Body dysmorphic disorder: a treatment synthesis and consensus on behalf of the International College of Obsessive-Compulsive Spectrum Disorders (ICOCS) and the Obsessive Compulsive and Related Disorders Network (OCRN) of the European College of Neuropsychopharmacology (ECNP)

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Abstract

Body dysmorphic disorder (BDD) is characterised by a preoccupation with a perceived appearance flaw or flaws that are not observable to others. BDD is associated with distress and impairment of functioning. Psychiatric comorbidities, including depression, social anxiety and obsessive-compulsive disorder are common and impact treatment. Treatment should encompass psychoeducation, particularly addressing the dangers associated with cosmetic procedures, and may require high doses of SSRI* and protracted periods to establish full benefit. If there is an inadequate response to SSRIs, various adjunctive medications can be employed including atypical antipsychotics*, anxiolytics* and the anticonvulsant levetiracetam*. However, large scale randomised controlled trials are lacking and BDD is not an approved indication for these medications. Oxytocin* may have a potential role in treating BDD, but this requires further exploration. Cognitive-behavioural therapy has good evidence for efficacy for BDD, and on-line and telephone-assisted forms of therapy are showing promise. CBT for BDD should be customised to address such issues as mirror use, perturbations of gaze and misinterpretation of others' emotions, as well as overvalued ideas about how others view the individual.

Key words: body dysmorphic disorder, obsessive compulsive disorder, serotonin reuptake inhibitors, antipsychotics, cognitive behaviour therapy.

Introduction

Body dysmorphic disorder (BDD) is a recognised psychiatric disorder characterised by a preoccupation that some aspect of the sufferer's physical appearance is ugly or perception of disfigurement, to the extent that they experience significant distress and/or disability. Frequently, there is no obvious abnormality in the individual's appearance, but sometimes there is a minor flaw which is not immediately obvious to others and, in both cases, the individual's response is excessive (Phillips et al, 1993; Castle et al, 2006; APA, 2013). DSM-5 has included BDD in the chapter on Obsessive Compulsive and Related Disorders (OCRDs; APA, 2013). The upcoming 11th edition of the International Classification of Diseases (ICD-11) takes a rather broader and more culturally informed approach to BDD but also plans to include it within the OCRDs grouping (https://icd.who.int/browse11/l-m/en#/http%3a%2f%2fid.who.int%2ficd%2fentity%2f731724655; Veale and Matsunaga, 2014).

The community point prevalence of BDD in nationwide studies has been estimated at around 1.7-2.9% (Koran et al, 2008; Rief et al, 2006; Scheiber et al, 2015; Veale et al, 2016). Many people with the disorder never come to the attention of health professionals, in part because of the shame they often feel about their problem. Indeed, when many people with BDD look for help, owing to limited insight into their problems, they access clinicians other than psychiatrists such as dermatologists, cosmetic surgeons, etc (see below). BDD tends to have a chronic persistent course unless adequately treated (Veale et al, 1996; Phillips et al, 2013) and

can be extremely debilitating. Suicidal ideation is common and suicide rates are amongst the highest of any psychiatric disorder. Indeed, a meta-analysis of 17 studies reported an odds ratio for suicidality in BDD (relative to the general population) of 3.63 (95% CI 2.62-4.63), BDD being associated with significantly higher levels of suicidality than other psychiatric disorders characterized by high risk for suicidal thoughts and acts (Angelakis et al, 2016). People with BDD often have associated social anxiety disorder and depression (Phillips et al, 2005). There is also a substantial overlap in symptoms with obsessive compulsive disorder (OCD). Nevertheless, there are some important differences between BDD and OCD in terms of symptoms, neurobiology, treatment response, and other characteristics (Malcolm et al, 2018; Simberlund and Hollander, 2017).

This article provides a summary of treatment strategies for BDD, including emerging novel approaches. Similarities and differences with OCD are emphasised. In spite of its substantial prevalence and morbidity, there is no drug officially approved for the treatment of BDD and the response to the different treatment strategies that have been tested is limited. While different evidence-based clinical guidelines for managing OCD have been published (reviewed in Fineberg et al 2020) there is limited available guidance for the treatment of BDD.

The International College of Obsessive-Compulsive Spectrum Disorders (www.ICOCS.org) is a global network of expert clinicians, researchers and “experts by experience of OCD”, whose principal objective is to support and stimulate the study and treatment of OC spectrum disorders. The Obsessive Compulsive and Related

Disorders Network (OCRN) of the ECNP Networks brings together researchers with different expertise to foster successful collaboration and sharing of ideas, discoveries and practices in translational neuroscience. In recognition of the need for updated clinical guidance on the treatment of BDD, both organizations, the ICOCS and OCRN, have developed this treatment synthesis, based on expert consensus. Agreement was reached on the key issues to be covered and the authors of each section were chosen based on their expertise in that area. Briefly, the recent advances in the field have been selected by a range of experts who have considered those of most relevance to the management of BDD. An initial draft was prepared, based on a literature review, and circulated first among the authors and iterative edits were incorporated. Drug treatments mentioned along the text have been selected according to the evidence from clinical and translational neuroscience, but they have been marked with an asterisk (*) to underline that no drug is labelled for BDD.

Initial treatment considerations

A number of important initial considerations should be noted when treating patients with BDD. First, unlike in OCD, where 'insight' is usually retained, people with BDD have a high likelihood of holding their beliefs about their appearance with delusional conviction (Toh et al., 2017b). BDD's delusional variant used to be considered a form of BDD which required additional DSM coding with delusional disorder; this has been abandoned in DSM-5, and an 'insight specifier' has been adopted (APA, 2013), such that BDD may be coded as being characterized by good or fair insight, poor insight, or absent insight (delusional beliefs). ICD-11 does not propose any additional coding for BDD patients whose appearance beliefs are held with delusional conviction but

condensed to two distinct levels of insight with fair to good insight and with poor to absent insight, for all OCRDs. (Veale and Matsunaga, 2014). Many in the field pragmatically consider 'delusional' BDD simply to reflect the severe end of the BDD spectrum (Labuschagne et al, 2010; Rossell et al, 2020); importantly, it is recognised that antipsychotic agents do not appear to be effective as monotherapy even if BDD is 'delusional' (Mancuso et al, 2010).

Because insight in people with BDD is usually absent or poor (that is, they are usually mostly or completely convinced that they look abnormal, ugly, or deformed), they may be reluctant to participate in mental health treatment; many prefer to receive cosmetic treatment, which is not recommended (see below). Thus, motivational strategies and more extensive psychoeducation may be required than for many other psychiatric disorders in order to engage and retain these patients in treatment (Veale et al, 2017).

Second, it is common for people with BDD to have a number of psychiatric comorbidities (although this is also the norm for most other psychiatric disorders if patients are systematically assessed for comorbidity). For example, in the study of Phillips and colleagues (1994), lifetime rates of psychiatric comorbidity amongst 100 BDD patients were 80% for major depressive disorder, 37% for social phobia and 34% for OCD. A more recent study of 293 BDD patients confirms these associations and also emphasises the extent of triple or even quadruple comorbidities: for example, only 2% had social anxiety disorder alone, whilst 32% had social anxiety disorder plus depression, and 14% had these two comorbidities plus OCD (Gunstad and Phillips,

2003). It is important to note that the studies of Phillips and colleagues (1994) and of Gunstad and Phillips (2003) consisted of samples who were seeking or receiving treatment, and the rates of comorbidities would be expected to be lower in community samples (Toh et al, 2017c) or non-specialist settings. In any event, the presence of such comorbidities requires therapeutic interventions to be sufficiently nuanced to adjust the treatment accordingly. For cognitive-behavioural therapy (CBT), usually a hierarchical approach is appropriate, with the most severe and disabling condition being prioritised (i.e., treated first) or explicitly worked into the treatment framework (Wilhem et al, 2013).

In some instances, the treatments for BDD are also useful for comorbid conditions. For instance, serotonergic antidepressants can be effective for the core symptoms of BDD but can also help address multiple other comorbidities, such as depressive and social anxiety comorbidity as well as comorbid OCD symptoms. Similarly, CBT for BDD is also associated with improvement in certain associated symptoms such as depression (Veale et al, 2014; Wilhelm et al., 2014; 2016). Other comorbidities are more difficult, notably bipolar disorder, where high doses of SRIs (the first line pharmacotherapy for BDD) can destabilise mood; however, patients who are first adequately treated with one or more mood stabilizers can be treated with SRIs with less difficulty. Comorbid anorexia nervosa (AN) is particularly challenging in patients with BDD (Phillipou et al, 2019), with the emphasis usually being initially on the disordered eating and ensuring medical stability. Suicidal ideation and acts are common in people with BDD, and careful attention needs to be given to assessment and appropriate interventions. Substance use disorders are also common in people with BDD, which are often an

attempt to cope with BDD-related distress. Both the substance use disorder and BDD need to be a focus of treatment.

