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# Body dysmorphic disorder: from clinical aspects to treatment

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Body dysmorphic disorder: from clinical aspects to treatment

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Faculdade de Medicina da Universidade do Porto, 13/01/21

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**Body dysmorphic disorder: from clinical aspects to treatment**


## ABSTRACT

Body dysmorphic disorder (BDD), also known as dysmorphophobia, is a relatively common disorder, with a point prevalence of 0.7% to 2.4% among the general population, that consists of a distressing or impairing preoccupation with imagined or slight defects in appearance, associated with repetitive behaviors and where insight regarding the appearance beliefs is often poor. Failure to recognize and diagnose this disorder can lead to poor physical and psychiatric outcomes for patients, thus it is important for the physicians to be aware to the clinical aspects of this disorder, for early detection of the condition and consequently give an appropriate treatment to the patients. The criteria for diagnosing BDD are described according to the DSM-IV and DSM 5.

The search for bibliographic information was held on the Pubmed platform with the keywords: body dysmorphic disorder. Only articles that obeyed the following conditions were analyzed: written in English or Portuguese and with free full access; having analyzed 494 articles, between 1994 and 2020. There were included 41 articles that appeared to be relevant for the Body dysmorphic disorder, and which aimed to clarify the clinical, diagnostic and therapeutic aspects of this clinical entity.

Serotonin reuptake inhibitors (SRIs) are currently considered the medication treatment of choice. According to all studies conducted do date, for an improvement of symptoms, a relatively high SRI dose and at least 12 weeks of treatment is often needed. The psychosocial treatment of choice is cognitive behavioral therapy. Additional treatment development and efficacy studies are urgently needed to better understand the therapeutic approach of this entity, in order to improve the quality of life of these patients.



Keywords: Body dysmorphic disorder

## Introduction

Body dysmorphic disorder is characterized by excessive concern and preoccupation with an imagined or a slight defect in body appearance that is not better accounted by another mental disorder. Patients with BDD typically describe themselves as looking ugly, abnormal, deformed, or disfigured. [1]

The concerns caused by the appearance are intrusive, unwanted, time-consuming and difficult to resist or control.[1] In response to their physical concerns, patients with BDD will follow compulsive behaviors in order to reduce the distress caused by their perceived deformations. [3] These preoccupations are associated with low self-esteem, feelings of shame, depressive symptoms, anxiety, and guilt and patients are often highly sensitive and vulnerable to rejection experiences. [3]

In 1891, Enrico Morselli, an Italian psychiatrist, coined the term dysmorphophobia to describe the condition of people who perceive themselves as flawed but with no apparent physical deformities.[6] This term was derived from the Greek word "dysmorfia," which means "ugliness."[6] Cases of this condition have been discussed by Pierre Janet, a French psychologist, who labeled it as "l'obsession de la honte du corps," which translates to "obsessions of shame of the body."[6] Sigmund Freud also detailed a case known as "Wolf Man," a man with an obsession with his nose, which caused him significant social distress. [6]

This condition was first recognized in the DSM in 1980 as an atypical somatoform disorder.[6] In 1987, it was classified as a distinct somatoform disorder.[6] DSM 5 now classifies BDD under obsessive-compulsive and related disorders, given its similarities with OCD in phenomenology, epidemiology, comorbidity, familial aggregation and response to treatment.[2, 40]

Body dysmorphic disorder (BDD) is a relatively common and severe illness that often presents to both mental health professionals and nonpsychiatric physicians.[19] However, for several reasons BDD usually goes unrecognized and undiagnosed in clinical settings.[19] Many patients are too ashamed of their symptoms to voluntarily disclose them to a clinician, even though most patients want their clinician to know about their appearance concerns.[19] In addition, many patients present to nonpsychiatric physicians (eg, primary care physicians, dermatologists, surgeons), often seek unnecessary dermatologic treatment and cosmetic surgery.[1, 19] However those treatments appear ineffective for BDD and may even be risky for physicians to provide.[19]

That BDD appears to be relatively common underscores the importance of its recognition in clinical settings.[19]

## Epidemiology

Epidemiologic studies have reported a point prevalence of 0.7% to 2.4% in the general population, making it more common than obsessive-compulsive disorder (OCD), anorexia nervosa or schizophrenia.[5,7]

BDD is sometimes considered a "female disorder" because the symptoms involve appearance. However, BDD appears as common or nearly as common in males as in

females.[19] The gender ratio appears to be in the range of 1:1 to 3:2 (female:male), although it has varied in different studies.[19] Larger epidemiologic studies that examine BDD's gender ratio are needed.[19]

A recent systematic review highlighted the prevalence of BDD within different settings. They found the weighted prevalence of BDD in adults in the community was estimated to be 1.9% though this was increased when looking at specific psychiatric settings (adult psychiatric outpatients (5.8%) and adult psychiatric inpatients 7.4%) and even further increased in the context of other nonpsychiatric specialties such as general cosmetic surgery 13.2%; in rhinoplasty surgery 20.1%; in orthognathic surgery 11.2%; in orthodontics/cosmetic dentistry settings 5.2%; in dermatology outpatients 11.3%; and in cosmetic dermatology outpatients 9.2%. [4] This suggests that dysmorphic concerns need to be incorporated into pre-operative and other non-psychiatric contexts. [3]

Studies in outpatient settings have reported rates of 8%-37% in patients with OCD, 11%-13% in social phobia, 26% in trichotillomania, 8% in major depression and 14%-42% in atypical major depression (MDD).[8] In a study of atypical depression, BDD was more than twice as common as OCD, and in another it was more common than many other disorders, including OCD, social phobia, simple phobia, generalized anxiety disorder, bulimia nervosa, and substance abuse or dependence. [8]

However, these numbers may underreport its prevalence.[5] Many individuals with BDD feel ashamed of their appearance and the fact that they are so focused on it and as a consequence, they may not report their BDD symptoms to clinicians.[5] In one study of psychiatric inpatients, only 15.1% had revealed their body image concerns to their mental health clinicians, and the most common reason for not disclosing their concerns was embarrassment (in 31. 3 %). [5]

**Risk Factors**

Emerging findings indicate that, like other psychiatric disorders, the etiology/pathophysiology of BDD is likely to be multifactorial and complex; BDD is currently understood to arise from a combination of biological, psychological, and environmental factors.[3,7.] The development of BDD is associated with past experiences of abuse, violence, and trauma; patients are not only more likely to have a history of traumatic experiences but also to experience them as more painful and to be able to recall them clearly.[3]A survey of patients with BDD found high rates of emotional neglect (68.0%), emotional abuse (56.0%), physical abuse (34.7%), physical neglect (33.3%), , and sexual abuse (28.0%).[41]In fact, patients with BDD have a distorted body image, which may be associated with bullying or abuse during childhood or adolescence. [20]

In addition, these findings indicate that BDD is a brain-based disorder - not vanity.[7]

## Criteria for BDD diagnosis

Detection of BDD is by clinical suspicion and, as such, knowledge of the DSM-IV criteria provides a good reference point for clinicians.[16] The DSM-IV criteria have been adapted to a self-report questionnaire with good sensitivity and specificity for detection of the disorder and clinicians likely to be in frequent contact with BDD could consider this simple and effective identification measure. [16]

The DSM-IV criteria for diagnosis of BDD are (**1**) preoccupation with an imagined or barely perceptible defect in appearance, (**2**) the preoccupation causes marked distress and impairment in social and occupational functioning, and (**3**) the appearance concern is not better accounted for by another mental disorder.[20] Appearance concerns are common in the general population; in BDD, the preoccupation must cause clinically significant distress or impaired social or occupational functioning, which differentiates the disorder from normal appearance concerns. [20]

According to the criteria adopted by the DSM-IV-TR (Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision), BDD is classified as a somatoform disorder, although its delusional variant may also be classified as a psychotic disorder (delusional disorder, somatic type).[27] Phillips et al investigated the 2 BDD variants (delusional and nondelusional) and found that 36% of BDD subjects were delusional (in which patients are completely convinced that they appear ugly or abnormal). [20]

Despite their separate classification, available evidence indicates that BDD's delusional and nondelusional forms have many similarities, suggesting that they may actually be the same disorder, characterized by a spectrum of insight. [18] In fact, it is one of the diagnostic entities that falls on the borderline between neurotic and psychotic spectrum of disorders.[18] However, the delusional variant appears more severe than non delusional BDD; delusional BDD patients had significantly lower educational attainment, were more likely to have attempted suicide, had poorer social functioning on several measures, were more likely to have drug abuse or dependence, were less likely to currently be receiving mental health treatment, and had more severe BDD symptoms.[18] DSM-TV allows BDD and its delusional disorder variant to be doublecoded; in other words, patients with delusional BDD can receive a diagnosis of both delusional disorder and BDD.[5] This double coding reflects evidence that BDD's delusional and nondelusional variants may in fact be variants of the same disorder.[5] Patients with delusional BDD beliefs would receive a DSM-TV-TR diagnosis of delusional disorder, or DSM-TV-TR diagnoses of both delusional disorder and BDD.[5]

The ICD-10 also classifies BDD as a somatoform disorder, ie, as a type of hypochondria. [27]

During the c development process, is has been taken into account the combination of BDD's delusional variant with its nondelusional variant into one disorder (BDD) while specifying degree of insight (with good or fair insight, with poor insight, or with delusional BDD beliefs).[5]

There have already been proposals in order to consider BDD as part of the obsessive-compulsive spectrum disorders in the DSM-5, even though current findings indicate that it is not simply a subtype of OCD. [20]

Body dysmorphic disorder, or BDD, formally known as dysmorphophobia, is a psychiatric condition defined in the DSM 5 as a preoccupation with a perceived defect or flaw in one's physical appearance that is either not noticeable or only slightly observable by others. (DSM-5 criterion A). [6,7]The individual at some point during the illness must engage in repetitive behaviors (such as excessive mirror checking, camouflaging, skin picking, excessive grooming, excessive weight lifting) or pervasive mental acts (such as comparing one's appearance to others) (DSM-5 criterion B).[6, 11]To meet DSM-5 BDD criteria, the appearance preoccupations and resulting repetitive behaviors must cause clinically significant distress or impairment in social, occupational or other important areas of functioning. (criterion c). [11] The appearance preoccupation is not better explained by concerns with body fat or weight in an individual whose symptoms meet diagnostic criteria for an eating disorder. (criterion D). [11]

Once the clinician has diagnosed BDD, he/she should consider the following two specifiers, which are new to DSM-5 and identify important subgroups of individuals with BDD: [7]

(1) Muscle dysmorphia: this specifier identifies individuals (usually men) who are preoccupied with the belief that their body build is too small or insufficiently muscular, even though they look normal or even very muscular because of excessive weight lifting or anabolic steroid use. This specifier is used even if a patient has additional, nonmuscle-focused preoccupations, which is usually the case.

(2) Insight: this specifier indicates the level of insight regarding BDD beliefs. Levels of insight in DSM-5 are 'with good or fair insight', 'with poor insight', and 'with absent insight/delusional beliefs'. This new insight specifier in DSM-5 is important for several reasons: (i) it clarifies that individuals who are completely convinced that their BDD belief is true should be diagnosed with 'BDD with absent insight/delusional beliefs' rather than a psychotic disorder; (ii) it implies that delusional and nondelusional BDD should be treated similarly - indeed, studies indicate that both delusional and nondelusional BDD respond to serotonin reuptake inhibitor (SRI) monotherapy and to cognitive-behavioral therapy (CBT), and (iii) specifying the level of insight allows the identification of patients with poorer insight, who may be more reluctant to accept the idea that they have a mental disorder (BDD) rather than actual physical deformities. Such patients may need more motivational interviewing and attention to the therapeutic alliance in order to engage and retain them in mental health treatment. [7]

**Differential Diagnosis**

Clinicians need to distinguish BDD from other disorders with similar symptoms. [13]

Differential diagnoses include eating disorders, other obsessive-compulsive disorders, illness anxiety disorders, depressive disorders, anxiety disorders, and psychotic disorders. BDD symptoms should not be better explained by any of these conditions, although these can co-occur with BDD.[6]

Social phobia and avoidant personality disorder share symptoms of self-consciousness and anxiety in social situations.[13] However social avoidance in social anxiety disorder is driven by a fear of saying or doing something that embarrass oneself. On the other hand, in BDD social anxiety is exclusively linked to a fear of negative judgements about perceived appearance defects. [40]

Both BDD and depression can involve feelings of ugliness as part of pervasive low self-esteem.[40] However concerns about appearance are not the primary concern in depression and not typically associated with the repetitive behaviors that are characteristic of BDD (eg, mirror checking, grooming). [40]

Sometimes, depressive symptoms, which coexist with BDD, are diagnosed but BDD is missed, or BDD symptoms are considered a symptom of depression.[19] It is also important for a clinician to ask about BDD symptoms in depressed patients and diagnose BDD if present, without assuming that appearance concerns are simply secondary signs of depression. [19]

