in our clinical experience 6–8 weeks is probably adequate to determine whether the augmentation approach will be effective (it is probably best to use a 12-week trial for clomipramine or venlafaxine augmentation, however).

It is not clear whether augmentation is better than switching to another SRI, or vice versa. In the above chart-review study, patients who had not adequately responded to an SRI and who received subsequent SRI augmentation had a response in 33% (*n*=8) of trials, whereas switching to a different SRI led to response in 44% (*n*=10) of trials (Phillips, Albertini, et al., 2001). Response to SRI augmentation was better when the augmenting agent was added to a partially effective SRI, as opposed to an ineffective SRI (response rates of 41% versus 18%) (Phillips, Albertini, et al., 2001). From a clinical perspective, augmentation may be a better choice than switching when response to an SRI, even though limited, is clinically meaningful – for example, when it allows a housebound patient to leave the house or diminishes severe depressive symptoms or suicidality. Another consideration is that if a patient has failed numerous SRI trials (for example, three trials) without any attempt at augmentation, it would seem that augmentation should be tried. On the other hand, if a patient has failed several augmentation strategies with one SRI, switching to another SRI might be considered. However, whether to switch or augment is a complex decision that requires clinical judgment and understanding of the particular patient.

Adjunctive benzodiazepines should be considered for very anxious or agitated patients (Phillips, 2005a). The potential for substance abuse or dependence must be considered, although in our clinical experience, relatively few BDD patients abuse these medications.

### Monotherapy with Non-SRI Medications

Non-SRI medications as single agents have not been well studied (Phillips, 2005a) A small open-label trial (*n*=11) found that venlafaxine significantly improved BDD symptoms in study completers (Allen et al., 2003). This approach has face validity, given venlafaxine's serotonergic properties. However, until more methodologically rigorous studies in larger samples are done, we would recommend an SRI rather than an SNRI such as venlafaxine as a first-line medication for BDD.

The clomipramine-desipramine study, described above, is the most methodologically rigorous study to examine the efficacy of a non-SRI medication for BDD. That study found that desipramine was inferior to clomipramine for both BDD and depressive symptoms (Hollander

et al., 1999). This finding is consistent with a retrospective study of 50 patients, which found that 35 SRI trials resulted in improvement in BDD symptoms, whereas 18 non-SRI tricyclic antidepressant trials led to no overall improvement in BDD symptoms (Hollander et al., 1994).

In an early case series of 30 patients, 58% responded to an SRI, whereas only 5% responded to a variety of other medications (Phillips et al., 1993). In an expansion of this series, which contained 130 patients who had received a total of 316 medication trials, 42% of 65 SRI trials led to clinical significant improvement, compared to 15% of 48 trials with non-SRI tricyclic antidepressants (Phillips, 1996b). The SRI response rate was lower in this study than in subsequent studies, probably reflecting the fact that clinical experience obtained after publication of this early study suggested that higher SRI doses and longer trials were often necessary to effectively treat BDD. Of interest, in this series 30% of 23 trials with MAO inhibitors were efficacious. Combined with some positive case reports (Phillips, 1991), this finding suggests that MAOIs might be tried with more treatment-refractory patients. Consistent with earlier case reports (Phillips, 1991), neuroleptics were effective in only 1 of 49 trials, even though about half of the patients in this series had had delusional BDD symptoms at some point during their illness. Although earlier case series suggested that the antipsychotic pimozide is effective for monosymptomatic hypochondriacal psychosis (Munro & Chmara, 1982), which includes delusional BDD, many patients in these series had parasitosis or olfactory reference syndrome rather than delusional BDD. The authors have found pimozide alone to be ineffective, although we have treated few patients with this medication as monotherapy ($n$ = 8) (Phillips, 1996b).

## CBT and Pharmacotherapy

No studies have directly compared the efficacy of CBT versus pharmacotherapy for BDD. A recent meta-analysis concluded that CBT may be more effective than medication for BDD (Williams, Hadjistavropoulos, & Sharpe, 2006). However, this conclusion is limited by the fact that two BDD pharmacotherapy studies were well controlled (i.e., all subjects received "treatment" and did not know which treatment they were receiving); response rates in well-controlled studies such as these are often lower than in uncontrolled studies. In contrast, in the two controlled CBT studies, all patients knew that they received CBT (this is a psychotherapy study design issue for which there are no easy solutions). In addition, the CBT studies had less methodologically rigorous control groups than the controlled medication studies (e.g., the CBT studies did not control for therapist time and attention). The question of whether medication or CBT is more efficacious needs to be prospectively studied in a head-to-head comparison. In our view, perhaps the more clinically relevant question is which treatment is better for whom? Future studies are needed to answer this important question.