Skin picking and hair pulling require a particular approach, in that people with BDD often engage in these activities to try to fix their perceived skin or hair 'defects' by removing skin irregularities, blemishes, or disliked hairs. This differs from the more impulsive picking/pulling seen in excoriation (skin-picking) disorder or trichotillomania, which are not triggered by thoughts that the skin or hair look abnormal or ugly (Veale and Matsunaga, 2014). Thus, the therapeutic strategies for skin picking or hair pulling in people with BDD include interventions for excoriation (skin-picking) disorder and trichotillomania, such as habit reversal, plus additional BDD-specific strategies.

A third issue is that BDD has a particular variant, where the focus of concern is body habitus, the individual 'seeing' their body composition as puny or slight, when it is actually normal or even very muscular, and seeking to achieve a muscular ideal (APA, 2013). This form of BDD (muscle dysmorphia) largely occurs in males and encompasses a number of behaviours not usually evident in people with BDD, notably excessive muscle-enhancing exercises; specific low-fat, high protein diets; and the use of supplements and potentially dangerous anabolic steroids, testosterone, and medications such as thyroid hormone, insulin, and oestrogen modulators (which may be illicitly obtained) (Tod et al, 2016; Blomely et al, 2018). These issues add a further layer of complexity to BDD management and requires attention to the habitual exercise and dietary regimens as well as advice about and treatment for abuse of muscle-enhancing agents and other substances if abused. It should be noted that

unlike the DSM 5 working group, the ICD-11 working group did not consider muscle dysmorphia to be 'sufficiently different' from other manifestations of BDD to warrant an additional specifier (Veale and Matsunaga, 2014).

There appear to be some differences in the presentation of BDD and the body parts which are focused on in men and women, although BDD in men and women has many more similarities than differences. In women BDD is more likely to be comorbid with an eating disorder, whereas in men BDD is more likely to be comorbid with a substance use disorder, which has implications for treatment (Tyagi et al, 2012; Grant and Phillips, 2004; Gazzarrini and Perugi, 2017)

A final consideration pertains to how we define response and remission in treatment trials of BDD. Most clinical trials use the well validated Yale-Brown Obsessive Compulsive Scale modified for BDD (BDD-YBOCS) (Phillips et al, 1997). Recently, Fernandez de la Cruz and colleagues (2019) pooled data from three CBT trials for BDD conducted across three countries (combined n = 153), to evaluate the way in which BDD-YBOCS performed in terms of predicting either response or remission. A reduction in scores of >= 30% on the BDD-YBOCS predicted response against the Clinical Global Impression Scale (CGI) with a sensitivity of 0.89 and specificity of 0.91, whilst partial or full remission was best predicted by a BDD-YBOCS score of =< 16 (sensitivity 0.85 and specificity 0.99). These cut-offs should thus be seen as the bench-marks for future research, but some published studies used different cut-offs and need to be interpreted in this light.

General treatment issues

One of the key issues in treating people with BDD is to ensure that it is recognised. As people with BDD often seek redress through cosmetic means (see below), screening for BDD within such settings seems appropriate. It has also been shown that most physicians have a low level of recognition of BDD, and even in psychiatric settings it is often missed. Sensitive questioning and the use of validated screening tools can enhance recognition and thus engagement in treatment. Such tools include:

- The Dysmorphic Concern Questionnaire (Oosthuizen et al, 1998): This is a seven item scale with each item scored from 0 ('not at all') to 3 ('much more than most people'): the minimum score is 0 and maximum is 21; a cut-off for BDD is 9 (Mancuso et al, 2010).
- The Body Dysmorphic Disorder Questionnaire (BDD-Q) (Phillips, 1996; 2005): This simple, brief self-report questionnaire screens for the presence of BDD. The BDDQ has excellent sensitivity and specificity for DSM-IV BDD in mental health and cosmetic treatment settings. It is also suitable for screening for DSM-5-defined BDD.
- Body Dysmorphic Disorder Questionnaire-Dermatology Version (BDDQ—Dermatology Version) (Dufresne et al, 2001): This simple, brief self-report questionnaire screens for the presence of BDD. It is very similar to the BDDQ (described above); some of the BDDQ's dichotomized yes/no questions are instead scored on 5-point Likert scales. This version of the BDD-Q also has strong psychometric properties.
- The Cosmetic Procedure Screening Questionnaire for BDD (COPS; Veale et al, 2012): Nine items cover essential features of BDD, each rated from 0 ('least

impaired') to 8 ('most impaired'). It was developed to screen for BDD in people seeking cosmetic procedures, with a score of 40 or over being and indicator that the individual requires a full assessment for BDD.

As with all psychiatric disorders, effective and thorough psychoeducation is an important core feature of a therapeutic framework for BDD. There is a number of useful self-help books, including "The Broken Mirror: Understanding and Treating Body Dysmorphic Disorder" (Phillips, 1996 and 2005); "Feeling Good About the Way You Look: A program for overcoming body image problems" (Wilhelm, 2006); and "Overcoming Body Image Problems (including Body Dysmorphic Disorder)" (Veale et al, 2009). Online interventions are also being developed and evaluated, as discussed below.

A very important consideration in BDD is that most affected people see their problem as physical rather than psychological or psychiatric, and thus seek cosmetic redress. The prevalence of BDD amongst people attending cosmetic specialists is high: rates of 12.3% have been reported in general cosmetic surgery settings, 20.1% in rhinoplasty patients, 5.2% in orthodontics/cosmetic dentistry and 9.2% in cosmetic dermatology (Veale et al, 2016). The majority of BDD patients has sought or received cosmetic treatment for BDD appearance concerns. A broad array of clinicians may be seen, such as dermatologists, plastic surgeons, maxillo-facial surgeons, and trichologists. In the largest study of this topic (n=250 adults with BDD), 76% of patients had sought cosmetic treatment (surgical, dermatologic, and other types of cosmetic interventions) in an attempt to fix their perceived appearance concerns, and 66% had actually

received aesthetic treatments, with 72% having sought and 60% having received treatment from either a dermatologist or a cosmetic surgeon (Phillips et al, 2001). Findings were nearly identical in a different and more broadly ascertained sample of 200 individuals with BDD (Crerand et al, 2005). There are also reports of BDD patients performing "DIY (do it yourself) surgery" when they cannot persuade cosmetic specialists to undertake their desired procedure (Veale, 2000, Phillips, 1996).

It is important for mental health clinicians dealing with people with BDD to have a clear and candid discussion about these matters and provide warnings about the very low likelihood of cosmetic interventions helping the BDD symptoms in the longer term: some people with BDD experience some brief 'relief' after a cosmetic procedure but often become dissatisfied with the outcome and/or seek further procedures for the same or another physical 'defect' (Crerand et al, 2005; Phillips et al, 2001). There may be some exceptions to this rule. For example, Veale and colleagues (2014) found that labiaplasty may carry a good psychosocial outcome even in patients with BDD; the same might occur for breast augmentation. However, the American College of Obstetrics and Gynaecology has stipulated caution needs to be exercised in any such procedures being undertaken in people with BDD (ACOG, 2017; 2020). More positive views of surgical outcomes with BDD are controversial, and BDD is widely considered a contraindication for cosmetic surgery. Facial procedures such as rhinoplasty are much more likely to have complex psychological outcomes, even in people without BDD (Honigman et al, 2003).

The study of Tignol et al (2007) is particularly instructive. These authors performed a 5-year follow up of 24 of 30 individuals with 'minimal defect in appearance' requesting cosmetic surgery (12 were diagnosed with BDD at baseline, of whom 10 were followed up). Fifteen individuals underwent cosmetic procedures. Self-reported satisfaction with the cosmetic outcome was in general high. However, at follow-up, six of the seven BDD patients who underwent cosmetic surgery still met criteria for BDD and carried higher levels of disability and psychiatric comorbidity than those without a baseline BDD diagnosis. Also, three non-BDD individuals 'developed' BDD over the follow-up period, reflecting the fact that the focus of concern may switch to a different body part following cosmetic surgery.

Thus, it is recommended to try to reach an agreement with the patient not to pursue such procedures. At the very least, they should be delayed until psychological and pharmacological approaches can be given an opportunity to show efficacy (6-12 months). Where appropriate, liaison with the cosmetic specialist can be fruitful, in discussing all potential risks and ensuring psychiatric assessment and treatment are effected.

Pharmacotherapy

Like OCD, the mainstay pharmacological therapies for BDD are serotonin reuptake inhibitors (SRIs), i.e., selective serotonin reuptake inhibitors* (SSRIs) and clomipramine*. Predominantly noradrenergic antidepressants* have not proven effective for BDD. For example, Hollander et al (1999) performed a randomised controlled cross-over study of 29 adult patients with BDD comparing clomipramine

(predominantly SRI) with desipramine (predominantly noradrenaline reuptake inhibition (NRI)) and showed superiority for the former agent in ameliorating BDD symptoms (65% response (defined as >= 25% reduction in BDD-YBOCS) with clomipramine vs. 35% with desipramine). Importantly the effects were independent of mood, underlining the primacy of serotonergic perturbations in BDD. This selective efficacy of SRI's vs NRI's in BDD is similar to that found in OCD (Goodman et al, 1990), and is one criterion that is supportive of inclusion of BDD as an OCRD in classification manuals.