It is not uncommon for individuals with BDD to be housebound, because they feel too ugly to be seen and are very anxious around other people.[19] Patients with features of agoraphobia should be asked why they are reluctant to go out, and BDD should be diagnosed if present.[19]

Both BDD and OCD are characterized by obsessions and compulsive behaviors.[19] However, these disorders have some important clinical important differences.[19] If the obsessions and compulsions focus on appearance, BDD is the more appropriate diagnosis.[19] BDD patients also have more delusional beliefs, greater suicidal feelings, and a higher prevalence of major depressive disorder than patients with OCD.[23]

Some people with BDD pull out or pluck their body hair resulting in noticeable hair loss. However, hair pulling in trichotillomania is not driven by an attempt to improve appearance, whereas hair pulling in BDD is intended to improve the appearance of perceived defects in facial or body hair. [19,40]

Because appearance beliefs may be delusional, and because ideas or delusions of reference may be prominent, BDD may be misdiagnosed as schizophrenia or psychotic Disorder-Not Otherwise Specified. If the psychotic symptoms are limited to BDD symptoms, BDD is the more appropriate diagnosis. [19]

Both BDD and excoriation disorder are characterized by repetitive skin picking. However, skin picking in excoriation disorder is not driven by an attempt to improve appearance, whereas skin picking in BDD is intended to improve the appearance of perceived defects in the skin. [40]

BDD and eating disorders are both characterized by distressing and impairing preoccupation with appearance.[40] However appearance preoccupation in eating disorders

is focused on body weight and shape, leading to dysfunctional eating behaviors in an attempt to lose weight.[40] Therefore, a patient with anorexia who focus their body image concerns only on the weight, should be diagnosed with anorexia, and not with BDD.[19] Patients with BDD are also concerned with other body parts (typically not weight) than eating disorder patients are, and they have more negative self-evaluation and self-worth, more avoidance of activities, and poorer functioning and quality of life due to appearance concerns.[23]

However, BDD and eating disorders commonly co-occur and this comorbidity can be particularly challenging to diagnose since both illnesses share a distorted body image and low self-esteem.[7,15]

However body image is a concept different from BDD, and those who have body image disturbance are not necessarily suffering from BDD.[20]

Many patients with anorexia have appearance concerns other than weight and body shape.[20] Kollei et al assessed 49 subjects with anorexia nervosa and 51 subjects with bulimia nervosa and found that 12% of individuals with eating disorders had comorbid BDD, with their body dysmorphic concerns being unrelated to weight and shape, focusing instead on skin, hair, teeth, nose, and height.[20]

In a study of 41 cases of anorexia nervosa, Grant and colleagues found that 16 (39%) also fulfilled diagnostic criteria for BDD unrelated to concerns about weight.[15] Comorbid cases appeared to have a more severe form of illness than non-comorbid BDD, with higher levels of delusional thinking and poorer social function.[15]

**Appearance concerns**

Individuals with BDD obsess over certain aspects of their appearance. These minimal or nonexistent appearance flaws are perceived by the patient to be unattractive, devastating, and the cause of great anxiety and distress.[5,12] Any part of the body may be implicated in BDD. However, the most common is the face/head, particularly the skin (73%), hair (56%), and nose (37%).[5]

The skin, which is the most common area of concern, most often perceived acne or scarring.[19] However patients may obsess about any aspect of their skin. Hair concerns which is the second most common, most often focus on hair loss, thinning, balding, or excessive facial or body hair. Nose concerns are also very common, with many patients worrying that their nose is too large or misshapen.[19] These concerns can be focused on any combination of body parts, and focus on multiple perceived defects is common.[3] The types of bodily concerns tend to vary with gender being the male gender, generally, more concern with their height, genitals, body hair and build, while females with their breasts, hips, legs and body weight.[10,33] On average, over their lifetime, people with BDD are preoccupied with 5–7 different body parts.[4] Concerns may also involve the appearance of the entire body for instance the muscle dysmorphia form of BDD which consists on the belief that one's body is too small and/or insufficiently muscular.[4]

Approximately 40% of individuals with BDD actively think about the disliked body parts for 3 to 8 hours per day, and 25% report thinking about them for more than 8 hours

per day.[5] The preoccupations are intrusive, unwanted, and associated with distressing emotions such as shame, disgust, anxiety, and sadness.[4] For this reason, it is ineffective to simply tell patients to stop worrying about how they look. [19]

**Insight regarding perceived appearance defects**

Insight regarding the perceived appearance defects varies.[5]
An important aspect of BDD is that insight is usually poor or absent. Approximately 35% to 40% of patients currently have no insight and are delusional.[19] In other words, they are completely convinced that their appearance beliefs are accurate and undistorted.[19]

Most nondelusional patients have poor insight, believing that the way they realize their appearance is probably accurate. Thus, prior to treatment very few patients have good insight. They do not recognize that their view is distorted and that their appearance concerns are due to a psychiatric disorder. Consistent with this poor or absent insight, many patients seek nonpsychiatric treatment to fix the problem they perceive. [19]
In fact, studies have consistently found that insight is poorer in BDD than in OCD, with 27% to 60% of BDD patients having delusional beliefs versus only 2% of OCD patients.[5]

About two thirds of BDD patients have past or current ideas or delusions of reference, that is, they think that other people take special notice of the supposed defect and perhaps talk about it or mock it.[5,12] Clinical impressions indicate that such referential thinking may lead to feelings of rejection and to anger (even violence, such as attacking someone they believe is mocking them).[5]

Patients with delusional BDD beliefs would receive a DSM-TV-TR diagnosis of delusional disorder, or DSM-TV-TR diagnoses of both delusional disorder and BDD.[5] Studies comparing delusional and nondelusional BDD patients reveal more similarities than differences between the two groups, and that the primary difference is BDD symptom severity.[5] Delusional BDD appears to respond to SRI monotherapy and may not respond to antipsychotic medications, suggesting that delusional BDD is not a typical psychotic disorder.[5] Thus, it may be more accurate to view insight as existing on a continuum and to consider BDD to encompass both delusional and nondelusional appearance beliefs. [5]
Furthermore, some individuals with BDD describe fluctuations in insight, such that they are completely convinced that they are ugly at some times but not convinced at others. [5]

**Compulsions, safety behaviors, and avoidance**

The DSM-IV-TR diagnostic criteria for BDD make no reference to compulsive and safety behaviors that are commonly associated with BDD. During the DSM-5 development process, the addition of these symptoms to BDD's diagnostic criteria are being taken in consideration.[5]

Nearly all people with BDD perform compulsive behaviors which are repetitive, time-consuming (about half of BDD patients spend 3 or more hours per day engaged in them) and hard to control and resist.[5, 9, 19]These behaviors arise in response to the obsessive

thoughts about appearance and focus on examining, improving, being reassured about, or hiding the perceived defect and are meant to reduce anxiety and other painful emotions.[5,9] As in OCD, the behaviors are not pleasurable. [5]

Most BDD patients perform multiple compulsive behaviors. One common behavior is comparing themselves with other people. Clinical impressions suggest that this usually happens quite automatically, and can cause anxiety and inability to concentrate. About 90% of BDD patients check themselves repeatedly and excessively in mirrors or other reflective surfaces. Typically, they do this hopping to see an acceptable look, but often, after seeing their reflection, they feel worse.[5]

Some behaviors, such as camouflaging disliked body parts, are called safety behaviors, because their function is to reduce or avoid painful emotions or prevent something bad from happening, such as being humiliated or embarrassed. [5]

Other common repetitive behaviors are excessive grooming (eg, combing their hair or washing their skin repeatedly), tanning (to improve their skin color or skin imperfections), reassurance seeking (asking whether one's appearance is acceptable), excessive shopping for beauty products (which can lead to financial indebtedness), changing their clothes repeatedly to find a more flattering outfit, excessive exercise (eg, weightlifting in the case of muscle dysmorphia), seeking surgery or medical treatment, and using potentially dangerous anabolic steroids to bulk up. [5, 9]

Many BDD patients (27% to 45%) pick at their skin in an attempt to improve perceived blemishes or imperfections; however, this behavior sometimes causes observable appearance defects and can even cause severe damage such as skin infections and rupture of blood vessels.[5]Skin picking, also described as neurotic excoriation, behavior varies in severity level patterns and often occurs in the presence of other psychiatric disorders. This disorder initially relieves tension and provides its own source of distress, shame, embarrassment, and impairment. It is time-consuming (often lasting 5–60 minutes per episode) and it may cause significant lesions, scarring, or even life-threatening injury by picking through major blood vessels. [19,20.]

Avoidance is a common behavior in BDD and may serve a similar purpose as the compulsive behaviors in the short term - that is, to temporarily relieve BDD-related anxiety and distress. However, clinical experience indicates that compulsions and avoidance seldom improve anxiety or reduce the intensity of BDD-related thoughts; rather these behaviors may contribute to the chronicity and severity of BDD.[5]

**Course of illness**

BDD usually begins during adolescence with two studies reporting a mean age at onset of 16 and a mode of 13 and can even occur in childhood, although treatment seeking for BDD is delayed an average of approximately 11 years after illness onset. [5, 19] Thus, many patients suffer for years without being treated.[19]

The disorder appears to be chronic, although patients who receive appropriate psychiatric treatment appear to have a generally favorable course.[12] RCTs have shown a response rate of 50% to 80% with pharmacological treatment and relapse rates were shown to decrease with long-term pharmacologic treatment.[6]

The only prospective study of BDD's course, it was found that the probability of full remission from BDD over 1 year of follow-up was only 0.09, which is lower than has been reported for mood disorders, most anxiety disorders, and personality disorders in other longitudinal studies.[5]

More severe BDD symptoms at intake, longer duration of BDD, and the presence of one or more comorbid personality disorders at intake predicted a lower likelihood of remission from BDD.[5]

A recent study found a low cumulative probability (20%) of full remission over 4 years of treatment.[3]

In addition, complete remission seems to be rare even after treatment.[20]

**Psychosocial functioning and quality of life**

BDD is associated with substantial impairment in psychosocial functioning and markedly poor life quality. Individuals with BDD have, on average, much poorer mental health, emotional well-being, social functioning, and overall lower life quality of life than the general population.[5]

In the only prospective study of BDD, overall functioning continued to be poor over 1 to 3 years, and poorer functioning was predicted by more severe BDD and greater delusionality of BDD beliefs at intake.[5]

Preoccupations and related behaviors can also impair concentration and consume a great deal of time.[12] Many individuals avoid work, relationships, and social situations because they worry that they look ugly or that others are laughing at them.[12] BDD can cause severe depression and anxiety, and can lead to unemployment.[12]They may have few or no friends, and they often avoid dating and other social interactions.[13] Housebound and psychiatric hospitalization is also relatively common.[12] In two studies, 27% to 31% of individuals with BDD had been completely housebound for at least 1 week due to BDD symptoms, and more than 40% had been psychiatrically hospitalized.[5]

Levels of functioning varies, however. Individuals with milder symptoms may, with effort, function well despite their distress, although usually below their capacity. Those with severe BDD may be completely incapacitated by their symptoms.[12]

Available data indicate that mental health-related quality of life is markedly poorer for these patients than for the general population, and it appears even poorer than for patients with type II diabetes, a recent myocardial infarction, or clinical depression (major depressive disorder and/or dysthymia). Available data also suggest that quality of life and psychosocial functioning in patients with BDD appear as poor as, or poorer than, in those with OCD.[13]

**Risk behaviors: suicide, substance abuse, and violence**

The associated feelings of guilt and shame appear to contribute to high rates of suicidal ideation and suicide attempts in BDD patients. Rates of suicide completion are significantly higher in psychiatric inpatients with BDD relative to those without, and relative to most other mental disorders as well. [3] Individuals with BDD have been found to be 2.6 times more likely to attempt suicide and 4 times more likely to experience suicidal ideation than someone without the condition.[6]

Approximately 80% of individuals with BDD report past or current suicidal ideation, and about one quarter have attempted suicide, which is often attributed to BDD symptoms.[5] The annual rate of completed suicide, while very preliminary because only one study has been done, appears markedly high at 0.3%, and may even be higher than for major depressive disorders, bipolar disorders, or eating disorders.[13, 19]. When controlling for age, gender, and geographic region, the standardized mortality ratio is markedly elevated.[24] These findings underscore the importance of screening patients for BDD and implementing appropriate treatment.[19] Consequently, a risk assessment is always necessary.[1] Suicide risk increases vastly if the patient considers to have come to the end of the line as far as possible treatment options are concerned and it also is higher in patients with delusional beliefs.[1]

Moreover, adolescent-onset BDD is associated with the development of more severe symptoms, greater lifetime comorbidity, and higher rates of attempted suicide, compared with adult-onset BDD.[29]