In the meantime, our field is fortunate to have both pharmacotherapy and psychotherapy treatments that appear efficacious for a majority of patients. The choice of which treatment to use will depend on many factors, such as patient preference and motivation, the availability of CBT-trained therapists, insurance coverage, and other factors. In our experience, these treatments are complementary and can be mutually enhancing. One consideration is that response to an SRI can make it more feasible for severely distressed, delusional, or suicidal patients to engage in and tolerate CBT. In our view, medication is always indicated for severely ill, severely depressed, or highly suicidal patients.

## Other Somatic Treatments

Very little data are available on ECT. Case reports and series suggest that ECT is only rarely effective for BDD symptoms, with response of BDD symptoms noted in only two of approximately 25 cases (Carroll, 1994; Phillips, 2005a). Occasional patients, however, had a

transient improvement, primarily in depressive symptoms. Careful clinical judgment is needed when considering use of ECT, however. ECT may be warranted, for example, when severe depressive symptoms do not appear to be entirely or largely due to BDD, or when depressive symptoms appear life-threatening.

Case reports have noted substantial improvement in BDD symptoms with a modified leucotomy (Hay, 1970), capsulotomy (P. Mindus, personal communication), bilateral anterior cingulotomy and subcaudate tractotomy (E. Cassem, personal communication), and anterior capsulotomy (S. A. Rasmussen, personal communication). In our view, neurosurgery should not be considered unless a patient has not responded to many adequate medication trials and to CBT. It is an option to consider, however, for patients who are severely ill or appear at high risk of suicide.

## Corrected Misconceptions About Medication Treatment of BDD

Myths and misconceptions about medications, including SRIs, are ubiquitous, especially with the availability of the internet. What follows are responses to some common misconceptions.

### SRIs appear to be good for the brain

Some people worry that psychiatric medications such as SRIs will harm their brain. There is no evidence that this is true. SRIs appear to correct a “chemical imbalance” in the brain and promote the healthy functioning of serotonin, a natural brain chemical. In addition, antidepressants such as SRIs appear to prevent brain cells from damage or death and can stimulate neurogenesis (the growth of new brain cells) (Jacobs, van Praag, & Gage, 2000). Furthermore, studies have found that patients treated with SRIs have a lower rate of myocardial infarction (heart attack) and cardiovascular mortality than non-SRI treated patients, perhaps due to SRIs’ positive effects on platelet aggregation (Taylor et al., 2005; Roose & Miyazaki, 2005). SRIs have also been shown to improve survival in depressed stroke patients.

### SRIs don’t cause suicide

The most concerning misconception we hear about SRIs is that these medications “make people kill themselves.” As discussed above, there is no evidence that this is the case. In fact, recent studies indicate that antidepressant medications are associated with a decreased risk, rather than an increased risk, of suicidal behavior. However, all BDD patients –whether or not they are taking an SRI -- need to be carefully monitored for suicidality.

### People act normally on SRIs

Some people worry that taking a psychiatric medication will make them behave abnormally or appear “zombie-like.” While occasional patients experience fatigue or agitation while taking an SRI, they do not make people appear “drugged” or ruin their personality. Research on disorders with similarities to BDD, such as OCD, indicate that when patients improve with an SRI, abnormal brain activity (as evidenced by functional neuroimaging, for example) becomes normalized (Saxena et al., 1999). Patients who get better on an SRI usually say they feel more normal, or that they feel more like themselves again.

### Medications can help patients make their best effort to get better

Some patients do not want to take medication because they want to get better on their own. Being motivated to get better is very important, but it is not realistic to try to recover from BDD, especially more severe BDD, solely on one’s own. We often tell our patients that they and the medication work “hand-in-hand” and are on the same team. Medication can make it easier for patients to make positive changes, and it is important for patients to take advantage of improvements that occur with medication treatment.

### Side effects, if they occur, are generally tolerable and often transient

SRIs are generally well tolerated. Many patients have no side effects; when they occur, they are usually tolerable and may resolve with time. More problematic side effects can sometimes be minimized or avoided by changing the dose or the timing of doses. It is important for patients and treating clinician to work collaboratively to try to minimize side effects if they occur.

### SRIs are not addicting or habit forming

Some patients experience transient symptoms (such as dizziness) when an SRI is discontinued (especially if it is discontinued abruptly), but these symptoms do not reflect an addiction to the medication.