Randomised placebo-controlled controlled trials in BDD have been reviewed by Phillipou et al (2016). Three trials met the inclusion criteria of being empirical research specifically of BDD patients, published in peer-reviewed journals in English, employing a controlled randomised design and reporting BDD symptoms pre- and post-intervention. One of these studies is the aforementioned cross-over trial of Hollander et al (1999). The other two were both from the same lab and had some overlap in terms of participants. The earlier of these two studies (Phillips et al, 2002) randomised 67 BDD adult patients to an initial dose of 20mg fluoxetine* daily, or placebo. Fluoxetine dose could be increased every 10 days, up to a maximum of 80mg daily. Three patients in the fluoxetine arm and five in the placebo arm withdrew prior to study completion. Using a BDD-YBOCS reduction of >=30% to define response, 53% of the fluoxetine group met response criteria, compared with 18% on placebo, a statistically significant difference. Response of BDD was independent of response of major depressive disorder, OCD or a personality disorder. Importantly, fluoxetine had a protective effect against suicidality worsening (Phillips and Kelly, 2009). Phillips et al

(2005) also utilized a placebo controlled randomized design to study augmentation with a typical neuroleptic* in BDD. The authors included 19 participants from the earlier study, as well as a further nine additional patients (i.e. total n=28), all of whom had received fluoxetine for at least 12 weeks, at a dose of 80mg per day, if tolerated: none had adequately responded to fluoxetine. Eleven of these patients were randomised to pimozide* (initially 1mg daily, increasing step wise to a maximum of 8mg daily) and 17 to placebo, over 8 weeks, whilst remaining on a fixed fluoxetine dose schedule. There was substantial drop-out, with only six pimozide patients and 11 placebo patients completing the study. There was no advantage seen for pimozide over placebo for BDD symptom reduction; although the sample size was small the effect size was also small. (After this study was completed, the combination of pimozide and SRIs became contraindicated in the U.S. due to concerns about the potential for QTc interval prolongation.)

Since the publication of the Phillipou et al's (2016) review, an additional study compared the efficacy of continuation pharmacotherapy in people with BDD who initially responded to medication. Phillips et al (2016) treated 100 BDD adult patients with open-label escitalopram* (mean dose at the end of 14 weeks was 26.2 mg/day (SD 7.2)), whereafter responders (n=58) were randomised to continuation pharmacotherapy or placebo, and followed for a further six months. The continuation phase showed that relapse was significantly reduced in the active treatment group (18% vs. 40% for placebo); and time to relapse was significantly delayed (hazard ratio 2.72 (95%CI 1.01-8.57)). Importantly, additional improvement in BDD symptoms was noted in over a third of participants in the escitalopram continuation phase,

supporting clinical observations that benefits from SRIs can continue to accrue over extended time periods.

Despite the limited RCTs of SRIs in BDD, with use of fluoxetine*, clomipramine*, or escitalopram*, it can reasonably be assumed that other SRIs* are also effective for BDD, as is the case for other psychiatric disorders. In clinical practice they are often used largely interchangeably, depending upon efficacy, tolerability, and treatment history. Furthermore, in a chart-review study of 90 patients who had received an SRI in a clinical practice, response rates were similar for each type of SRI (Phillips et al, 2001).

A number of these agents has been investigated in open trials or reported as case series. Phillips et al (2003) found that citalopram* (mean dose 51.3 ± 16.9 mg/day) improved BDD symptoms in over 80% of a group of 15 patients, as well as quality of life, over 12 weeks. Escitalopram* (mean dose 28 +/- 6.5mg/day) showed similar favourable outcomes on BDD symptoms (73% response rate) in an open trial of 15 patients over 12 weeks (Phillips, 2006), as did fluvoxamine* (mean dose 238mg/day +/- 85mg per day) in 30 patients treated for a mean of 6.1 (+/- 3.7) weeks (Phillips et al, 1998). In all of the above studies, participants with the delusional variant of BDD (under DSM-IV nosology: see above) showed similar response rates to those whose beliefs were not delusional; most studies also found that insight significantly improved with treatment.

Like OCD, doses of SRIs employed in BDD are often higher than those usually used for depression. Doses of 300mg or 400mg a day of sertraline* equivalent may be required

for efficacy. Maximum doses for the various SSRIs are: sertraline 400 mg/day, fluoxetine* 120 mg/day, citalopram* 40 mg/day; escitalopram* 60 mg/day (with an ECG recommended at doses exceeding 20 mg/day), fluvoxamine* 450 mg/day; and paroxetine* 100 mg/day. These doses are above maximum dosages recommended by most countries' regulatory agencies, and patients need to be made aware of this. However, they are identical to maximum doses for OCD in the American Psychiatric Association's Practice Guideline for OCD (2007) (with the exception of citalopram, as the maximum dose has since been lowered). In addition, the SSRIs have a high therapeutic index, and the higher doses are usually safe and well tolerated.

There is a very low risk of serotonin syndrome and some concerns regarding prolongation of the QTc interval, albeit initial warnings about citalopram in this regard have not been supported by subsequent scrutiny of the relevant data (Hutton et al, 2016). Having said this, it would be sensible to obtain an ECG when there is any history of cardiac conduction problems and when using escitalopram* at doses exceeding 20mg/day and citalopram*, which has a black box warning regarding QTc prolongation at daily doses above 40 mg. Some experts would check an ECG for any SSRI when the dose being used is above the maximum recommended by regulatory agencies. However, clinical practices in this regard vary somewhat among pharmacotherapy experts. For example, a participant of the present consensus (KAP) does not obtain ECGs solely when using doses above the regulatory maximum (except for escitalopram at 40 mg/day or higher) and does not use citalopram for BDD because the US regulatory maximum dose of 40 mg/day is firmer than for the other SSRIs and is often too low to treat BDD effectively. Also, as in OCD, effects of medication might take

some weeks to accrue, hence a step-wise dosing schedule is suggested, with 2-3 weekly increases dependent upon efficacy and tolerability. A slower schedule with lower total dose is recommended in youth, those with sensitivity to medication side effects, the elderly, and people with physical comorbidities, such as hepatic dysfunction and cardiac conduction problems.

Whether clomipramine* has any added benefit for BDD over the SSRIs remains unstudied, but some patients do respond well to it: the side effects, notably histaminergic and muscarinic anticholinergic effects such as weight gain, sedation, dry mouth and orthostatic hypotension can limit dose. Daily doses of up to 250mg have been used, albeit some patients respond to lower doses or cannot tolerate the higher doses. Monitoring of ECGs and blood levels is recommended, with dosing guided by blood levels. A dose of 250 mg/day should not be exceeded due to this medication's low therapeutic index. We know of only a single published study of intravenous clomipramine in BDD. In that study (Pallanti and Koran, 1996), two patients meeting DSM-IV criteria for BDD (delusional variant) were administered pulse-loaded intravenous clomipramine (150 mg on day 1, 200 mg on day 2). Both patients showed around a 30% reduction in BBD-YBOCS scores 4.5 days after intravenous dosing, and improved further over the ensuing two months on oral medication, with marked improvement in social functioning. These authors suggest that pulse-loaded, intravenous clomipramine may have benefit for rapid symptom reduction in some people with BDD.

There are earlier case reports of patients with BDD responding to other tricyclic antidepressants*, including doxepin* (200mg daily) (Brotman and Jenike, 1984) as well as the monoamine oxidase inhibitor (MAOI) tranylcypromine* (30mg daily) (Jenike, 1984). However, available data (case series) indicate that these medications are unlikely to be efficacious for BDD (Phillips et al, 1993; 1994) and thus their use is not routinely recommended. However, MAOIs* have been found to be useful for people with severe social anxiety disorder (Menkes et al, 2016), raising the question of whether they might be useful in BDD patients with pervasive and severe social anxiety, but this question remains to be empirically tested, and these medications are complicated to prescribe and can be difficult to tolerate.

In BDD treatment, a 12 to 14 week trial of an SRI* is recommended, with at least 3-4 of these weeks at the maximum dose recommended by regulatory agencies, in order to determine whether the medication is helpful enough to continue it. A longer trial is needed if slower titration is used. Duration of therapy is usually guided by clinical response and any side effects experienced. With SSRIs, sexual side effects may occur and can lead to discontinuation (Read and Williams, 2018); however, sexual functioning is often impaired due to BDD or comorbid depression and may therefore improve with SRI treatment. In addition, sexual side effects may resolve with time (up to 6 months or so), and, if not, treatment for more problematic or persistent sexual dysfunction may be effective.

In the only published long-term double-blind randomised discontinuation study in the field (detailed above), Phillips et al (2016) found benefits with continued escitalopram*

over a 6-month period beyond the acute treatment phase of 14 weeks. Studies over longer time period are required, but clinical experience suggests continuing with the dose which was initially effective for at least several years and then, if indicated, trying a gradual staged reduction with careful monitoring for recurrence of symptoms. However, BDD is a chronic condition, and patients often remain on their medication indefinitely, similarly to OCD patients. For those with multiple hospitalizations and/or suicide attempts, indefinite treatment with medication is usually recommended.