Approximately one third of people with BDD report violent behavior that they attribute primarily to BDD symptoms.[5] Clinical impressions suggest that anger or violence may be fueled by anger about looking "deformed," inability to fix the "defect," delusions of reference, and feeling rejected by others because of the "defect."[5] In addition, anger or even violent behavior may be caused by dissatisfaction with cosmetic procedures.[5] In fact such treatment appears to only rarely improve overall BDD symptoms which can lead to serious negative consequences for both patients and physicians.[5] According to one survey of cosmetic surgeons, 40% of the respondents indicated that dissatisfied BDD patients had threatened them physically or legally. There are also occasional reports of individuals with probable BDD who attacked and even killed their plastic surgeon after being distraught by the outcome of a cosmetic procedure.[5] In fact, patients report a high degree of dissatisfaction with cosmetic surgery and frequently an increase in symptoms of BDD.[16]

Many individuals with BDD abuse of alcohol or drugs. In one study, 48.9% of BDD participants were diagnosed with a lifetime substance-use disorder, with 42.6% reporting an alcohol-use disorder and 30.1% reporting a cannabis-use disorder. Onset of BDD preceded onset of a substance-use disorder by at least 1 year in 60% of the participants, followed onset of the substance-use disorder in 19% of the participants, and began in the same year in 21%. When asked about the relationship between substance use and BDD symptoms, 68% said that BDD symptoms contributed to the substance use becoming problematic.[5]

**Comorbidity**

Many psychiatric illnesses have been reported to cooccur with BDD, the most common being major depressive disorder, social phobia, obsessive-compulsive disorder, and substance misuse disorders.[4]

In the two largest phenomenology studies of individuals ascertained for BDD, which assessed all participants with the Structured Clinical Interview for DSM, major depressive disorder was the most common comorbid disorder, with a lifetime prevalence of about 75% in both samples. The other most common lifetime comorbid disorders were substance-use disorders (30% to 48.9%), OCD (32% to 33%), and social phobia (37% to 39%).[5] In addition, a majority of patients with BDD also have a personality disorder (most often, avoidant).[8,12]

Over the past century, BDD has been hypothesized to be related to OCD; but, more recently, BDD has also been hypothesized and argued to be a form of affective spectrum disorder. Nearly all studies report a high level of comorbidity with depression and social phobia, which is estimated to occur in more than 70% of BDD patients and could explain the response of the disorder to selective serotonin reuptake inhibitors SSRIs.[20]

In this study, onset of major depression most often occurred after onset of BDD, consistent with clinical impressions that depression is often (although not always) secondary to BDD. [8]

Anxiety disorders frequently co-occur with BDD. Over 60% of BDD patients were reported to have a lifetime history of an anxiety disorder. The lifetime co-occurrence rate for social phobia is roughly 38%, which tends to predate the onset of BDD.[20] In a study of BDD among individuals with anxiety disorders, Wilhelm and colleagues (1997) found that BDD was most common among individuals with SAD (social anxiety disorder) (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. 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. [28]

The psychiatric literature focused on the obsessions and repetitive behaviors of BDD has debated its relationship with OCD. On the basis of the high comorbidity of BDD with OCD, the concept of shared etiology/pathophysiology was proposed to explain this co-occurrence, and it was suggested that BDD may be considered an obsessive-compulsive spectrum disorder because of similarities between both conditions.[20]

A study of Axis I comorbidity in patients with BDD showed that 21.7% of BDD participants had 1, 28.6% had 2, and 41.4% had 3 or more Axis I psychiatric comorbidities. [20]

Gunstad and Phillips found co-occurrence of OCD plus major depression was the second most frequent Axis I comorbid disorder in BDD (25%). [20]

Patients with BDD have high levels of distress, are highly symptomatic, and have poor well-being; they also have high scores for depression, anxiety, and anger-hostility on assessment questionnaires compared with normal controls. However, the delusional BDD variant is associated with more comorbidity than the nondelusional variant. [20]

**Pharmacological treatment**

Pharmacological intervention is often more readily available than CBT, and may present a flexible modality by which comorbidities can also be addressed; such comorbidities include MDD, social anxiety disorder, and OCD. [3] Medication treatment is appropriate for individuals who meet full DSM-IV criteria for BDD, and it is essential for more severely ill and suicidal patients. [36]

First-line pharmacological treatment of BDD (including delusional BDD) is centered on the use of selective serotonin reuptake inhibitors (SSRIs), with the incorporation of clomipramine when necessary. [3, 23, 36]Following reports of cases that responded to SRIs, a largely retrospective study of 30 patients found that 58% responded to SRIs compared to only 5% for other medications.[8]

SRI medication refers to all of the selective SRI (SSRI) class of antidepressants (fluoxetine, sertraline, paroxetine, citalopram, escitalopram, and fluvoxamine) and one antidepressant from the tricycle class, clomipramine, which is a potent SRI.[4] They are the medication of choice for OCD and they 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.[36] A growing literature also suggests that SRIs are also often effective for compulsive skin picking.[12]

Successful SRI treatment results in less frequent and intense appearance preoccupations, decreased BDD related distress, less intense urges and less time spent performing compulsive/safety behaviors, and better control over BDD preoccupations and compulsions.[5] Most studies have found that associated symptoms, such as depressive symptoms, anxiety, anger-hostility, functioning and life quality, often improve as well.[13] In addition, most studies have found that insight regarding the perceived appearance flaws improves with SRI treatment.[5] Importantly, SRIs may protect against worsening of suicidal behaviors and decrease suicidal ideation in individuals with BDD.[7]

Symptoms significantly improved in open-label studies of fluvoxamine, citalopram, and escitalopram (Ns ranging from 15 to 30), and response rates across these studies ranged from 53% to 73%. [13]
Five open-label trials of fluvoxamine, citalopram, and escitalopram found that these SRI's improved BDD and associated symptoms in 63%–83% of patients.[4]
Among SSRIs, there is currently no particularly beneficial drug of choice, although escitalopram and fluoxetine are often administered. The exception is citalopram; BDD treatment requires relatively high doses, and citalopram has been associated with cardiac side effects at the dosages required. [3]

Although fluvoxamine and clomipramine are the most thoroughly studied SRIs, clinical experience suggests that all of the SRIs may be effective for BDD.[3] For an individual patient, one SRI may be more effective than another, although the SRI that is most effective for a given patient must be determined by trial and error and cannot be predicted. [12]

SRI monotherapy appears as efficacious for patients with delusional BDD beliefs as for those with nondelusional beliefs.[7] SRIs also appear more efficacious than non-SRI antidepressants or other types of psychotropic medication.[7] Althougt some non-SRI

psychotropic medications may be useful as adjunctive treatments with SRIs, they are generally ineffective for BDD when used as single agents.[12] Of interest, SRIs as monotherapy appear to effectively treat delusional BDD, whereas antipsychotics—as either monotherapy or SRI augmentors—do not appear to. [34]

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). 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). In this study, no non-SRI medications (haloperidol, perphenazine, imipramine, carbamazepine, clonidine, lithium, methylphenidate, and dextroamphetamine; n=8 trials) were effective for BDD.[36]

In addition, while the above studies indicate that a majority of patients with BDD significantly improve with SRI treatment, response is often partial, and a substantial minority of patients do not improve in a significant clinically significant degree. [35]

SSRIs are recommended for moderate-to- severe BDD, with the proviso that a high rate of relapse is likely to occur on their discontinuation. [26] In fact, in a study – a small chart-review study from a clinical practice – 83% (N=31) of patients who discontinued an effective SRI experienced subsequent relapse of BDD. [30]

**Clomipramine**

In a controlled and blinded crossover study (N=29), clomipramine (a tricyclic antidepressant with SRI properties) was significantly more efficacious than desipramine (a non-SRI antidepressant) for BDD symptoms, depressive symptoms, and functional disability, with 8 weeks of treatment with each medication.[13, 36] 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). [36]

In fact the clomipramine-desipramine study is the most methodologically rigorous study to examine the efficacy of a non-SRI medication for BDD and it was 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.[36] However clomipramine is usually reserved for cases in which SSRIs have not proven to be of benefit, as the side effect profile of SSRIs tends to be milder than that of tricyclic antidepressants (TCAs). [3]

Of interest, in this series 30% of 23 trials with MAO inhibitors were efficacious. Combined with some positive case reports, this finding suggests that MAOIs might be tried with more treatment-refractory patients.[36]

**Venlafaxine**

A small open-label trial (n=11) found that venlafaxine significantly improved BDD symptoms in study completers. However, until more methodologically rigorous studies in larger samples are done, it is recommended an SRI rather than an SNRI such as venlafaxine as a first-line medication for BDD.[36]

Results of a small open-label trial (N=17) also suggested that venlafaxine may be efficacious for BDD; however, serotonin-norepinephrine reuptake inhibitors have not received additional investigation, and therefore they are not currently considered a first-line treatment.[13]

The use of medications should be considered within a broader framework of their potential benefits and adverse effects. [5]

**Citalopram**

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. [36]

Psychosocial functioning and mental health-related quality of life also significantly improved. [36]

**Escitalopram**

An open-label study of escitalopram in BDD that included 14 subjects found that almost one-half of subjects were very much improved, and one-third were much improved. There also was a significant decrease in delusionality of appearance beliefs. [20]

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.[36]

The first study to investigate the efficacy and tolerability of escitalopram in BDD found significant improvement across numerous domains.[31] Fifteen subjects with BDD were treated with escitalopram and assessed with reliable and valid measures. BDD symptoms significantly improved ($P < 0.001$), and 73.3% (n = 11) of subjects were responders. [31]

Of note, delusional patients were as likely to improve as nondelusional patients, which is consistent with previous BDD studies.[31] Functioning and quality of life also significantly improved after only 12 weeks of treatment, as in previous studies. SF-36 and Q-LES-Q scores were markedly poor at baseline (mean of 1.8 SD poorer compared to the general population or community norms); at study termination, mean scores were only 0.2 SD poorer. [31] It is notable that almost one-half (46.7%) of patients were very much improved on the BDD-CGI. [31] BDD responded relatively quickly (4.7 ± 3.7 weeks), which is similar to a study with citalopram (4.6 ± 2.6 weeks). By contrast, the mean time to response in other SRI studies was 6–9 weeks. However, these comparisons should be made with

caution because no direct study has been made comparing different SRIs, and such studies are needed to better understand the time of response and the degree of response. [31]

In another study 100 adults with DSM-IV BDD received open-label escitalopram for 14 weeks (Phase 1); 58 responders were then randomized to double-blind continuation treatment with escitalopram versus switch to placebo for six months (Phase 2).[30]Consistent with the prior research, BDD-related insight, depressive symptoms, psychosocial functioning, and quality of life also improved significantly with open-label escitalopram. The mean Phase 1 escitalopram dose (26.2 ± 7.2 mg/day) is higher than doses often used to treat anxiety disorders and depression but similar to doses usually recommended for OCD and typically used for BDD. However, no dose-finding studies have been conducted in BDD. [30]

Of note, delusional patients were as likely to improve as nondelusional patients, which is also consistent with previous BDD studies. However, unlike prior studies, there was a trend for those with nondelusional BDD beliefs to be more likely to respond to escitalopram. [30]

A recent trial of open-label escitalopram for use in maintenance therapy for six months found that the time to relapse was longer with the administration of escitalopram relative to placebo and that the rates of relapse were less for those on escitalopram compared to those switched to placebo (18% vs. 40%). This study highlights that escitalopram is an effective treatment for BDD compared to placebo and additionally that there is a risk of relapse when an efficacious SRI medication is stopped.[3, 4] These findings are particularly relevant clinically because BDD is often chronic. [30]

Indefinite continuation of therapy may maintain remission but may be associated with side-effects due to medication profile. Discontinuation, if chosen, should occur slowly, with a tapering process over several months.[3]

**Fluvoxamine**

The best-studied SRIs in BDD are fluvoxamine and clomipramine. [12] A 16-week open-label fluvoxamine treatment study that included 27 subjects showed significant improvement in BDD symptoms for both delusional and nondelusional patients. [20]

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 ($p<.001$). Of the 30 subjects, 63% responded to fluvoxamine and fluvoxamine response was not related to illness severity. [36]

In another 10-week open-label fluvoxamine study of 15 patients, 10 of them resulted in a much or very much improvement on the Clinical Global Impressions Scale (CGI) and ten of the 12 patients who completed the study were responders. [36]

**Fluoxetine**

A 12-week randomized trial compared fluoxetine with placebo in 67 subjects with BDD. As measured by the Yale-Brown Obsessive Compulsive Scale, Modified for Body Dysmorphic Disorder (BDD-YBOCS), fluoxetine response (53%) was markedly greater

than with placebo (18%). Delusionality did not influence response to treatment, and treatment response was independent of symptom severity, duration, and the presence of any comorbidities. [3]

In another study including 74 patients with BDD or its delusional variant found fluoxetine to be safe and more effective than placebo in delusional and nondelusional patients with BDD. [20]