### Adequate dosing and duration of medication treatment is needed

Some patients say that medication doesn't work for them, and they are reluctant to give it another try. In our experience, such patients often haven't received a recommended medication at the dose and for the duration recommended for BDD (see above). In such cases, another try, following recommended guidelines, is often helpful.

### Medication treatment must be tailored to each patient

The information in this article provides general suggested guidelines that may need to be modified for an individual patient based on mediation tolerability, comorbid disorders, co-occurring symptoms, past treatment response, and other factors. Weighing such factors and making prescribing decisions may require considerable clinical judgment and expertise.

## Conclusions and Needed Research

Knowledge about effective pharmacotherapy for BDD has dramatically increased over the past decade, to the point where a majority of patients with this severe illness can substantially improve. However, additional pharmacotherapy studies are greatly needed, as research on this disorder is still in its early stages. Needed studies include additional placebo-controlled SRI studies, studies that compare SRIs to other medication classes, continuation and maintenance SRI studies, SRI augmentation studies, relapse prevention studies, SRI studies in children and adolescents, studies that investigate non-SRI medications (e.g., antipsychotics), and effectiveness studies with broad inclusion criteria which examine medication response in diverse clinical settings. Studies are also needed that compare SRIs to CBT, investigate augmentation of one of these approaches with the other, examine combined SRI/CBT treatment, and develop other psychotherapy approaches for BDD. Elucidation of BDD's underlying neurobiology, which has received little investigation, may provide fruitful leads for new and innovative pharmacologic approaches.

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**Table 1**

Controlled and Open-Label Pharmacotherapy Studies in Body Dysmorphic Disorder[a]

| Medication | Study Design | N | Trial Duration and Mean Dose (mg/day) | Results[b] | Reference |
|---|---|---|---|---|---|
| Clomipramine(Anafranil) vs desipramine | Randomized, double blind controlled cross-over trial | 40 entered; 29 randomized | - 16 weeks (8 weeks on each medication)<br>- CMI: 138 ± 87<br>- DMI: 147 ± 80 | Clomipramine was significantly more effective than desipramine for BDD symptoms and functional disability; response rate of 65% vs 35% on BDD-YBOCS[c] | Hollander et al., 1999 |
| Fluoxetine (Prozac) vs placebo | Randomized, double blind, placebo-controlled, parallel group trial | 74 entered; 67 randomized | - 12 weeks<br>- 77.7 ± 8.0<br>range, 40–80) | Fluoxetine was significantly more effective than placebo; response rate of 53% vs 18% on BDD-YBOCS[d]; effect size: d=. 70 | Phillips et al., 2002 |
| Fluvoxamine (Luvox) | Open-label trial | 30 | - 16 weeks<br>- 238.3 ± 85.8<br>(range, 50–300) | 63% of subjects responded to fluvoxamine on BDD-YBOCS[d] | Phillips et al., 1998 |
| Fluvoxamine (Luvox) | Open-label trial | 15 | - 10 weeks<br>- 208.3 ± 63.4<br>(range, 100–300) | 10 subjects responded to fluvoxamine on the CGI | Perugi et al., 1996 |
| Citalopram (Celexa) | Open-label trial | 15 | - 12 weeks<br>- 51.3 ± 16.9<br>(range, 10–60) | 73% of subjects responded to citalopram on BDD-YBOCS[d]; quality of life and functioning also significantly improved | Phillips and Najjar, 2003 |
| Escitalopram (Lexapro) | Open-label trial | 15 | - 12 weeks<br>- 28.0 ± 6.5<br>(range, 10–30) | 73% of subjects responded to escitalopram on BDD-YBOCS[d]; quality of life and functioning also significantly improved | Phillips, 2006 |

[a] Case reports, case series, and retrospective studies are not included in the table but are described in the text

[b] Results are reported for an intent-to-treat analysis for all studies except for the clomipramine/desipramine trial, which used a minimum treatment analysis.

[c] Response was defined as 25% or greater decrease in total BDD-YBOCS score; the BDD-YBOCS (Phillips et al., 1997) assessed BDD severity during the past week based on: (1) preoccupation with the perceived defect (time occupied, interference with functioning due to the preoccupation, distress, resistance, and control), (2) associated compulsive behaviors such as mirror checking (time spent, interference with functioning, distress if the behaviors are prevented, resistance, and control), (3) delusionality/insight, and (4) avoidance.

[d] Response was defined as 30% or greater decrease in total BDD-YBOCS score