Adjunctive pharmacologic agents:

As with OCD, many people with BDD do not experience full resolution of symptoms with SSRIs*. In this scenario, usual clinical practice would be to increase the dose of the SSRI above regulatory maximum doses (except for clomipramine* and citalopram*), to the doses noted previously, which not uncommonly improves symptoms. Alternatively, the clinician can try a different SSRI or clomipramine. If such measures fail, a number of adjunctive medications can justifiably be used. Of course, due attention needs to be paid to drug-drug interactions and the potential for cumulative side effects.

Antipsychotics:

Perhaps informed by evidence for the efficacy of antipsychotics* as adjuncts to SRIs in OCD (Kim et al, 2018; Brakoulias and Stockings, 2019), practitioners have prescribed these agents for BDD. The assumption that efficacy in OCD necessarily translates to efficacy in BDD is not necessarily valid, as the underlying neurobiology of the conditions differs from each other in certain respects (Rossell et al, 2015; Grace et al,

2017; Malcolm et al., 2018). In part, the use of antipsychotics in BDD is also driven by an implicit assumption that, because BDD patients are sometimes 'psychotic' in the sense that they hold their beliefs with delusional conviction, they require an antipsychotic. Nevertheless, such an assumption is again not supported by clinical studies, which show that even 'psychotic' BDD can respond to SRIs* alone (Phillips et al, 2017). Also, BDD may be comorbid with bipolar disorder in clinical practice, in which case mood stabilizing antipsychotics are often required as a first step in the treatment hierarchy prior to using SSRIs to treat the BDD.

Very few studies have formally evaluated the use of antipsychotics* as augmenting agents for BDD, and most have actually been negative. The only published RCT is the small add-on study using pimozide*, discussed above (Phillips et al, 2005). Phillips (2005) also reported an open-label study (n=6) with olanzapine* (mean dose mean dose 4.6 mg/day (SD3.3)) as an adjunct to fluoxetine* (mean dose 70mg), which did not show any benefits in terms of BDD symptoms for four participants and minimal benefit in a further two. Despite these negative findings, the use of antipsychotics in BDD patients, who have failed to respond to SRIs, is common in specialist practice (Rashid et al, 2014). Case reports have described potential benefit from the addition of olanzapine* (Grant, 2001; Nakaaki et al, 2008), quetiapine* (Mancuso et al, 2010) and risperidone* (Goulia et al, 2011) to an SRI, in some BDD patients. Case reports, of course, suffer from reporting and publication bias, and there is little clarity about which BDD patients are particularly likely to respond to which of these agents. As always, potential side effects need to be weighed against potential benefits.

The dopamine D2 partial agonist aripiprazole* has been used in clinical practice as an SRI* augmenting agent in BDD. Again, no open-label or RCTs have been conducted, but the use in BDD mirrors the use of aripiprazole in OCD as well as in depression (Veale et al, 2014). Beneficial effects can be seen for both BDD and mood. Doses usually range from 2-10mg per day. In the only published report of which we are aware, that specifically used aripiprazole in BDD, Uzun and Ozedemir (2010) successfully treated a 43 year old woman with BDD with the addition of 10mg aripiprazole to 400mg fluvoxamine. In the experience of some of this paper's authors, this medication may be quite effective as an SRI augmentation agent. We are aware of no published studies of the newer dopamine D2 partial agonists (brexpiprazole*, cariprazine*) in BDD. It is important to keep in mind that the antipsychotics are a large class of medications, with varying efficacy for different symptoms (Huhn et al, 2019). In the authors' experience and opinion, second generation antipsychotics are more likely than first generation to be efficacious for BDD and accompanying depression. Research on this important issue is greatly needed.

Other pharmacologic agents

Many other augmenting strategies have been employed in specialist practice to try to help people with BDD, but none have been subject to robust research evaluation. The field is again guided to a large extent by the experience of augmenting agents in OCD. We would advocate for an approach to augmentation that responds to the particular profile of the individual and which targets specific symptom sets. For example, patients with features of generalised anxiety might benefit from buspirone*, clonazepam* or pregabalin*. All of these agents are off-label for BDD and clonazepam

has the potential for habituation and addiction. Phillips et al (1996) showed in a small open trial that buspirone (mean dose 48.3mg/day) was beneficial as an add-on to fluoxetine* or clomipramine* in 46% of 13 BDD patients, but we are aware of no specific published trials using clonazepam or pregabalin in BDD. There is some evidence that the supplement N-Acetylcysteine* (NAC) may be efficacious for OCD, and clinical experience suggests that it can be helpful as an SRI adjunct in BDD. Also intranasal esketamine* has been used in clinical practice with some benefit for BDD comorbid with resistant depression (unpublished data).

A small open label trial (n=17) showed significant improvement with the anticonvulsant levetiracetam* (500-2000mg per day) in patients with BDD (Phillips et al, 2009). It can be used either as an adjunct to an SSRI or as monotherapy. Another small open-label trial (n=17) with the serotonin-norepinephrine reuptake inhibitor venlafaxine* similarly led to significant improvement in BDD symptoms (Allen et al, 2008). However, due to the lack of RCTs and small sample sizes, these medications should be used only when optimized SRI trials have not been effective.

Medications under investigation for BDD have been recently reviewed by Dong et al (2019). These agents include silymarin* (an extract of milk thistle) and memantine*, based on their efficacy for some patients with OCD. Results of these studies have not yet been reported but will clearly be of interest in terms of new therapeutic modalities for BDD and can also inform understandings of underlying neurobiology.

Psychological approaches:

An early review and meta-analysis (Williams et al, 2005) of treatments for BDD included case series as well as RCTs. There were nine studies which employed psychological therapies, but there was substantial heterogeneity. The results suggested similar efficacy for exposure/response prevention (ERP) and CBT in BDD, with effect sizes of 1.43 and 1.78, respectively. The more recent systematic review of Phillipou et al (2016) found six RCTs of psychological interventions in BDD, with a total of 165 participants (range 10-53) reaching the predefined study end-point (ranging from 8 to 24 weeks): drop-out rates ranged from 0% to 33.3%. All studies were arguably underpowered and many did not define a specific response criterion. Most had wait list control conditions (Rosen et al, 1995; Veale et al, 1996; Wilhelm et al, 2014; Rabiei et al, 2012), whilst in the study of McKay et al (1997) there was no treatment offered to controls. Lack of active controls does not allow determination of the effect of study participation parameters, such as clinician contact time and attention. A notable exception is one of the studies by Veale and colleagues (2014), which employed an anxiety management comparator.

The content of the therapy across these studies was fairly heterogeneous. Most used some variant of CBT but the additional content (e.g. the study of Wilhelm et al (2014) included advanced cognitive restructuring and additional optional modules (for patients who had relevant symptoms) addressing skin picking, muscularity concerns (muscle dysmorphia), cosmetic treatment, and mood management), and format, duration and time between sessions were variable. Rabiei et al (2012) employed a metacognitive approach, while McKay et al (1997) essentially used exposure/response prevention strategies. Furthermore, not all samples were representative of BDD

patients in the community: for example, Rosen et al (1995) included only females, most of whom had predominantly weight and shape concerns.

Given the heterogeneity and major methodological differences across studies, Phillipou et al (2016) did not believe the data met criteria for meta-analysis. Subsequently, Harrison et al (2016) performed a systematic review and meta-analysis of randomised controlled studies of CBT in BDD. They included seven studies (total n=299) and reported CBT to be superior to wait list or psychological placebo in reducing BDD symptoms (seven studies: delta -1.22; 95%CI -1.66 to - 0.79) as well as depression (five studies: delta -0.49; 95% CI -0.76 to -0.22). They also found four studies specifically addressing BDD-related insight, with an overall beneficial effect of CBT (delta – 0.56; 95%CI -0.93 to -0.19). 'Delusional' BDD responded with a similar effect size to 'non-delusional' in most CBT trials.

As pointed out by Mennin (2019), many published psychological trials in BDD have substantial methodological constraints, including issues with randomisation, blinding, use of active vs. 'placebo' comparators, and lack of manualisation and specification of the intervention. It is also the case that many of these studies were small, and thus were statistically underpowered. Many of the pharmacological treatment trials in BDD had some of the same limitations, as outlined above. On the other hand, many of the CBT trials were earlier proof of concept studies, and trials using a wait-list control group are generally warranted before embarking on large, expensive, and labour-intensive controlled trials. Some of these studies also had particular strengths, such as appropriate randomization and use of manualized treatment.

In this context, a recent 24-week RCT (Wilhelm et al, 2019) comparing supportive psychotherapy (SPT) with CBT for BDD (CBT-BDD) addresses many of these methodological constraints. The study was adequately powered (n=120); the intervention was manualised and specifically developed for BDD; randomisation and blinding were of high quality; and analyses employed intention-to-treat. Overall outcomes were excellent, with 84% of the CBT-BDD participants meeting response criteria; most maintained gains at 6-month follow-up. However, the difference in effectiveness between CBT-BDD and SPT was site specific: at one site, no significant difference was detected, whereas at the other site, CBT-BDD led to significantly greater reductions in BDD, compared with SPT. One site showed a response rate to SPT of 46%, and the other 64%: in fact, analysis of data from the second site did not show statistical separation from CBT-BDD. The paper authors suggest the high response to SPT at the second site might reflect the fact that that site offers predoctoral and postdoctoral training in supportive or integrative psychotherapy. Thus, supportive psychotherapy at that site is likely superior to that offered in other academic medical or community settings, including the other site in this study. In addition, because SPT primarily emphasizes common factors (rather than specific skills, as CBT does), therapist factors may have had a greater effect on treatment, leading to more variable outcomes across the two sites. Of course this was not just 'any' SPT, as it was being delivered as part of a treatment trial, at a site that specializes in both BDD and SPT, and with considerable patient contact time. But STP has been shown to be effective for depression, albeit with a small effect size (Cuijpers et al, 2012). Overall there is a need

for appropriately powered controlled trials performed in general psychiatric settings to test the efficacy of therapies in a wider context.