In a 12-week placebo-controlled study of fluoxetine in the treatment of BDD, the authors investigated change in psychosocial functioning and mental health-related quality of life in 60 subjects. It is notable that psychosocial functioning as assessed by the SOFAS and LIFE-RIFT improved significantly more with fluoxetine than placebo after only 12 weeks of treatment. [32] The mean LIFE-RIFT scores in the fluoxetine group were generally in the range of satisfactory functioning to mildly impaired functioning. The mean SOFAS score in the fluoxetine group at study endpoint (mean = 71.1 [SD = 17.8]) approached the low end of the normal range. These improvements were similar to those in a small open-label study of citalopram in BDD. [32] Once functioning and quality of life are critically important components in treatment efficacy, further studies are needed to confirm those findings and to determine how generalizable those results are to other body dysmorphic disorder patients.[32]

Of note, fluoxetine was not associated with worsening or emergence of suicidality in BDD, a disorder with a high risk of suicidality.[38] In fact, fluoxetine was found not to differ from placebo in the emergence of suicidality in subjects with BDD, suggesting that fluoxetine also has a specific effect in reducing suicidality. [3] This result is consistent with recent studies in other disorders suggesting that antidepressants are associated with a decrease, rather than an increase, suicide tendencies. [38]

**Levetiracetam**

A reason to examine the efficacy of the antiepileptic levetiracetam for BDD is that an open-label study suggested that levetiracetam may be efficacious for social phobia, which has similarities to BDD.[35] Similarities include, in BDD, high levels of social anxiety, avoidance of social and a tendency to misinterpret ambiguous social scenarios as threatening. In addition, the presences of high rates of comorbid lifetime social phobia in BDD, suggest that BDD and social phobia may be related disorders.[35]

In a pilot study which explored the efficacy and safety of the antiepileptic medication levetiracetam for BDD, 17 subjects with DSM-IV BDD participated in a 12-week open-label levetiracetam trial. Subjects were assessed at regular intervals with standard measures.[35] In intention-to-treat analyses, scores on the BDD-YBOCS, the primary outcome measure significantly decreased, and 52.9% of subjects responded to levetiracetam. The response rate was higher (63.6%) in a secondary analysis of those subjects who completed the study.[35] This pilot study provides preliminary data suggesting that levetiracetam may diminish BDD symptoms, BDD-related delusionality, and depressive symptoms, and may also improve psychosocial functioning/quality of life.[35] Importantly, levetiracetam was relatively well tolerated.[35]

The underlying mechanism of levetiracetam for improving BDD symptoms is unknown. However, levetiracetam's effects on gamma-aminobutyric acid (GABA) receptors might have a role in treating BDD, and this possibility should be explored in further studies. [37]

**SRI Dosing**

There is an absence of dose-finding studies of SRI medications in BDD; however, the clinical experts in the field have suggested that higher doses are required comparing to depression and to those typically recommended for eating disorders, [4, 14]

In a chart-review study (n=90), the mean SRI doses were 66.7 ± 23.5 mg/day of fluoxetine, 308.3 ± 49.2 mg/day of fluvoxamine, 55.0 ± 12.9 mg/day of paroxetine, 202.1 ± 45.8 mg/day of sertraline, and 203.3 ± 52.5 mg/day of clomipramine. Some patients only respond to higher doses than the maximum recommended dose.[8]

The current recommendations are that the SRI should be taken for at least 12 weeks before determining whether it is efficacious. [5] In most studies, in which fairly rapid dose titration were used, the average time required for BDD to respond was 6-9 weeks and in some cases 12 or even 14 weeks. It is therefore recommended that patients receive an SRI for at least 12 weeks before switching to another SRI, and that the highest SRI dose recommended by the manufacturer (if tolerated) is reached if lower doses are ineffective.[8] However**,** not uncommonly, for some patients, the response is optimized when the maximum SRI dose recommended by the manufacturer is exceeded. In other words, some patients may benefit from SSRI doses that exceed the maximum dose recommended by the pharmaceutical company (although this dose should not be exceeded for clomipramine or citalopram).[7,36] 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. [36]

How rapidly the dose is raised will depend on several factors. Generally it is suggested 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. It is also suggested 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, may make it more difficult to determine whether a lower dose might be effective for a particular patient. 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. [36]

## 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.[36]

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.[36]

## Continuation and Maintenance SRI Treatment

No published continuation or maintenance studies have been done. 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. 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 1point improvement on this scale between the beginning and end of the 6-month continuation phase. This finding is consistent with 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.[36] Based on clinical experience, it is recommended that an effective SRI should be continued for several years, if not longer. It 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.[36]

## 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 are from a chart-review study in which 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 ($p<.0001$). The average 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. [36]

Relapse appears to be common after discontinuation of an effective SRI, and longer-term SRI treatment is often needed.[13] For patients who appear at high risk for suicide, lifelong SRI treatment is recommended, as suicides have been known to occur after SRI discontinuation.[13]

## 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.[36]

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. [36]

## 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.[36]

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. In the desipramine/clomipramine study clomipramine was even more effective for delusional patients than for nondelusional patients. [36]

These findings are consistent with earlier findings from case reports, clinical series and open-label trials which also indicated that delusional patients have a high response rate to SRIs alone (i.e., without an antipsychotic). These data also suggest that antipsychotics alone are only rarely effective for delusional BDD. Thus, it is currently recommended the treatment of delusional BDD with an SRI and not with an antipsychotic alone.[36]

In contrast, available data also suggest that greater delusionality/overvalued ideation in BDD may be associated with a poorer response to CBT.[36]

## 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.[36]

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. [36]

Switching from one SRI to another is a good option. In a 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. [36]

Only one controlled study has examined SRI augmentation in BDD. A 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. 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 and there was no significant effect of baseline delusionality on endpoint BDD severity (i.e., more delusional patients did not have a better response). [36]

However, data are still very limited, and additional studies of SRI augmentation with antipsychotics are needed. 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 of suicidal or violent behavior.

In addition an atypical (or second-generation) antipsychotic may be more helpful than a typical antipsychotic, and ziprasodone seems particularly promising. According to this study patients should receive an antipsychotic for BDD only in combination with an SRI.[36]

Augmentation with buspirone, a 5HT1A partial agonist, is appealing because this medication is usually 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. Six subjects (46%) improved. In a chart-review study, 33.3% (n=12) of SRI augmentation trials with buspirone led to significant improvement, with a large effect size. The mean buspirone dose was 56.5 ± 15.2 mg/day. [36]

Adding clomipramine to an SSRI, or vice versa, is another option. In a chart-review study, this approach resulted in a response of 44.4% (n=4) of cases, with a small to medium effect size. 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, when adding clomipramine to an SSRI clomipramine levels should be monitored, starting at a clomipramine dose of only 25 – 50 mg/day. [36]

Clinical experience suggests that occasional patients respond well to SRI augmentation with lithium or methylphenidate. These approaches may also improve depressive symptoms, suicidality, or anergia.[36]

Venlafaxine augmentation of SSRIs is also promising.[36]

Adjunctive benzodiazepines should be considered for very anxious or agitated patients. The potential for substance abuse or dependence must be considered, although clinical experience suggests that relatively few BDD patients abuse these medications. [36]

However, 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 clinical experience suggests that 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). [36]

It not also clear whether augmentation is better than switching to another SRI, or vice versa. In a 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. 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%).[36] Whether to switch or augment is a complex decision that requires clinical judgment and understanding of the particular patient. [36]

**Cognitive Behavioral Therapy**

Available research suggests that cognitive-behavioral therapy (CBT) may be efficacious for BDD.[5] Most studies have examined a combination of cognitive components (eg, cognitive restructuring that focuses on changing appearance-related assumptions and beliefs) with behavioral components, consisting mainly of exposure and response prevention (ERP) to reduce avoidance and compulsive and safety behaviors.[5]
Exposure consists on having patients exposing the perceived defect in social situations (for example, going to formerly avoided restaurants or stores without a hat or heavy makeup, or sitting in a crowded waiting room).[12] Response prevention consists on helping patients avoiding BDD behaviors, using techniques such as covering or removing mirrors, limiting grooming time, covering skin areas that are picked, and stopping makeup use and seeking reassurance. Cognitive restructuring, which helps patients change erroneous beliefs about their appearance and the importance attributed to appearance, is also often part of treatment.[12]

CBT for BDD is based on a neurobiologically informed cognitive behavioral model of BDD. This treatment consists of psychoeducation; case formulation; setting valued goals; motivational enhancement; cognitive restructuring; exposure and ritual prevention; mindfulness and attentional retraining; and advanced strategies to modify self-defeating assumptions about the importance of appearance and to enhance self-acceptance, self-esteem, and self-compassion.[22] An intensive ritual prevention is a core component of treatment for BDD, which is not needed for depression or social phobia.[23]
In addition, certain BDD symptoms may require specialized techniques, such as the use of habit reversal training for compulsive skin-picking or hair-plucking.[5]

Treatment must also target other problematic symptoms linked to BDD, such as surgery seeking and skin picking/hair pulling done in an attempt to improve one's appearance**.**[23] Some protocols include additional modules that allow for the therapist to address BDD-specific symptoms that might not affect all patients (eg, skin picking/hair pulling, surgery-seeking, and shape/weight concerns) in a flexible manner. [27]

Treatment with CBT involves determining the maladaptive thoughts and behaviors that are maintaining the patient's BDD symptoms and undermining their functioning and finding ways to change these maladaptive thoughts and behaviors.[20] The treatment also usually involves relaxation training, including deep breathing and muscle relaxation, which could also be applied for patients with BDD. [20]

Although there is some evidence that both the behavioral and cognitive aspects of CBT are effective in isolation, the relative contribution of these respective elements in combined treatment requires further investigation. Nevertheless, there are preliminary indications that the behavioral component of CBT may result in lower relapse rates that those found in SRI trials. [33]
Early case reports indicated a successful outcome with exposure therapy and cognitive plus behavioral techniques.[8]

Findings from neuropsychological research support the use of cognitive-behavioral strategies to help patients reducing the focus on minor details of their appearance and to instigate a view of their body in a much "holistically" way.[5]

In adult populations, six RCTs have demonstrated CBT to be efficacious in reducing BDD severity compared with no treatment or wait-list control conditions, supportive therapy, and anxiety management.[29] They have demonstrated the efficacy of CBT for BDD in adults, with response rates ranging from 48% to 82%. [21]

A pilot controlled trial conducted by Veale et al tested a specific CBT model for BDD patients, patients with real disfigurements who seek cosmetic surgery but are emotionally well-adjusted, and healthy controls without any defect. They found significant changes in the group treated with CBT who obtained a 50% reduction in symptoms using the Yale-Brown Obsessive Compulsive Scale (modified for BDD) and Depression Rating Scale as outcome measures.[20]

As insight regarding the appearance beliefs is often poor and patients may be very reluctant about initiate or remain in psychological treatment, motivational interviewing techniques will often need to be applied at later stages of therapy as well.[4,23] In fact BDD patients typically need more intensive engagement and ongoing motivational interventions than patients with other disorders such as OCD. [23]

Cognitive interventions are more complex and intensive for BDD than for OCD or social phobia because of the delusional nature of BDD beliefs and delusions of reference. Exposure exercises and behavioral experiments are needed for prominent social avoidance, unlike treatment for OCD, depression, or eating disorders. [23]

In a report of five patients with BDD, four improved using such approaches in 90-minute sessions one day or five days per week.[12]

Early case reports indicated that exposure therapy may be effective. In a subsequent series, in which BDD patients (n=17) received 20 sessions of daily individual 90-minute CBT, BDD symptom severity significantly decreased.[5] In an open trial of group CBT (n=13), administered in twelve- 90-minute sessions, BDD and depressive symptoms significantly improved (from severe to moderate).[5]

Two wait-list controlled studies have been published.[8] In a randomized study of individual CBT (N=19) patients who received 12 weekly sessions of 60-minute individual CBT improved significantly more than those in a no-treatment wait-list control condition.[8, 13] In another study (n=54), women randomized to cognitive therapy plus ERP (provided in 8 weekly 2-hour group sessions) improved more than those randomized to a no-treatment wait-list control condition.[8] BDD symptoms were significantly decreased in therapy subjects and the disorder was eliminated in 82% of cases at posttreatment and 77% at follow-up.[20] Overall psychological symptoms and self-esteem also improved in therapy subjects. [17]

In another study, exposure and response prevention plus cognitive techniques were effective in 77% of 27 women who received this treatment in eight weekly two-hour group sessions. [12]

In a study of ten participants who received thirty 90-minute individual of ERP sessions without a cognitive component, and 6 months of relapse prevention, improvement was maintained at up to 2 years. [5]

There is only one study that has compared CBT for BDD versus another type of therapy. This study randomly allocated 46 adult patients with BDD to either receive CBT (with individual sessions of 1h at weekly intervals) or anxiety management (AM)