One potential method of enhancing CBT is with a pharmacologic agent such a D-cycloserine* (DCS) that may boost extinction learning that occurs during exposure exercises. DCS augmented behavior therapy has been tested with mixed results in disorders similar to BDD. Weingarden et al (2019) conducted a double blind RCT comparing DCS to placebo- augmented CBT for BDD (*N* = 26). Over 10 weeks of treatment, BDD severity as well as insight and depression improved significantly in both treatment arms, but there were no differences between the two conditions.

So-called 'third wave' psychological therapies are increasingly popular and are gaining an evidence base across a number of psychiatric disorders. There is some emerging evidence for efficacy in OCD, notably for Acceptance and Commitment Therapy (ACT) (Bluett et al, 2014). For BDD, we are aware of no robust clinical trial evidence for ACT, but many practitioners incorporate elements of this approach in treating such patients, and anecdotally the ACT 'dialogue' can assist engagement.

Children and adolescents

BDD usually first manifests in childhood or adolescents. However, there is often a substantial delay in diagnosis and appropriate treatment. In terms of treatments, few studies have specifically addressed young people. Greenberg and colleagues (2016) tested CBT outcomes in 13 adolescents with BDD. After 12 sessions, BDD and related symptoms (e.g., insight, mood) were significantly improved. Seventy-five percent of

adolescents who started treatment and 100% of completers were treatment responders. Treatment gains were maintained at 3- and 6-month follow-up.

Mataix-Cols and colleagues (2015) randomized 30 adolescents with BDD and their families, to either 14 sessions of CBT delivered over 4 months or a control condition consisting of written psycho-education materials and weekly telephone calls. The CBT group showed a significantly greater improvement in BDD symptoms (and secondary symptoms) than the control group.

SRIs, often at relatively high doses, appear efficacious for children and adolescents with BDD. Data are quite limited in this age group, but clinical experience indicates that SRIs are usually efficacious for youth with BDD; in addition, in other psychiatric disorders, medications that are effective for adults are usually also effective for youth.

In addition to multiple case reports reporting efficacy for SRIs in children and adolescents, Phillips et al (1995) described the treatment of four adolescents with severe BDD who substantially improved with fluoxetine* or paroxetine*. In a subsequent series of 33 children and adolescents with BDD (14.9 +/- 2.2 years of age), among those treated with an SRI 53% (n = 19) had significant improvement in BDD. In the subset of 13 SRI trials that were conducted by the authors, which tended to use higher doses than trials not conducted by the authors, 62% led to significant improvement in BDD symptoms. In contrast, no non-SRI medication was effective in decreasing BDD symptoms (Albertini and Phillips, 1999). When treating children, it is

recommended that SRIs be initiated at lower doses than in adults and that doses be limited to the regulatory maximum dose.

Online and smartphone based interventions

Two evidence-based CBT treatment manuals for BDD have been published, which enable CBT therapists without expertise in BDD to treat these patients (Veale and Neziroglu, 2010, Wilhelm et al, 2013). However, to meet the demand for expert psychological care in people with BDD, the potential of on-line interventions is exciting. Such interventions can also help deliver expert care to rural and remote communities, as well as reach people who might, due to shame and stigma, not otherwise seek appropriate help. Enander et al (2014) developed a 12-week on-line CBT program for BDD (BDD-NET). In an open-label feasibility study of 23 individuals with BDD, BDD-NET showed promising outcomes, including high acceptability. Significant within-group improvement was found on the BDD-YBOCS (Cohen's d=2.01 (95%CI 1.05 to 2.97), representing a large effect size). Fully 82% of participants were classed as responders (>= 30% reduction in BBD-YBOCS), and gains were maintained at 3-month follow-up. Improvements were also seen on secondary outcome measures, including global functioning, quality of life and depression.

The same research group (Enander et al, 2016) subsequently reported a 12-week single-blind randomised trial of BDD-NET (n=47) vs. SPT delivered via the internet (n=47). BDD-NET showed superiority to SPT on the BDD-YBOCS (group difference -7.1 points; 95%CI -9.8 to -4.4) as well as on ratings of depression, global functioning and quality of life. Among BDD-NET participants, 56% were rated as responders vs. 13% of

those receiving supportive psychotherapy. The number needed to treat (NNT) was 2.34 (95%CI 1.71 to 4.35) and self-reported satisfaction was high. Patients who received SPT were subsequently offered BDD-NET and all but four accepted. A two-year follow up of 88 of the 90 people who had received BDD-NET (two were lost to follow up) showed persistence of gains for BDD symptoms and global functioning but not quality of life (Enander et al, 2019).

Recently, Gentile et al (2019) translated BDD-NET from Swedish to English and completed the first Internet-based, therapist-guided, CBT for BDD with global inclusion criteria. Thirty-two patients from nine different countries participated in this uncontrolled pilot study**.** BDD symptoms improved significantly over the course of the 12 week treatment phase, and therapeutic gains were maintained at 3-month follow-up. The study showed that ICBT can be safely delivered across international borders to patients who otherwise might not have access to specialty care.

Recently, Wilhelm et al (2020) developed and tested the first smartphone CBT app for BDD to examine the potential of another low cost, accessible and standardized BDD intervention. The program was developed with extensive input from BDD patient consultants as well as engineering, design, and clinical psychology experts. The app offered CBT skills, and an asynchronous chat feature that allowed for brief interactions with a therapist. The 12-week open pilot trial (N = 10) showed that smartphone-based CBT for BDD may be feasible, and acceptable. In fact, nobody dropped out of the study and treatment satisfaction was high. The study also showed improved BDD symptom severity, as well as improved BDD-related insight, functional impairment, and quality

of life. Ninety percent of participants were responders at posttreatment and 3-month follow-up.

These studies are highly encouraging and speak to the ability of technology to reach people who either cannot access face-to-face therapists, or who simply prefer that mode of delivery. Further studies should compare outcomes with face-to-face therapy as well as delineate which individuals with BDD are best suited to which mode of delivery.

Gaze and eye movements

Distinguishing features of BDD from other OCRDs have also been noted regarding gaze and visual perception. Reflecting the tendency of those with BDD to focus their attention on specific facial or bodily features that are the area of preoccupation, research studies have shown an imbalance in the local and global visual processing systems within this population (see Beilharz et al., 2017 for a review). These findings indicate that people with BDD display a visual attention bias for specific details or features (local), rather than perceiving an image as a whole (global), whereas a combination of both strategies is effectively used by non-BDD populations (Kimchi, 1992; Love et al, 1999).

While abnormalities in basic eye movements or saccades have been noted within other psychiatric conditions, including OCD, anorexia nervosa and schizophrenia (Phillipou et al, 2014, 2016; Gadel et al, 2012; Karoumi et al, 1998; Landgraf et al, 2008; McDowell et al, 1995), there is preliminary evidence that these eye movements

are generally intact in BDD patients (Beilharz et al, 2020). It appears likely then, that abnormalities within higher order levels of visual processing, such as patterns of scanning complex images, may be responsible for the differences in perception apparent among people with BDD.

The primary evidence of disrupted higher order processing in BDD comes from the literature on face processing. Individuals with BDD typically have higher error rates and slower response times when recognising the identity and emotions of face stimuli (Feusner et al, 2006, 2010; Buhlmann et al, 2002, 2004, 2006, 2016; Grace et al., 2019a; Jefferies et al, 2012; Toh et al, 2015). Abnormalities in eye movements have also been noted when viewing these images, indicating a pattern of 'hyposcanning', with higher mean saccade amplitude, fewer fixations of extended duration and more blinks (Toh et al, 2015, 2017; Greenberg et al, 2014; Grocholewski et al, 2012), The location of an individual's gaze can also indicate patterns of disrupted perception, as individuals with BDD tend to avoid the most salient facial features (eyes, nose, mouth) and instead focus upon or avoid the perceived areas of concern. Similar patterns have been noted for own and others' faces among those with BDD, which may be analogous to the compulsive behaviours of repeatedly checking or avoiding one's appearance in the mirror or comparing to others.

Given the strong research literature of visual perception abnormalities within BDD, specific strategies targeting perception are recommended as part of treatment. Within CBT, this includes perceptual mirror retraining, where individuals are taught to view themselves in a more holistic and non-judgmental manner (Wilhelm et al, 2013, 2014).

Directions for future research also include specific visual training programs to support more traditional therapies for BDD, such as cognitive remediation, which has effectively been used within other psychiatric disorders (Beilharz et al, 2018). Indeed, as with OCD, specific areas of cognitive dysfunction may turn out to represent novel treatment targets for people with BDD. For example, a small cognitive affective neuroscience study showed that individuals with BDD performed poorly on a variety of neurocognitive tests of cognitive flexibility, reward and motor impulsivity and affective processing, similar to the areas of cognitive dysfunction seen in OCD. However, these data also hinted at additional areas of decision-making abnormality that might contribute specifically to the psychopathology of BDD (Jefferies et al, 2017).

Oxytocin*

As noted above, one of the most well-replicated empirical findings in BDD pertains to poor social cognition, especially facial affect perception. BDD patients make more errors when asked to perceive facial emotions, especially when viewing neutral and negative expressions. Oxytocin* (OXT) is a neuropeptide that acts as a neurotransmitter and has been documented to be a key modulator of complex social behaviours and social cognition throughout mammalian evolution. It is well-known for its role in attachment, social exploration, social recognition, fear extinction and anxiety reduction. Intranasal delivery is the most common method of administration, argued to provide a direct pathway into the brain. OXT receptors are present in the limbic and reward-related regions of the brain, including the amygdala; the amygdala is a key region in the 'social brain', and these areas are associated with social cognitive performance.

Fang et al (2019) administered intranasal OXT (24 international units) or placebo to 18 BDD patients and 16 healthy controls, using a within-subject cross-over design. They failed to find an effect of OXT on emotion recognition accuracy for either self- or other-referent tasks. In the BDD participants, OXT actually worsened the tendency to internal attributions on other-referent tasks, relative to controls. The authors conclude that caution needs to be exerted in using OXT in BDD.

More recent evidence has established that BDD patients exhibit abnormal amygdala-temporal connectivity during a resting state functional magnetic resonance scan, and that OXT administration (24 international units) restored this deficit, increasing connectivity to levels equivalent in BDD patients (n=19) relative to a group of healthy controls (n=17) during their placebo session (Grace et al, 2019b). In psychotic disorders, neurobiological change during acute OXT trials has shown translation into substantial clinical improvements when delivered daily (i.e. 6-8weeks). Thus, the study of Grace and colleagues (2019b) provides promising data, and suggests that OXT should be further investigated as a novel intervention for those with BDD. Such studies need to be cognisant, however, of the concerns raised by Fang et al (2019) based on their pilot treatment study findings.

Neurostimulation

Although emerging evidence supports the use of neurostimulation paradigms, notably repetitive transcranial magnetic stimulation* (rTMS) in OCD, there is substantial variability in the methodology, including anatomical site, total number of stimuli per

session, duration of trial, frequency and bilateral vs. unilateral application (Lusicic et al, 2018). We are not aware of any published studies specifically of neurostimulation in BDD, but it would appear to be an area worthy of attention. Neuroanatomical targets and stimulation parameters would not necessarily be the same as those used in OCD research; the occipital lobe might be a justifiable target, given the prominence of visual cortical involvement in neurobiological models of BDD (see above). To our knowledge, deep brain stimulation*, which has been effectively used in cases of treatment refractory OCD, has not been specifically studied in BDD and thus its efficacy for this indication is unknown.

Electroconvulsive therapy* (ECT) is not usually recommended for BDD; limited case series data suggest that it is not typically effective (Phillips, 2017). However, it can be considered if there is severe comorbid depression and high levels of suicidality which would meet criteria for ECT in itself, as indicated for patients with OCD (Koran et al., 2007). Mahato et al (2016) reported a case in whom both depressive and BDD symptoms responded to ECT.

Conclusions

BDD is a common and often severe psychiatric disorder. Patients often do not seek help directly from mental health professionals, and sensitive questioning is required for case ascertainment. There are established screening, diagnostic and outcome measures for BDD. The preferences of the patient need to be included in treatment planning. The mainstay of pharmacological therapy is SRIs*, which often require high doses and protracted periods to establish full benefit. SNRIs* may be considered as a

second-line treatment. Various adjunctive medications can be considered, including atypical antipsychotics*, anxiolytics*, and the anticonvulsant levetiracetam*; large scale RCTs are, however, lacking. BDD is not an approved indication for these medications because no pharmaceutical company has pursued an indication for BDD. The potential role of oxytocin* in treating BDD requires further exploration. The first-line psychological therapy is CBT that is specifically tailored to BDD's unique clinical features. The nuancing of these treatments to address such issues as mirror use, perturbations of gaze and misinterpretation of the emotions of others, is important, and may involve specific training of visual processes. On-line and telephone-assisted forms of psychological therapies are emerging and seem to be effective and well accepted by patients with BDD, although additional studies are needed, including which patients with BDD these treatments are best suited for.

Authors' contributions.

All authors were involved in drafting the manuscript and agreed to its publication. All authors read and approved their sections of the final version of the manuscript**.**

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References

Albertini RS, Phillips KA (1999) Thirty- three cases of body dysmorphic disorder in children and adolescents. Journal of The American Academy of Child and Adolescent Psychiatry 38: 453– 459

Allen A, Hadley SJ, Kaplan D, et al (2008) An open-label trial of venlafaz=xine in body dysmorphic disorder. CNS Spectrums 13: 138-144

Angelakis I, Gooding PA, Panagioti M (2016) Suicidality in body dysmorphic disorder (BDD): A systematic review with meta-analysis. Clinical Psychology Review 49: 55-66

American College of Obstetricians and Gynecologists*,* 2017, 2020

American Psychiatric Association (1980) The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) Washington DC: American Psychiatric Association

American Psychiatric Association. Practice guideline for the treatment of patients with obsessive-compulsive disorder. Arlington, VA: American Psychiatric Association, 2007. Available online at http//www.psych.org/psych_pract/treatg/pg/ prac_ guide.cfm.

American Psychiatric Association (2013) The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) Washington DC: American Psychiatric Association

Beilharz F, Castle DJ, Grace S, Rossell SL (2017) A systematic review of visual processing and associated treatments in body dysmorphic disorder. Acta Psychiatrica Scandinavica 136: 16-36

Beilharz F, Castle DJ, Phillipou A, Rossell S (2018) Visual training program for body dysmorphic disorder: protocol for a novel intervention pilot and feasibility trial. Pilot and Feasibility Studies 4: 198

Beilharz F, Phillipou A, Castle DJ, Rossell S (2020) Saccadic eye movements in body dysmorphic disorder. Journal of Obsessive and Compulsive Disorders 25: https://doi.org/10.1016/j.jocrd.2020.100526

Blomeley D, Phillipou A, Castle DJ (2018) Sizing it up: A systematic review of the nosology of muscle dysmorphia. Clinical Research in Psychology 1: 1-10

Bluett EJ, Homan KJ, Morrison KL, et al (2014) Acceptance and commitment therapy for anxiety and OCD spectrum disorders: An empirical review. Journal of Anxiety Disorders 28: 612-624

Brakoulias V, Stockings E (2019) A systematic review of the use of risperidone, paliperidone and aripiprazole as augmenting agents for obsessive-compulsive disorder. Expert Opinion on Pharmacotherapy 20: 47-53

Brotman AW, Jenike MA (1984) Monosymptomatic hypochondriasis treated with tricyclic antidepressants. American Journal of Psychiatry 141: 1608-1609

Buhlmann U, Etcoff NL, Wilhelm S (2006) Emotion recognition bias for contempt and anger in body dysmorphic disorder. Journal of Psychiatric Research 40: 105-111

Buhlmann U, Gleiss MJ, Rupf L, Zschenderlein K, Kathmann N (2011) Modifying emotion recognition deficits in body dysmorphic disorder: an experimental investigation. Depression Anxiety 28: 924-931

Buhlmann U, McNally RJ, Etcoff NL, Tuschen-Caffier B, Wilhelm S (2004) Emotion recognition deficits in body dysmorphic disorder. Journal of Psychiatry Research 38: 201-206

Buhlmann U, McNally RJ, Wilhelm S, Florin I (2002) Selective processing of emotional information in body dysmorphic disorder. Journal of Anxiety Disorders 16: 289-298

Castle DJ, Phillips KA (2006) The OCD spectrum of disorders: a defensible construct? Australian and New Zealand Journal of Psychiatry 40: 114-120

Castle DJ, Rossell S, Kyrios M (2006) Body dysmorphic disorder. Psychiatric Clinics of North America 29: 521-538

Crerand CE, Phillips KA, Menard W, Fay C (2005). Nonpsychiatric medical treatment of body dysmorphic disorder. Psychosomatics. 46 :549–555

Cuijpers P, Driessen E, Hollon SD, et al (2012) The efficacy of non-directive supportive therapy for depression: a meta-analysis. Clinical Psychology Reviews 32: 280-291

Dong N, Nezgovorova V, Hong K, Hollander E (2019) Pharmacotherapy in body dysmorphic disorder: relapse prevention and novel treatments. Expert Opinion On Pharmacotherapy 20: 1211-1219

Dufresne RG, Phillips KA, Vittorio CC, Wilkel CS (2001). A screening questionnaire for body
dysmorphic disorder in a cosmetic dermatologic surgery practice. Dermatologic Surgery
27: 457- 462