(provided once a week for 12 weeks, with each session lasting 1h). AM treatment followed a standard protocol and consisted of (1) practicing progressive muscle relaxation and breathing daily, (2) identifying triggers and physical symptoms associated with appearance-related anxiety and (3) utilizing brief muscle relaxation and breathing techniques in trigger situations.[4,26]
Fifty-four percentage of participants were classified as having a delusional BDD. [4] At 12 weeks, CBT was found to be significantly superior to AM in what concerns reducing symptom severity and on improving the secondary outcome measures such as quality of life and level of insight.[4] Outcomes improved even further after 4 additional CBT sessions, and gains were maintained at the 1-month follow-up.[7]
This effect was also seen for those individuals with delusional beliefs or depression suggesting that CBT is just as effective as reducing BDD severity in these more impaired groups. [4]

The National Institute for Health and Clinical Excellence guidelines recommend CBT that is specific for BDD, which follows a protocol over 16–24 sessions. [4]

Although it is well established that CBT for BDD is associated with significant symptom relief in the short term, longer-term outcomes are less clear. A recent meta-analysis of CBT for BDD concluded that gains are likely to be maintained for a least 2–4 months following treatment.[29]
Existing RCTs in adults have included follow-up periods ranging from 1 to 6 months and have shown preservation of gains over this period. [29]

Only two studies have examined longer-term outcomes (McKay, 1999, Veale et al., 2015). McKay (1999) found that gains were maintained at 2-year follow-up among 10 patients who had received behavior therapy with or without an additional relapse prevention program. In a larger study, Veale et al. (2015) examined outcomes among 30 patients 1–4 years after completing CBT. Overall, symptoms remained stable and the relapse rate was relatively low (n = 4, 13.3%). However, it is of note that 12 patients (30.8%) were on medication at the long-term follow-up and 10 (25.6%) had received further psychological treatment, which may have contributed to the positive outcomes. [29]

**Individual vs Group CBT**

Although methodologically heterogeneous, studies suggest that individual and group CBT are empirically supported treatments for BDD. [27]

Two open studies and at least one controlled study (using a waiting list) have suggested that individual CBT is an effective treatment for BDD. The combination of ERP and cognitive therapy (ie, CBT) has been shown to be potentially effective in BDD in different forms, ie, weekly or intensive CBT, CBT associated with role playing, "modular" CBT, and CBT associated with medication or psychosocial rehabilitation, even among patients who present comorbidity with various personality disorders.

Patients treated with individual CBT showed decreased concern with their imagined physical defects, their overvalued and delusional ideas, their obsessive and compulsive symptoms, anxiety and depression, as well as in their professional performance and social

activities.[27] Nevertheless, one controlled study suggests that cognitive therapy added to ERP does not result in significant gains as compared with pure ERP.[27]

On the other hand, one open study and one controlled study (using a waiting list) revealed favorable effects of group CBT in the treatment of BDD, both in relation to symptomatology of the disorder and in relation to comorbid depression. These results reinforce the importance of controlled studies which investigate the use of group CBT on the treatment of BDD because this method of treatment may be financially advantageous for the public health system, as it allows the treatment of a greater number of patients at a lower cost. [27]

**Modular Cognitive–Behavioral Therapy for Body Dysmorphic Disorder**

This CBT model is based on the premise that individuals with BDD misinterpret visual input of normal appearance features or minor appearance flaws in biased ways that result in negative cognitive, emotional, and behavioral consequences of BDD. Furthermore, according to this cognitive–behavioral model, individuals with BDD tend to elevate the meaning and importance of these minor, or even nonexistent, physical imperfections and react to negative interpretations with anxiety, depressed mood, shame, and further excessive attention to the perceived flaws. Additionally, the model postulates that negative feelings resulting from this faulty cognitive processing lead to attempts to neutralize these feelings with ritualistic behaviors (e.g., surgery seeking) and avoidance of situations (e.g., social situations) that trigger these unpleasant feelings. Because these ritualistic behaviors and avoidance behaviors sometimes temporarily diminish painful emotions, they are negatively reinforced, and, in this way, are hypothesized to maintain dysfunctional BDD-related behaviors. [24]

This manual which is broadly applicable to BDD patients, including those with delusional beliefs, includes core treatment elements and optional treatment modules that allows a more flexible, personalized treatment for each patient. [24]

Briefly, subjects received 22 sessions of CBT-BDD that included the following core components: 1) *psychoeducation and case formulation* based on the information obtained during the assessment, the therapist and patient together derived a cognitive–behavioral model for the patient's specific BDD symptoms, including hypothesized mechanisms that were causing and/or maintaining the symptoms; **2)** *motivational enhancement strategies* to be used whenever patients exhibited ambivalence about starting treatment or to enhance motivation as needed during the course of their treatment. **3)** *cognitive restructuring*, identifying unrealistic negative thoughts and beliefs about appearance; **4)** *exposure and ritual prevention*: after identifying avoidance behaviors, the therapist and the patient developed an exposure hierarchy of situations that provoked anxiety or discomfort. To reduce ritualistic behaviors like excessive mirror checking, participants monitored the frequency and contexts in which they performed their rituals, and identified strategies to resist them in order to reduce avoidance and safety behaviors/rituals; **5)***mindfulness interventions and perceptual retraining* to help patients learn to broaden their perspective and attend to

aspects of appearance other than perceived defects. This helped patients learn to observe and describe their entire body in objective, non-judgmental language while standing a normal distance (e.g., two to three feet) from the mirror and refraining from safety or ritualistic behaviors while in front of the mirror. In addition, patients learned to retrain their attention when interacting with other people; rather than just comparing their disliked body parts to the same body parts of others, they learned to pay attention to other aspects of appearance and nonappearance characteristics of others, as well as their surroundings. 6) *Advanced cognitive restructuring* helped patients learn to modify deeply held core beliefs and included interventions to decrease the importance of appearance which help patients to base their self-esteem on other aspects of themselves (e.g., talents, intelligence, moral values). ***7)*** *relapse prevention* focused on consolidation of skills and maintenance of gains. During relapse prevention, therapists helped patients prevent, expect, and react effectively to setbacks, and taught patients to conduct self-therapy sessions. [24, 25] **8)** *modular interventions:* In addition to these core components, which all participants received, the treatment included four optional treatment modules selected for use by therapists if relevant to a participant's particular symptoms. Therapists chose from the following modules:[24,25] (a) Skin picking and hair plucking: this module uses habit reversal for compulsive skin picking and hair pulling, which more than one-third of BDD patients engage in to try to improve their appearance; (b) Muscularity and shape/weight module: this module is more often used for patients (mostly males) with muscle dysmorphia, a particularly malignant form of BDD associated with anabolic steroid abuse in which patients think they are insufficiently big and muscular. This module is also used for patients who think they are too fat [24] ;
(c) Cosmetic treatment module: this module contains psychoeducation for patients who are considering cosmetic treatment (e.g., surgical, dermatologic) for BDD symptoms**.** A majority of BDD patients receive cosmetic treatment for their perceived appearance flaws.[24] In fact in the largest study to date, 76% of 250 adults with BDD sought, and 66% received non-psychiatric treatment for their perceived appearance defect, most commonly dermatological and surgical.[16] However, nonpsychiatric treatment rarely improves overall BDD symptoms. [39] In fact, in a study conducted with 200 individuals with BDD, subjects retrospectively reported that only 3.6% of all treatments resulted in overall improvement in BDD.[5] Cognitive and motivational strategies are used to address maladaptive beliefs about the perceived benefits of surgery as well as to enhance patients' engagement in CBT.[24];
(d) Mood management module: this module includes activity scheduling and other approaches for more severely depressed patients.[24] Usually a therapist would select specialized modules after the patient had been introduced to all core CBT-BDD skills**.** [25]

Results from this pilot study suggest that this modular manualized CBT treatment for BDD is feasible to implement and appears to be an effective treatment for patients with this disorder.
Mean BDD symptom severity scores improved from moderately severe to the subclinical range, and depressive symptoms significantly improved from moderate to mild. The relatively high retention rate suggests that this intervention is acceptable to most patients, as more than 80% of those who began treatment completed it. Moreover, follow-up data

showed that patients who completed treatment maintained their gains for at least 6 months following treatment completion. In addition, patients reported very high levels of satisfaction with the treatment (88.9%) based on the CSI. [24]

When examining the efficacy of CBT-BDD relative to a waitlist control condition in adults with BDD, the high response rate after 22 sessions of treatment (81-83%) is similar to what was reported in an open trial of this new treatment in which 75% of the ITT sample and 80% of treatment completers were responders based on the BDD-YBOCS criterion. Consistent with earlier CBT studies, this research showed that post-treatment gains of CBT-BDD were maintained during an extended follow-up period.[25] These findings suggest that CBT specifically developed for BDD also improves BDD-related insight and associated symptoms such as depression and disability. The seven (19%) participants who dropped out or were withdrawn early from the study had a higher BDD-YBOCS total score, a higher BDI-II total score, and were more likely to have a personality disorder than those who completed the study. [25] From a clinical perspective, it appears that those who terminated early were more severely ill. Of the seven patients who dropped out, four terminated prior to beginning CBT (2 at baseline and 2 during/immediately after the waitlist). It might not have been tolerable for those who were suffering to this degree to comply with all of the study procedures and a delayed start of treatment. The remaining three drop-outs dropped during CBT-BDD (1 at week 4 and 2 at week 8). Thus, while this study is small and available information is limited, it appears that dissatisfaction with treatment does not appear to be the primary reason for dropout, other reasons appear to be more compelling (e.g., moving out of the area).[25]

Predictors of early termination indicate that in future treatment studies special attention may be needed to retain participants with more severe BDD, more severe depression, and those with comorbid personality disorders. [25]

**Predictors of Response to CBT for Body Dysmorphic Disorder**

While CBT for BDD is effective, not everyone improves or improves fully. Additionally, BDD often requires a longer treatment (e.g., 22 sessions), thus utilizing significant resources. Identifying predictors of CBT response could inform important targets to address prior to or at the onset of treatment to optimize outcome. [21]

This propose treatment was delivered in weekly individual sessions over 18–22 weeks and results indicated that greater motivation/readiness to change (University of Rhode Island Change Assessment Questionnaire), greater treatment expectancy (Treatment Credibility/Expectancy Questionnaire), and better baseline BDD-related insight (Brown Assessment of Beliefs Scale) significantly predicted better CBT response at post-treatment.[21]

On the other hand, poorer BDD-related insight at baseline significantly predicted a lower chance of treatment response. However, another study of CBT for BDD did not find that poorer insight predicted CBT response, although statistical power was limited (n=46, 13 responders; Veale et al., 2014). Thus, the relationship between BDD-related insight and treatment outcome is not entirely clear. [21]

In addition, baseline BDD symptom severity, depression, and impairment in psychosocial functioning were not significant predictors of post-treatment outcome, consistent with the results from a recent meta-analysis Harrison et al., (2016) and the Veale et al. (2014) trial, in which baseline depression did not predict response. These results suggest that even individuals with severe BDD, severe depression, and high levels of functional impairment can be treated successfully with CBT for BDD. This is encouraging, given the frequency of comorbid major depressive disorder and often high levels of depression, as well as substantial functional impairment, in individuals with BDD. The results support previous findings that depression and psychosocial impairment improve in response to a targeted CBT for BDD, without requiring additional treatment.[21]

Taken together, CBT for BDD is often efficacious even for those who are severely ill, depressed, and functionally impaired (none of which significantly predicted treatment outcome). However, because these patients may be at greater risk of dropping out in an early stage of the treatment, specific strategies, such as behavioral activation, in addition to motivational enhancement, may be needed early and often to ensure these individuals receive the full treatment.[21]

**Pharmacologic treatment vs CBT**

At this time it is not known whether medication or CBT are more efficacious for BDD, as no randomized controlled studies have directly compared them.[4] Furthermore, it is not known whether a combination of medication and CBT is more efficacious than either treatment alone.[5] However, based on clinical experience, the authors recommend that all patients with severe BDD, severe depressive symptoms, or active suicidal ideation receive an SRI and ideally both treatments.[5] An SRI is recommended for those patients, as partial response may make CBT more tolerable and enable patients to participate in CBT treatment.[8]

However, clinical experience suggests that some patients respond well to either an SRI or CBT alone, whereas others benefit from treatment with both types of treatment.[9]

Before instituting an SRI and/or CBT, it is also important to provide psychoeducation on BDD. For patients who are reluctant to accept the diagnosis and treatment (e.g., delusional patients), it can be helpful to emphasize that treatment is likely to decrease their suffering and improve functioning.[8]

**Alternative psychosocial treatments**

Currently, CBT is the only psychosocial treatment with preliminary empirical support. Some patients, however, refuse CBT or terminate prematurely the therapy. Therefore, alternative treatments are needed. [5]