Enander J, Ivanov VZ, Andersson E, et al (2014) Therapist-guided, Internet-based cognitive-behavioural therapy for body dysmorphic disorder (BDD-NET): a feasibility study. BMJ Open 4: e005923

Enander J, Andersson E, Mataix-Cols D, et al (2016) Therapist-guided, internet-based cognitive-behavioural therapy for body dysmorphic disorder: single-blind randomised controlled trial. BMJ 352: i241

Enander J, Ljotsson B, Anderhell L, et al (2019) Long-term outcome of therapist-guided, Internet-based cognitive-behavioural therapy for body dysmorphic disorder (BDD-NET): a naturalistic 2-year follow-up after a randomised controlled trial. BMJ Open 9: e024307

Fang A, Lawson EA, Wilhelm S (2019) Intranasal oxytocin modulates higher order social cognition in body dysmorphic disorder. Depression and Anxiety 36: 153-161

Fernandez de la Cruz L, Enander J, Ruck C, et al (2019) Empirically defining treatment response and remission in body dysmorphic disorder. Psychological Medicine doi.org/10.1017/50033291719003003

Feusner JD, Bystritsky A, Hellemann G, Bookheimer S (2010) Impaired identity recognition of faces with emotional expressions in body dysmorphic disorder. Psychiatry Research 179: 318-323

Feusner JD, Townsend J, Bystritsky A, Bookheimer S (2006) Visual information processing of faces in body dysmorphic disorder. Neuropsychopharmacology 31: S210-S211.

Fineberg NA, Hollander E, Pallanti S, Walitza S, Grünblatt E, Dell'Osso BM, Albert U, Geller DA, Brakoulias V, Janardhan Reddy YC, Arumugham SS, Shavitt RG, Drummond L, Grancini B, De Carlo V, Cinosi E, Chamberlain SR, Ioannidis K, Rodriguez CI, Garg K, Castle D, Van Ameringen M, Stein DJ, Carmi L, Zohar J, Menchon JM. Clinical advances in obsessive-compulsive disorder: a position statement by the International College of Obsessive-Compulsive Spectrum Disorders. .Int Clin Psychopharmacol. 2020 May 18. doi: 10.1097/YIC.0000000000000314. Online ahead of print.PMID: 32433254
Gadel R, Coen C, Seassau M, et al (2012) First results from an antisaccade task and memory-guided saccade task in a neurodevelopmental approach to schizophrenia. Schizophrenia Research 136: S324-S324.

Gazzarrini D and Perugi G (2017) Gender and body dysmorphic disorder. In: Body Dysmorphic Disorder: Advances in Research and Clinical Practice (Phillips KA, Ed.) New York, NY: Oxford University Press

Gentile A, La Lima C, Flygare O, et al (2019) Internet-based, therapist guided, cognitive behavioral therapy for body dysmorphic disorder with global eligibility for inclusion: An uncontrolled pilot study. BMJ Open 9(3). PMCID: PMC6475214

Grace S, Labuschagne I, Kaplan RA, et al (2017) The neurobiology of body dysmorphic disorder: a systematic review and theoretical model. Neuroscience and Biobehavioural Reviews 83: 83-96

Grace SA, Toh WL, Buchanan B, Castle DJ, Rossell SL (2019a) Impaired recognition of negative facial emotions in body dysmorphic disorder. Journal of International Neuropsychological Society 25: 884-889

Grace SA, Labuschagne I, Castle DJ, Rossell SL (2019b) Intranasal oxytocin alters amygdala-temporal resting-state functional connectivity in body dysmorphic disorder: A double-blind placebo-controlled randomized trial. Psychoneuroendocrinology 107: 179-186

Goodman WK, Price LH, Delgado PL, et al (1990) Specificity of serotonin reuptake inhibitors in the treatment of obsessive-compulsive disorder: comparison of fluvoxamine and desipramine. Archives of General Psychiatry 47: 577- 585

Greenberg JL, Mothi SS, Wilhelm S (2016) Cognitive-Behavioral Therapy for Adolescent Body Dysmorphic Disorder: A Pilot Study. Behavior Therapy 47: 213-24

Greenberg JL, Reuman L, Hartmann, AS, Kasarskis I, Wilhelm S (2014) Visual hot spots: An eye tracking study of attention bias in body dysmorphic disorder. Journal of Psychiatric Research 57: 125-132

Grocholewski A, Kliem S, Heinrichs N (2012) Selective attention to imagined facial ugliness is specific to body dysmorphic disorder. Body Image 9: 261-269

Harrison A, Fernandes de la Cruz L, Enander J, et al (2016) Cognitive behavioural therapy for body dysmorphic disorder: a systematic review and meta-analysis of randomised controlled trials.

Hollander E, Allen A, Kwon J, et al (1999) Clomipramine vs desipramine crossover trial in body dysmorphic disorder: selective efficacy of a serotonin reuptake inhibitor in imagined ugliness. Archives of General Psychiatry 56: 1033-1039

Huhn M, Nikolakopoulou A, Schneider-Thoma J, et al (2019). Comparative efficacy and tolerability of 32 oral antipsychotics for the acute treatment of adults with multi-episode

schizophrenia: a systematic review and network meta-analysis. The Lancet 394; 939-951

Hutton MJ, Cave AJ, St-Jean R, et al (2016) Should we be worried about QTc prologation using citalopram? Journal of Pharmacy Practice doi.org/10.1177/0897190015624862

Jenike MA (1984) A case report of successful treatment of dysmorphophobia with tranylcypromine. American Journal of Psychiatry 141: 1463-1464

Jefferies K, Laws K, Fineberg NA (2012) Superior face recognition in Body Dysmorphic Disorder. Journal of Obsessive-Compulsive and Related Disorders 1: 175-179

Jefferies-Sewell K, Chamberlain SR, Fineberg NA and Laws KR. Cognitive dysfunction in Body Dysmorphic Disorder: New implications for nosological systems & neurobiological models. CNS Spectr. 2017 Feb; 22(1): 51–60.

Karoumi B, Ventre-Dominey J, Vighetto A, et al (1998) Saccadic eye movements in schizophrenic patients. Psychiatry Research 77: 9–19

Kimchi R (1992) Primacy of holistic processing and global/local paradigm: A critical review. Psychological Bulletin 112: 24–38

Koran L, Abujaoude E, Large MD, Serpe RT (2008) The prevalence of body dysmorphic disorder in the United States adult population. CNS Spectrums 13: 316-322

Labuschagne I, Dunai J, Castle DJ, Kyrios M, Rossell SL (2010) An examination of cognitive styles in Body Dysmorphic Disorder (BDD), particularly their delusional thinking. Australian and New Zealand Journal of Psychiatry 44: 706-712

Landgraf S, Amado I, Bourdel MC, Leonardi S, Krebs MO (2008) Memory-guided saccade abnormalities in schizophrenic patients and their healthy, full biological siblings. Psychologie Medicale 38: 861–870

Love BC, Rouder JN, Wisniewski EJ (1999) A structural account of global and local processing. Cognitive Psychology 38: 291–316.

Lusicic A, Schruers KRJ, Pallanti S, Castle DJ (2018) Transcranial magnetic stimulation in the treatment of obsessive-compulsive disorder: current perspectives. Neuropsychiatric Disease and Treatment 14, 1721

Mahato RS, San Gabriel MCP, Longshore CT, Schnur DB (2016) A case of treatment-resistant depression and Body Dysmorphic Disorder: the role of electroconvulsive therapy revisited. Innovations in Clinical Neuroscience 13: 37-40

Malcolm A, Labuschagne I, Castle D, et al (2018). The relationship between body dysmorphic disorder and obsessive-compulsive disorder: A systematic review of direct comparative studies. Australian and New Zealand Journal of Psychiatry 52: 1030-1049

Mancuso P, Knoesen NP, Castle DJ (2010) Delusional versus nondelusional body dysmorphic disorder. Comprehensive Psychiatry 51: 177-182

Mataix-Cols D, de la Cruz LF, Isomura K, et al (2015) A pilot randomised controlled trial of cognitive-behavioral therapy for adolescents with body dysmorphic disorder. 54: 895-904

McDowell JE, Farber RH, Clementz BA (1995) Memory-guided saccade performance among schizophrenia and obsessive-compulsive disorder patients. Schizophrenia Research 15: 180–181

McKay D, Torado J, Bjureberg J, et al (1997) Body dysmorphic disorder: a preliminary evaluation of treatment and maintenance using exposure with response prevention. Behaviour Research and Therapy 35: 67-70

Menin DS (2019) Identifying efficacious treatment elements for refractory conditions, such as body dysmorphic disorder. JAMA Psychiatry 76: 357-358

Menkes D, Bosanac P, Castle DJ (2016). MAOIs: does the evidence warrant their resurrection? Australasian Psychiatry 24: 371-373

Oosthuizen P, Lambert T, Castle DJ (2010) Dysmorphic concern: prevalence and associations with clinical variables. Australian and New Zealand Journal of Psychiatry 32: 129-132

Pallanti S, Koran LM (1996) Intravenous, pulse-loaded clomipramine in body dysmorphic disorder: two case reports. CNS Spectrums 1: 54-57