Interpersonal psychotherapy (IPT) may offer a promising substitute. Individuals with BDD often have a history of emotional abuse, long-standing interpersonal conflicts, and may suffer from crippling social anxiety and interpersonal problems. IPT enables patients to develop more effective strategies to reduce interpersonal distress, poor self-esteem, and depressed mood, which are hypothesized to maintain body image concerns. Results

from a small open trial pilot (n=9) regarding the preliminary efficacy of IPT for BDD are promising, and a randomized controlled trial is currently under way**.** [5]

Research on insight-oriented and supportive psychotherapy is extremely limited but suggests that BDD symptoms - especially severe symptoms - are unlikely to significantly improve with these treatments alone. However, they can be helpful for other problems the patient may have and may be a useful adjunct to CBT and/or an SRI. [8]

Supportive psychotherapy focuses on maintaining, improving, and restoring self-esteem and adaptive coping skills as well as on reflecting and expressing emotions about current life issues. The intent of this nondirective treatment is to help patients learn to cope with external and psychological challenges, emphasizing the therapeutic relationship and self-esteem as vehicles for patient improvement. [22]

In the first comparative study of therapist-delivered CBT-BDD and supportive psychotherapy, CBT-BDD was more consistently efficacious in reducing BDD-symptom severity, whereas the outcome was more variable with supportive psychotherapy. Changes in secondary symptoms mirrored those observed in BDD severity, although most were not statistically significant.[22] Of note, the supportive psychotherapy response rate (69.2% at MGH Massachusetts General Hospital and 45.5% at RIH Rhode Island Hospital among completers) exceeds the anxiety disorders and is much higher than the rate in a 12-week trial of internet-based CBT (54%) vs supportive psychotherapy (6%) for BDD. [22] Thus BDD severity and associated symptoms appeared to improve with both CBT-BDD and supportive psychotherapy, although CBT-BDD was associated with more consistent improvement in symptom severity and quality of life. [22]

## Conclusion

Despite BDD's prevalence and severity, this disorder remains underdiagnosed in clinical settings. [5] Given the markedly poor functioning and quality of life as well as high rates of suicidality among these patients, it is important that BDD is recognized and accurately diagnosed.[5] Of note, undiagnosed BDD commonly complicates affective and anxiety disorders, in particular social anxiety disorder. [15]

Available data suggest that cosmetic treatment is almost never effective for BDD; can make symptoms worse, and can trigger even violent behavior towards clinicians who provide such treatment.[7]

In contrast, an appropriate psychiatric treatment is often effective and can even be life-saving.[19]

Interventional research on BDD is still limited. However, available treatment data are promising and indicate that most patients improve with appropriate treatment that targets BDD symptoms specifically.[5]

No medication is approved by the FDA for the treatment of BDD. In fact, studies that are required for FDA approval have not been conducted in BDD.[5]

However, SRIs appears to be efficacious. In fact, all studies conducted to date, report that the majority of BDD patients shows an improvement, particularly when relative high doses are given. Therefore, they are recommended as the first-line medication for BDD, including delusional BDD.[5,9,36] It is suggested the use of the maximum SRI dose recommended by the pharmaceutical company, if tolerated, unless a lower dose is effective and for some patients it can be helpful to exceed this dose. [36] Additionally long-term treatment appears often necessary.[8]

In addition, CBT, using a combination of cognitive and behavioral techniques, may also be beneficial.[9] In fact, CBT is the best tested and most promising psychosocial treatment for adults with BDD.[23]

To sum up, treatment mainstays of BDD typically involve some combination of cognitive behavioral therapy (CBT) modalities and pharmacotherapy.[3] However, additional treatment development and efficacy studies are urgently needed, including larger CBT studies, CBT studies with control groups that receive treatments commonly used in the community (e.g. supportive psychotherapy), studies of CBT augmentation of SRIs and vice versa, and studies of other psychotherapies, non-SRI medications, and other somatic treatments.[7]

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# ANEXOS

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**Acta Med Port 2016, 30 dezembro 2016**

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## 5. CRITÉRIO DE AUTORIA

A revista segue os critérios de autoria do "International

Commitee of Medical Journal Editors" (ICMJE).

Todos designados como autores devem ter participado significativamente no trabalho para tomar responsabilidade pública sobre o conteúdo e o crédito da autoria.

Autores são todos que:

1. Têm uma contribuição intelectual substancial, directa, no desenho e elaboração do artigo
2. Participam na análise e interpretação dos dados
3. Participam na escrita do manuscrito, revendo os rascunhos; ou na revisão crítica do conteúdo; ou na aprovação da versão final
4. Concordam que são responsáveis pela exactidão e integridade de todo o trabalho

As condições 1, 2, 3 e 4 têm de ser reunidas.

Autoria requer uma contribuição substancial para o manuscrito, sendo pois necessário especificar em carta de apresentação o contributo de cada autor para o trabalho.

Ser listado como autor, quando não cumpre os critérios de elegibilidade, é considerado fraude.

Todos os que contribuíram para o artigo, mas que não encaixam nos critérios de autoria, devem ser listados nos agradecimentos.

Todos os autores, (isto é, o autor correspondente e cada um dos autores) terão de preencher e assinar o "Formulário de Autoria" com a responsabilidade da autoria, critérios e contribuições; conflitos de interesse e financiamento e transferência de direitos autorais / *copyright (modelo disponível em http://www.actamedicaportuguesa.com/info/AMP_template-Declaracao-Responsabilidade-Autoral.doc).*

O autor Correspondente deve ser o intermediário em nome de todos os co-autores em todos os contactos com a Acta Médica Portuguesa, durante todo o processo de submissão e de revisão. O autor correspondente é responsável por garantir que todos os potenciais conflitos de interesse mencionados são correctos. O autor correspondente deve atestar, ainda, em nome de todos os co-autores, a originalidade do trabalho e obter a permissão escrita de cada pessoa mencionada na secção "Agradecimentos".

## 6. *COPYRIGHT* / DIREITOS AUTORAIS

Quando o artigo é aceite para publicação é mandatório o carregamento na plataforma electrónica de documento digitalizado, assinado por todos os Autores, com a partilha dos direitos de autor entre autores e a Acta Médica Portuguesa.

O(s) Autor(es) deve(m) assinar uma cópia de partilha dos direitos de autor entre autores e a Acta Médica Portuguesa quando submetem o manuscrito, conforme minuta publicada em anexo:

Nota: Este documento assinado só deverá ser enviado quando o manuscrito for aceite para publicação.

Editor da Acta Médica Portuguesa

O(s) Autor(es) certifica(m) que o manuscrito intitulado: __ _____ (ref. AMP_____) é original, que todas as afirmações apresentadas como factos são baseados na investigação do(s) Autor(es), que o manuscrito, quer em parte quer no todo, não infringe nenhum *copyright* e não viola nenhum direito da privacidade, que não foi publicado em parte ou no todo e que não foi submetido para publicação, no todo ou em parte, noutra revista, e que os Autores têm o direito ao *copyright*.

Todos os Autores declaram ainda que participaram no trabalho, se responsabilizam por ele e que não existe, da parte de qualquer dos Autores conflito de interesses nas afirmações proferidas no trabalho.

Os Autores, ao submeterem o trabalho para publicação, partilham com a Acta Médica Portuguesa todos os direitos a interesses do *copyright* do artigo.

***Todos os Autores devem assinar***

Data:_____

Nome (maiúsculas):_____

Assinatura:_____

## 7. CONFLITOS DE INTERESSE

O rigor e a exactidão dos conteúdos, assim como as opiniões expressas são da exclusiva responsabilidade dos Autores. Os Autores devem declarar potenciais conflitos de interesse. Os autores são obrigados a divulgar todas as relações financeiras e pessoais que possam enviesar o trabalho.

Para prevenir ambiguidade, os autores têm que explicitamente mencionar se existe ou não conflitos de interesse.

Essa informação não influenciará a decisão editorial mas antes da submissão do manuscrito, os autores têm que assegurar todas as autorizações necessárias para a publicação do material submetido.

Se os autores têm dúvidas sobre o que constitui um relevante interesse financeiro ou pessoal, devem contactar o editor.

## 8. CONSENTIMENTO INFORMADO e APROVAÇÃO ÉTICA

Todos os doentes (ou seus representantes legais) que possam ser identificados nas descrições escritas, fotografias e vídeos deverão assinar um formulário de consentimento informado para descrição de doentes, fotografia e vídeos. Estes formulários devem ser submetidos com o manuscrito (modelo disponível em http://www.actamedicaportuguesa.com/info/consentimento_informado_do_doente.doc).

A Acta Médica Portuguesa considera aceitável a omissão de dados ou a apresentação de dados menos específicos para identificação dos doentes. Contudo, não aceitaremos a alteração de quaisquer dados.

Os autores devem informar se o trabalho foi aprovado pela Comissão de Ética da instituição de acordo com a declaração de Helsínquia.

## 9. LÍNGUA

Os artigos devem ser redigidos em português ou em inglês. Os títulos e os resumos têm de ser sempre em português e em inglês.

## 10. PROCESSO EDITORIAL

O autor correspondente receberá notificação da recepção do manuscrito e decisões editoriais por *email*.

Todos os manuscritos submetidos são inicialmente revistos pelo editor da Acta Médica Portuguesa. Os manuscritos são avaliados de acordo com os seguintes critérios: originalidade, actualidade, clareza de escrita, método de estudo apropriado, dados válidos, conclusões adequadas e apoiadas pelos dados, importância, com significância e contribuição científica para o conhecimento da área, e não tenham sido publicados, na íntegra ou em parte, nem submetidos para publicação noutros locais.

A Acta Médica Portuguesa segue um rigoroso processo cego (*single-blind*) de revisão por pares (*peer-review*, externos à revista). Os manuscritos recebidos serão enviados a peritos das diversas áreas, os quais deverão fazer os seus comentários, incluindo a sugestão de aceitação, aceitação condicionada a pequenas ou grandes modificações ou rejeição. Na avaliação, os artigos poderão ser:

a) aceites sem alterações;

b) aceites após modificações propostas pelos consultores científicos;

c) recusados.

Estipula-se para esse processo o seguinte plano temporal:

• Após a recepção do artigo, o Editor-Chefe, ou um dos Editores Associados, enviará o manuscrito a, no mínimo, dois revisores, caso esteja de acordo com as normas de publicação e se enquadre na política editorial. Poderá ser recusado nesta fase, sem envio a revisores.

• Quando receberem a comunicação de aceitação, os Autores devem remeter de imediato, por correio electrónico, o formulário de partilha de direitos que se encontra no *site* da Acta Médica Portuguesa, devidamente preenchido e assinado por todos os Autores.

• No prazo máximo de quatro semanas, o revisor deverá responder ao editor indicando os seus comentários relativos ao manuscrito sujeito a revisão, e a sua sugestão de quanto à aceitação ou rejeição do trabalho. O Conselho Editorial tomará, num prazo de 15 dias, uma primeira decisão que poderá incluir a aceitação do artigo sem modificações, o envio dos comentários dos revisores para que os Autores procedam de acordo com o indicado, ou a rejeição do artigo.

Os Autores dispõem de 20 dias para submeter a nova versão revista do manuscrito, contemplando as modificações recomendadas pelos peritos e pelo Conselho Editorial. Quando são propostas alterações, o autor deverá no prazo máximo de vinte dias, carregar na plataforma electrónica da Acta Médica Portuguesa uma versão revista do artigo, com as alterações inseridas destacadas com cor diferente, bem como um novo Documento Suplementar respondendo a todas as questões colocadas.

• O Editor-Chefe dispõe de 15 dias para tomar a decisão sobre a nova versão: rejeitar ou aceitar o artigo na nova versão, ou submetê-lo a um ou mais revisores externos cujo parecer poderá, ou não, coincidir com os resultantes da primeira revisão.

• Caso o manuscrito seja reenviado para revisão externa, os peritos dispõem de quatro semanas para o envio dos seus comentários e da sua sugestão quanto à aceitação ou recusa para publicação do mesmo.

• Atendendo às sugestões dos revisores, o Editor-Chefe poderá aceitar o artigo nesta nova versão, rejeitá-lo ou voltar a solicitar modificações. Neste último caso, os Autores dispõem de um mês para submeter uma versão revista, a qual poderá, caso o Editor-Chefe assim o determine, voltar a passar por um processo de revisão por peritos externos.

• No caso da aceitação, em qualquer das fases anteriores, a mesma será comunicada ao Autor principal. Num prazo inferior a um mês, o Conselho Editorial enviará o artigo para revisão dos Autores já com a formatação final, mas sem a numeração definitiva. Os Autores dispõem de cinco dias para a revisão do texto e comunicação de quaisquer erros tipográficos. Nesta fase, os Autores não podem fazer qualquer modificação de fundo ao artigo, para além das correcções de erros tipográficos e/ou ortográficos de pequenos erros. Não são permitidas, nomeadamente, alterações a dados de tabelas ou gráficos, alterações de fundo do texto, etc.

• Após a resposta dos Autores, ou na ausência de resposta, após o decurso dos cinco dias, o artigo considera-se concluído.

• Na fase de revisão de provas tipográficas, alterações de fundo aos artigos não serão aceites e poderão implicar a sua rejeição posterior por decisão do Editor-Chefe.

Chama-se a atenção que a transcrição de imagens, quadros ou gráficos de outras publicações deverá ter a prévia autorização dos respectivos autores para dar cumprimentos às normas que regem os direitos de autor.

## 11. PUBLICAÇÃO *FAST-TRACK*

A Acta Médica Portuguesa dispõe do sistema de publicação *Fast-Track* para manuscritos urgentes e importantes desde que cumpram os requisitos da Acta Médica Portuguesa para o *Fast-Track*.

a) Os autores para requererem a publicação *fast-track* devem submeter o seu manuscrito em http://www.actamedicaportuguesa.com/ "submeter artigo" indicando claramente porque consideram que o manuscrito é adequado para a publicação rápida. O Conselho Editorial tomará a decisão sobre se o manuscrito é adequado para uma via rápida (*fast-track*) ou para submissão regular;

b) Verifique se o manuscrito cumpre as normas aos autores da Acta Médica Portuguesa e que contém as informações necessárias em todos os manuscritos da Acta Médica Portuguesa.

c) O Gabinete Editorial irá comunicar, dentro de 48 horas, se o manuscrito é apropriado para avaliação *fast-track*. Se o Editor-Chefe decidir não aceitar a avaliação *fast-track*, o manuscrito pode ser considerado para o processo de revisão normal. Os autores também terão a oportunidade de retirar a sua submissão.

d) Para manuscritos que são aceites para avaliação

*fast-track*, a decisão Editorial será feita no prazo de 5 dias úteis.

e) Se o manuscrito for aceite para publicação, o objectivo será publicá-lo, online, no prazo máximo de 3 semanas após a aceitação.

## 12. REGRAS DE OURO ACTA MÉDICA PORTUGUESA

a) O editor é responsável por garantir a qualidade da revista e que o que publica é ético, actual e relevante para os leitores.

b) A gestão de reclamações passa obrigatoriamente pelo editor-chefe e não pelo bastonário.

c) O peer review deve envolver a avaliação de revisores externos.

d) A submissão do manuscrito e todos os detalhes associados são mantidos confidenciais pelo corpo editorial e por todas as pessoas envolvidas no processo de peer-review.

e) A identidade dos revisores é confidencial.

f) Os revisores aconselham e fazem recomendações; o editor toma decisões.

g) O editor-chefe tem total independência editorial.

h) A Ordem dos Médicos não interfere directamente na avaliação, selecção e edição de artigos específicos, nem directamente nem por influência indirecta nas decisões editoriais.

i) As decisões editoriais são baseadas no mérito de trabalho submetido e adequação à revista.

j) As decisões do editor-chefe não são influenciadas pela origem do manuscrito nem determinadas por agentes exteriores.

k) As razões para rejeição imediata sem peer review externo são: falta de originalidade; interesse limitado para os leitores da Acta Médica Portuguesa; conter graves falhas científicas ou metodológicas; o tópico não é coberto com a profundidade necessária; é preliminar de mais e/ou especulativo; informação desactualizada.

l) Todos os elementos envolvidos no processo de peer review devem actuar de acordo com os mais elevados padrões éticos.

m) Todas as partes envolvidas no processo de peer review devem declarar qualquer potencial conflito de interesses e solicitar escusa de rever manuscritos que sintam que não conseguirão rever objectivamente.

## 13. NORMAS GERAIS

### ESTILO

Todos os manuscritos devem ser preparados de acordo com o "AMA Manual of Style", 10th ed. e/ou "Uniform Requirements for Manuscripts Submitted to Biomedical Journals".

Escreva num estilo claro, directo e activo. Geralmente, escreva usando a primeira pessoa, voz activa, por exemplo, "Analisámos dados", e não "Os dados foram analisados". Os agradecimentos são as excepções a essa directriz, e deve ser escrito na terceira pessoa, voz activa; "Os autores gostariam de agradecer". Palavras em latim ou noutra língua que não seja a do texto deverão ser colocadas em itálico.

Os componentes do manuscrito são: Página de Título, Resumo, Texto, Referências, e se apropriado, legendas de figuras. Inicie cada uma dessas secções em uma nova página, numeradas consecutivamente, começando com a página de título.

Os formatos de arquivo dos manuscritos autorizados incluem o *Word* e o *WordPerfect*. Não submeta o manuscrito em formato PDF.

### SUBMISSÃO

Os manuscritos devem ser submetidos online, via "Submissão Online" da Acta Médica Portuguesa http://www.actamedicaportuguesa.com/revista/index.php/amp/about/submissions#onlineSubmissions.

Todos os campos solicitados no sistema de submissão *online* terão de ser respondidos.

Após submissão do manuscrito o autor receberá a confirmação de recepção e um número para o manuscrito.

**Na primeira página/ página de título:**

a) Título em **português** e **inglês**, conciso e descritivo

b) Na linha da autoria, liste o Nome de todos os Autores (primeiro e último nome) com os títulos académicos e/ou profissionais e respectiva afiliação (departamento, instituição, cidade, país)

c) Subsídio(s) ou bolsa(s) que contribuíram para a realização do trabalho

d) Morada e *e-mail* do Autor responsável pela correspondência relativa ao manuscrito

e) Título breve para cabeçalho

**Na segunda página**

a) Título (sem autores)

b) Resumo em **português** e **inglês**. Nenhuma informação que não conste no manuscrito pode ser mencionada no resumo. Os resumos não podem remeter para o texto, não podendo conter citações nem referencias a figuras.

c) Palavras-chave (*Keywords*). Um máximo de 5 *Keywords* em inglês utilizando a terminologia que consta no Medical Subject Headings (MeSH), http://www.nlm.nih.gov/mesh/MBrowser.html, devem seguir-se ao resumo.

**Na terceira página e seguintes:**

**Editoriais:**

Os Editoriais serão apenas submetidos por convite do Editor. Serão comentários sobre tópicos actuais. Não devem exceder as 1.200 palavras nem conter tabelas/figuras e terão um máximo de 5 referências bibliográficas. Não precisam de resumo.

**Perspectiva:**

Artigos elaborados apenas por convite do Conselho Editorial. Podem cobrir grande diversidade de temas com interesse nos cuidados de saúde: problemas actuais ou emergentes, gestão e política de saúde, história da medicina, ligação à sociedade, epidemiologia, etc.

Um Autor que deseje propor um artigo desta categoria

deverá remeter previamente ao Editor-Chefe o respectivo resumo, indicação dos autores e título do artigo para avaliação.

Deve conter no máximo 1200 palavras (excluindo as referências e as legendas) e até 10 referências bibliográficas. Só pode conter uma tabela ou uma figura. Não precisa de resumo.

**Artigos Originais:**

O texto deve ser apresentado com as seguintes secções: Introdução (incluindo Objectivos), Material e Métodos, Resultados, Discussão, Conclusão, Agradecimentos (se aplicável), Referências, Tabelas e Figuras.

Os Artigos Originais não deverão exceder as 4.000 palavras, excluindo referências e ilustrações. Deve ser acompanhado de ilustrações, com um máximo de 6 figuras/tabelas e 60 referências bibliográficas.

O resumo dos artigos originais não deve exceder as 250 palavras e serão estruturados (com cabeçalhos: Introdução, Materiais e Métodos, Resultados, Discussão e Conclusão).

A Acta Médica Portuguesa, como membro do ICMJE, exige como condição para publicação, o registo de todos os ensaios num registo público de ensaios aceite pelo ICMJE (ou seja, propriedade de uma instituição sem fins lucrativos e publicamente acessível, por ex. clinicaltrials.gov). Todos os manuscritos reportando ensaios clínicos têm de seguir o CONSORT *Statement* http://www.consort-statement.org/.

Numa revisão sistemática ou meta-análise siga as PRISMA *guidelines*.

Numa meta-análise de estudos observacionais, siga as MOOSE *guidelines* e apresente como um ficheiro complementar o protocolo do estudo, se houver um.

Num estudo de precisão de diagnóstico, siga as STARD *guidelines*.

Num estudo observacional, siga as STROBE *guidelines*.

Num *Guideline* clínico incentivamos os autores a seguir a GRADE *guidance* para classificar a evidência.

**Artigos de Revisão:**

Destinam-se a abordar de forma aprofundada, o estado actual do conhecimento referente a temas de importância. Estes artigos serão elaborados a convite da equipa editorial, contudo, a título excepcional, será possível a submissão, por autores não convidados (com ampla experiência no tema) de projectos de artigo de revisão que, julgados relevantes e aprovados pelo editor, poderão ser desenvolvidos e submetidos às normas de publicação.

Comprimento máximo: 3500 palavras de texto (não incluindo resumo, legendas e referências). Não pode ter mais do que um total de 4 tabelas e / ou figuras, e não mais de 50-75 referências.

O resumo dos artigos de revisão não deve exceder as 250 palavras e serão estruturados (com cabeçalhos: Introdução, Materiais e Métodos, Resultados, Discussão, Conclusão.

**Caso Clínico:**

O relato de um caso clínico com justificada razão de publicação (raridade, aspectos inusitados, evoluções atípicas, inovações terapêuticas e de diagnóstico, entre outras). As secções serão: Introdução, Caso Clínico, Discussão, Referências.

A linha de autoria deste tipo de artigos não deverá exceder quatro autores. Outros contributos poderão ser reconhecidos no final do texto, sob o parágrafo "Agradecimentos".

O texto não deve exceder as 1.000 palavras e 15 referências bibliográficas. Deve ser acompanhado de figuras ilustrativas. O número de tabelas/figuras não deve ser superior a 5.

Inclua um resumo não estruturado que não exceda 150 palavras, que sumarie o objectivo, pontos principais e conclusões do artigo.

**Imagens em Medicina (Imagem Médica):**

A Imagem em Medicina é um contributo importante da aprendizagem e da prática médica. Poderão ser aceites imagens clínicas, de imagiologia, histopatologia, cirurgia, etc. Podem ser enviadas até duas imagens por caso.

Deve incluir um título com um máximo de oito palavras e um texto com um máximo de 150 palavras onde se dê informação clínica relevante, incluindo um breve resumo do historial do doente, dados laboratoriais, terapêutica e condição actual. Não pode ter mais do que três autores e cinco referências bibliográficas. Não precisa de resumo.

Só são aceites fotografias originais, de alta qualidade, que não tenham sido submetidas a prévia publicação. Para informação sobre o envio de imagens digitais, consulte as «Normas técnicas para a submissão de figuras, tabelas ou fotografias».

***Guidelines* / Normas de orientação:**

As sociedades médicas, os colégios das especialidades, as entidades oficiais e / ou grupos de médicos que desejem publicar na Acta Médica Portuguesa recomendações de prática clínica, deverão contactar previamente o Conselho Editorial e submeter o texto completo e a versão para ser publicada. O Editor-Chefe poderá colocar como exigência a publicação exclusiva das recomendações na Acta Médica Portuguesa.

Poderá ser acordada a publicação de uma versão resumida na edição impressa cumulativamente à publicação da versão completa no *site* da Acta Médica Portuguesa.

**Cartas ao Editor:**

Devem constituir um comentário a um artigo da Acta Med Port ou uma pequena nota sobre um tema ou caso clínico. Não devem exceder as 400 palavras, nem conter mais de uma ilustração e ter um máximo de 5 referências bibliográficas. Não precisam de resumo.

Deve seguir a seguinte estrutura geral: Identificar o artigo (torna-se a referência 1); Dizer porque está a escrever; fornecer evidência (a partir da literatura ou a partir de uma

experiência pessoal) fornecer uma súmula; citar referências.

A(s) resposta(s) do(s) Autor(es) devem observar as mesmas características.

Uma Carta ao editor discutindo um artigo recente da Acta Med Port terá maior probabilidade de aceitação se for submetida quatro semanas após a publicação do artigo.

**Abreviaturas**: Não use abreviaturas ou acrónimos no título nem no resumo, e limite o seu uso no texto. O uso de acrónimos deve ser evitado, assim como o uso excessivo e desnecessário de abreviaturas. Se for imprescindível recorrer a abreviaturas não consagradas, devem ser definidas na primeira utilização, por extenso, logo seguido pela abreviatura entre parenteses. Não coloque pontos finais nas abreviaturas.

**Unidades de Medida:** As medidas de comprimento, altura, peso e volume devem ser expressas em unidades do sistema métrico (metro, quilograma ou litro) ou seus múltiplos decimais.

As temperaturas devem ser dadas em graus Celsius (ºC) e a pressão arterial em milímetros de mercúrio (mm Hg).

Para mais informação consulte a tabela de conversão "Units of Measure" no *website* da AMA Manual Style.

**Nomes de Medicamentos, Dispositivos ou outros Produtos:** Use o nome não comercial de medicamentos, dispositivos ou de outros produtos, a menos que o nome comercial seja essencial para a discussão.

## IMAGENS

Numere todas as imagens (figuras, gráficos, tabelas, fotografias, ilustrações) pela ordem de citação no texto.

Inclua um título/legenda para cada imagem (uma frase breve, de preferência com não mais do que 10 a 15 palavras).

A publicação de imagens a cores é gratuita.

No manuscrito, são aceitáveis os seguintes formatos: BMP, EPS, JPG, PDF e TIF, com 300 dpis de resolução, pelo menos 1200 *pixeis* de largura e altura proporcional.

As Tabelas/Figuras devem ser numeradas na ordem em que são citadas no texto e assinaladas em numeração árabe e com identificação, figura/tabela. Tabelas e figuras devem ter numeração árabe e legenda. Cada Figura e Tabela incluídas no trabalho têm de ser referidas no texto, da forma que passamos a exemplificar:

Estes são alguns exemplos de como uma resposta imunitária anormal pode estar na origem dos sintomas da doença de Behçet (Fig. 4).

Esta associa-se a outras duas lesões cutâneas (Tabela 1).

Figura: Quando referida no texto é abreviada para Fig., enquanto a palavra Tabela não é abreviada. Nas legendas ambas as palavras são escritas por extenso.

Figuras e tabelas serão numeradas com numeração árabe independentemente e na sequência em que são referidas no texto.

Exemplo: Fig. 1, Fig. 2, Tabela 1

**Legendas:** Após as referências bibliográficas, ainda no ficheiro de texto do manuscrito, deverá ser enviada legenda detalhada (sem abreviaturas) para cada imagem. A imagem tem que ser referenciada no texto e indicada a sua localização aproximada com o comentário "Inserir Figura nº 1… aqui".

**Tabelas:** É obrigatório o envio das tabelas a preto e branco no final do ficheiro. As tabelas devem ser elaboradas e submetidas em documento *word*, em formato de tabela simples (*simple grid*), sem utilização de tabuladores, nem modificações tipográficas. Todas as tabelas devem ser mencionadas no texto do artigo e numeradas pela ordem que surgem no texto. Indique a sua localização aproximada no corpo do texto com o comentário "Inserir Tabela nº 1… aqui". Neste caso os autores autorizam uma reorganização das tabelas caso seja necessário.

Quaisquer tabelas submetidas que sejam mais longas/largas do que duas páginas A4 serão publicadas como Apêndice ao artigo.

As tabelas devem ser acompanhadas da respectiva legenda/título, elaborada de forma sucinta e clara.

Legendas devem ser auto-explicativas (sem necessidade de recorrer ao texto) – é uma declaração descritiva.

Legenda/Título das Tabelas*:* Colocada por cima do corpo da tabela e justificada à esquerda. Tabelas são lidas de cima para baixo. Na parte inferior serão colocadas todas as notas informativas – notas de rodapé (abreviaturas, significado estatístico, etc.) As notas de rodapé para conteúdo que não caiba no título ou nas células de dados devem conter estes símbolos *, †, ‡, §, ||, ¶, **, ††, ‡‡, §§, ||||, ¶¶.

**Figuras**: Os ficheiros «figura» podem ser tantos quantas imagens tiver o artigo. Cada um destes elementos deverá ser submetido em ficheiro separado, obrigatoriamente em versão electrónica, pronto para publicação. As figuras (fotografias, desenhos e gráficos) não são aceites em ficheiros *word*.

Em formato TIF, JPG, BMP, EPS e PDF com 300 *dpis* de resolução, pelo menos 1200 *pixeis* de largura e altura proporcional.

As legendas têm que ser colocadas no ficheiro de texto do manuscrito.

Caso a figura esteja sujeita a direitos de autor, é responsabilidade dos autores do artigo adquirir esses direitos antes do envio do ficheiro à Acta Médica Portuguesa.

Legenda das Figuras: Colocada por baixo da figura, gráfico e justificada à esquerda. Gráficos e outras figuras são habitualmente lidos de baixo para cima.

Só são aceites imagens de doentes quando necessárias para a compreensão do artigo. Se for usada uma figura em que o doente seja identificável deve ser obtida e remetida à Acta Médica Portuguesa a devida autorização. Se a fotografia permitir de forma óbvia a identificação do doente, esta poderá não ser aceite. Em caso de dúvida, a decisão final será do Editor-Chefe.

- **Fotografias**: Em formato TIF, JPG, BMP e PDF com 300 *dpis* de resolução, pelo menos 1200 *pixeis* de largura e altura proporcional.
- **Desenhos e gráficos**: Os desenhos e gráficos devem ser enviados em formato vectorial (AI, EPS) ou em ficheiro bitmap com uma resolução mínima de 600 dpi. A fonte a utilizar em desenhos e gráficos será obrigatoriamente Arial.

As imagens devem ser apresentadas em ficheiros separados submetidos como documentos suplementares, em condições de reprodução, de acordo com a ordem em que são discutidas no texto. As imagens devem ser fornecidas independentemente do texto.

**AGRADECIMENTOS** (facultativo)

Devem vir após o texto, tendo como objectivo agradecer a todos os que contribuíram para o estudo mas não têm peso de autoria. Nesta secção é possível agradecer a todas as fontes de apoio, quer financeiro, quer tecnológico ou de consultoria, assim como contribuições individuais. Cada pessoa citada nesta secção de agradecimentos deve enviar uma carta autorizando a inclusão do seu nome.

**REFERÊNCIAS**

Os autores são responsáveis pela exactidão e rigor das suas referências e pela sua correcta citação no texto.

As referências bibliográficas devem ser citadas numericamente (algarismos árabes formatados sobrescritos) por ordem de entrada no texto e ser identificadas no texto com algarismos árabes. **Exemplo: "**Dimethylfumarate has also been a systemic therapeutic option in moderate to severe psoriasis since 1994[13] and in multiple sclerosis.[14]**"**

Se forem citados mais de duas referências em sequência, apenas a primeira e a última devem ser indicadas, sendo separadas por traço.[5-9]

Em caso de citação alternada, todas as referências devem ser digitadas, separadas por vírgula.[12,15,18]

As referências são alinhadas à esquerda.

Não deverão ser incluídos na lista de referências quaisquer artigos ainda em preparação ou observações não publicadas, comunicações pessoais, etc. Tais inclusões só são permitidas no corpo do manuscrito (ex: P. Andrade, comunicação pessoal).

As abreviaturas usadas na nomeação das revistas devem ser as utilizadas pelo National Library of Medicine (NLM) *Title Journals Abbreviations* http://www.ncbi.nlm.nih.gov/nlmcatalog/journals

**Notas**: Não indicar mês da publicação.

Nas referências com 6 ou menos Autores devem ser nomeados todos. Nas referências com 7 ou mais autores devem ser nomeados os 6 primeiros seguidos de "et al".

Seguem-se alguns exemplos de como devem constar os vários tipos de referências.

**Artigo:**

Apelido Iniciais do(s) Autor(es). Título do artigo. Título das revistas [abreviado]. Ano de publicação;Volume: páginas.

1. Com menos de 6 autores

Miguel C, Mediavilla MJ. Abordagem actual da gota. Acta Med Port. 2011;24:791-8.

2. Com mais de 6 autores

Norte A, Santos C, Gamboa F, Ferreira AJ, Marques A, Leite C, et al. Pneumonia Necrotizante: uma complicação rara. Acta Med Port. 2012;25:51-5.

**Monografia:**

Autor/Editor AA. Título: completo. Edição (se não for a primeira). Vol.(se for trabalho em vários volumes). Local de publicação: Editor comercial; ano.

1. Com Autores:

Moore, K. Essential Clinical Anatomy. 4th ed. Philadelphia: Wolters Kluwer Lippincott Williams & Wilkins; 2011.

2. Com editor:

Gilstrap LC 3rd, Cunningham FG, VanDorsten JP, editors. Operative obstetrics. 2nd ed. New York: McGraw-Hill; 2002.

**Capítulo de monografia:**

Meltzer PS, Kallioniemi A, Trent JM. Chromosome alterations in human solid tumors. In: Vogelstein B, Kinzler KW, editors. The genetic basis of human cancer. New York: McGraw-Hill; 2002. p. 93-113.

**Relatório Científico/Técnico:**

Lugg DJ. Physiological adaptation and health of an expedition in Antarctica: with comment on behavioural adaptation. Canberra: A.G.P.S.; 1977. Australian Government Department of Science, Antarctic Division. ANARE scientific reports. Series B(4), Medical science No. 0126

**Documento electrónico:**

1.CD-ROM

Anderson SC, Poulsen KB. Anderson's electronic atlas of hematology [CD-ROM]. Philadelphia: Lippincott Williams & Wilkins; 2002.

2. Monografia da Internet

Van Belle G, Fisher LD, Heagerty PJ, Lumley TS. Biostatistics: a methodology for the health sciences [e-book]. 2nd ed. Somerset: Wiley InterScience; 2003 [consultado 2005 Jun 30]. Disponível em: Wiley InterScience electronic collection

3. Homepage/Website

Cancer-Pain.org [homepage na Internet]. New York: Association of Cancer Online Resources, Inc.; c2000-01; [consultado 2002 Jul 9].Disponível em: http://www.cancer-pain.org/.

**PROVAS TIPOGRÁFICAS**

Serão da responsabilidade do Conselho Editorial, se os Autores não indicarem o contrário. Neste caso elas deverão ser feitas no prazo determinado pelo Conselho Editorial, em função das necessidades editoriais da Revista. Os autores receberão as provas para publicação em formato PDF para correcção e deverão devolvê-las num prazo de 48 horas.

## ERRATA E RETRACÇÕES

A Acta Médica Portuguesa publica alterações, emendas ou retracções a um artigo anteriormente publicado. Alterações posteriores à publicação assumirão a forma de errata.

## NOTA FINAL

Para um mais completo esclarecimento sobre este assunto aconselha-se a leitura do *Uniform Requirements for Manuscripts Submitted to Biomedical Journals* do International Commitee of Medical Journal Editors), disponível em http://www.ICMJE.org.

# Scale for the Assessment of Narrative Review Articles – SANRA

Please rate the quality of the narrative review article in question, using categories 0–2 on the following scale. For each aspect of quality, please choose the option which best fits your evaluation, using categories 0 and 2 freely to imply general low and high quality. These are not intended to imply the worst or best imaginable quality.

Nota: Para a UC de Dissertação/Projecto, todos os seis items devem ser cumpridos em nível 2.

## 1) Justification of the article's importance for the readership

The importance is not justified. — 0
The importance is alluded to, but not explicitly justified. — 1
The importance is explicitly justified. — 2

2

Página 1: "thus it is important for the physicians to be aware to the clinical aspects of this disorder, for early detection of the condition and consequently give an appropriate treatment to the patients."

## 2) Statement of concrete aims or formulation of questions

No aims or questions are formulated. — 0
Aims are formulated generally but not concretely or in terms of clear questions. — 1
One or more concrete aims or questions are formulated. — 2

2

Pagina 1: "and which aimed to clarify the clinical, diagnostic and therapeutic aspects of this clinical entity. "

## 3) Description of the literature search

The search strategy is not presented. — 0
The literature search is described briefly. — 1
The literature search is described in detail, including search terms and inclusion criteria. — 2

2

Página 1: "The search for bibliographic information was held on the Pubmed platform with the keywords: body dysmorphic disorder. Only articles that obeyed the following conditions were analyzed: written in English or Portuguese and with free full access; having analyzed 494 articles, between 1994 and 2020. There were included 41 articles that appeared to be relevant for the Body dysmorphic disorder, and which aimed to clarify the clinical, diagnostic and therapeutic aspects of this clinical entity. "

## 4) Referencing

Key statements are not supported by references. — 0
The referencing of key statements is inconsistent. — 1
Key statements are supported by references. — 2

2

Página 8: "Nearly all people with BDD perform compulsive behaviors which are repetitive, time-consuming (about half of BDD patients spend 3 or more hours per day engaged in them) and hard to control and resist. (5, 9, 19)"

## 5) Scientific reasoning

*(e.g., incorporation of appropriate evidence, such as RCTs in clinical medicine)*

The article's point is not based on appropriate arguments. — 0
Appropriate evidence is introduced selectively. — 1
Appropriate evidence is generally present. — 2

2

## 6) Appropriate presentation of data

*(e.g., absolute vs relative risk; effect sizes without confidence intervals)*

Data are presented inadequately. — 0
Data are often not presented in the most appropriate way. — 1
Relevant outcome data are generally presented appropriately. — 2

2

Ponto 5) e ponto 6):
Página 19: "The only data available are from a chart-review study in which 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). The average time to relapse in the SRI discontinuation group was approximately 75 days. "

**Sumscore** 12

**Fig. 1** SANRA - Scale