Phillipou A, Rossell SL, Castle DJ, Gurvich C, Abel LA (2014) Square wave jerks and anxiety as distinctive biomarkers for anorexia nervosa. Investigative Ophthalmology and Visual Science 55: 8366

Phillipou A, Rossell SL, Wilding HE, Castle DJ (2016a) Randomised controlled trials of psychological and pharmacological treatments for body dysmorphic disorder: a systematic review. Psychiatry Research 245: 179-185

Phillipou A, Rossell SL, Gurvich C, Castle DJ, Abel LA (2016b) The eyes have it: Eye movements and anorexia nervosa. Australian and New Zealand Journal of Psychiatry 50: 806–807

Phillipou A, Castle DJ, Rossell SL (2019) Direct comparisons of anorexia nervosa and body dysmorphic disorder: a systematic review. Psychiatry Research 274: 129-137

Phillips KA (2005) Olanzapine augmentation of fluoxetine in body dysmorphic disorder. American Journal of Psychiatry 162: 1022-1023

Phillips KA (2005) Placebo-controlled study of pimozide augmentation of fluoxetine in body dysmorphic disorder. American Journal of Psychiatry 162: 377-379

Phillips KA. The Broken Mirror: Understanding and Treating Body Dysmorphic Disorder. New York, NY: Oxford University Press, 1996 (Revised and Expanded Edition, 2005)

Phillips K (2006) An open-label study of escitalopram in body dysmorphic disorder. International Journal of Clinical Psychopharmacology 21: 177-179

Phillips KA. Pharmacotherapy and other somatic treatments for body dysmorphic disorder.
In: *Body Dysmorphic Disorder: Advances in Research and Clinical Practice.* Phillips KA, editor.
New York, NY: Oxford University Press, 2017

Phillips KA, Albertini RS, Siniscalchi JM, Khan A, Robinson M (2001). Effectiveness of pharmacotherapy for body dysmorphic disorder: a chart- review study. J Clin Psychiatry
62: 721– 727

Phillips KA, Atala KD, Albertini RS (1995). Case study: body dysmorphic disorder in adolescents. J Am Acad Child Adolesc Psychiatry 34: 1216– 122

Phillips KA, Grant J, Siniscalchi J, Albertini RS (2001). Surgical and nonpsychiatric medical treatment of patients with body dysmorphic disorder. Psychosomatics 42 :504– 510.

Phillips KA, Kelly MM (2009). Suicidality in a placebo-controlled fluoxetine study of body dysmorphic disorder. *International Clinical Psychopharmacology* 24: 26-28

Phillips KA, Menard W (2006) Suicidality in body dysmorphic disorder: a prospective study. American Journal of Psychiatry 163: 1280-1282

Phillips KA, Menard W, Quinn E, Didie ER, Stout RL (2103). A four-year prospective observational follow-up study of course and predictors of course in body dysmorphic disorder. Psychological Medicine 43: 1109-1117

Phillips KA, Najjar F (2003) An open-label study of citalopram in body dysmorphic disorder. Journal of Clinical Psychiatry 64: 715-720

Phillips KA, McElroy SL, Keck PE, et al (1993) Body dysmorphic disorder: 30 cases of imagined ugliness. American Journal of Psychiatry 150: 302-308

Phillips KA, McElroy SL, Keck PE Jr, Pope HG Jr, Hudson JI (1994). A comparison of delusional and nondelusional body dysmorphic disorder in 100 cases. Psychopharmacology Bulletin 30: 179-186

Phillips KA, Albertini RS, Rasmussen SA (2002) A randomised placebo-controlled trial of fluoxetine in body dysmorphic disorder. Archives of General Psychiatry 59: 381-388

Phillips KA, Menard W, Fay C, Weisberg (2005) Demographic characteristics, phenomenology, comorbidity, and family history in 200 individuals with body dysmorphic disorder. Psychosomatics 46: 317-325

Phillips KA, Keshaviah A, Dougherty DD, et al (2016) Pharmacotherapy relapse prevention in body dysmorphic disorder: a double-blind, placebo-controlled trial. American Journal of Psychiatry 173: 887-895

Rabiei M, Mulkens S, Kalantari M, Molavi H, Bahrami F (2012) Metacognitive therapy for body dysmorphic disorder patients in Iran: a proof of concept study. Journal of Behavioural Therapy and Experimental Psychiatry 43: 724-729

Rashid H, Khan AA, Fineberg NA. Adjunctive antipsychotic in the treatment of body dysmorphic disorder - A retrospective naturalistic case note study. Int J Psychiatry Clin Pract. 2014 Nov 28:1-6.

Read J, Williams J (2018) Adverse effects of antidepressants reported by a large international cohort: emotional blunting, suicidality and withdrawal effects. Current Drug Safety 13: 176-186

Rief W, Buhlmann U, Wilhelm S, Borkenhagen A, Brahler E (2006) The prevalence of body dysmorphic disorder: a population-based survey. Psychological Medicine 36: 877-885

Rosen JC, Reiter J, Oroson P (1995) Cognitive-behavioural body image therapy for body dysmorphic disorder. Journal of Consulting and Clinical Psychology 63: 263-269

Rossell SL, Harrison BJ, Castle D (2015) Can understanding the neurobiology of body dysmorphic disorder inform treatment? Australasian Psychiatry 23: 361-364

Rossell SL, Labuschagne I, Castle DJ, Toh WL (2020) Delusional themes in Body Dysmorphic Disorder (BDD): comparisons with psychotic disorders and non-clinical controls. Psychiatry Research doi: 10.1016/j.psychres.2019.112694.

Schieber K, Kollei I, de Zwaan M, Martin A (2015) Classification of body dysmorphic disorder: What is the advantage of the new DSM- 5 criteria? J Psychosomatic Research 78: 223-227

Simberlund J, Hollander E: The relationship of body dysmorphic disorder to obsessive-compulsivedisorder and the concept of the obsessive-compulsive spectrum. In:

Phillips KA (editor). *Body Dysmorphic Disorder: Advances in Research and Clinical Practice.* New York, NY: Oxford University Press, 2017

Tyagi H, Govender A, Drummond LM (2012) Gender Differences in Body Dysmorphic Disorder. 165th Annual Meeting of the American Psychiatric Association, Philadelphia

Tod D, Edwards C, Cranswick I (2016) Muscle dysmorphia: current insights. Psychology Research and Behaviour Management 9: 179-188

Toh W-L, Castle DJ, Rossell SL (2015) Facial affect recognition in body dysmorphic disorder versus obsessive-compulsive disorder: An eye-tracking study. Journal of Anxiety Disorders 35: 49-59

Toh W-L, Castle DJ, Rossell SL (2017a) How individuals with body dysmorphic disorder (BDD) process their own face: a quantitative and qualitative investigation based on an eye-tracking paradigm. Cognitive Neuropsychiatry 22: 213-232

Toh WL, Castle DJ, Mountjoy RL, et al (2017b) Insight in body dysmorphic disorder (BDD) relative to obsessive-compulsive disorder (OCD) and psychotic disorders: revisiting this issue in light of DSM-5. Comprehensive Psychiatry 77:100-108

Toh WL, Castle DJ, Rossell SL (2017c) Characterisation of body dysmorphic disorder (BDD) versus obsessive-compulsive disorder: in light of current DMS-5 nosology. Journal of Obsessive-Compulsive and Related Disorders 12: 117-126

Uzun O, Ozdemir B (2010) Aripiprazole as an augmentation agent in treatment-resistant body dysmorphic disorder. Clinical Drug Investigation 30: 707-710

Veale D (2000) Outcome of cosmetic surgery and DIY surgery in patients with Body Dysmorphic Disorder. Psychiatric Bulletin, 24: 218-221

Veale D, Matsunaga H (2014) Body dysmorphic disorder and olfactory reference disorder: proposals for ICD-11. Brazilian Journal of Psychiatry 36 (suppl 1): doi.org/10.1590/1516-4446-2013-1238

Veale D, Willson R, Clark A (2009) Overcoming body image problems (including body dysmorphic disorder) Constable Robinson: London.

Veale D, Anson M, Miles S, et al (2014) Efficacy of cognitive behaviour therapy versus anxiety management for body dysmorphic disorder: a randomised controlled trial. Psychotherapy and Psychosomatics 83: 341-353

Veale D, Gournay K, Dryden W, et al (1996) Body dysmorphic disorder: a cognitive behavioural model and pilot randomised controlled trial. Behaviour Research and Therapy 34: 717-729

Veale D, Ellison N, Werner T, Dodhia R, Serfaty M, Clarke A (2012) Development of a cosmetic procedure screening questionnaire (COPS) for Body Dysmorphic Disorder. Journal of Plastic Reconstructive and Aesthetic Surgery 65: 530-532

Veale D, Miles S, Smallcombe N, Ghezai H, Goldacre B, and Hodsoll J (2014) Atypical antipsychotic augmentation in SSRI treatment refractory obsessive-compulsive disorder: a systematic review and meta-analysis. BMC Psychiatry 14: 317

Veale D, Gledhill LJ, Christodoulou P, Hodsoll J (2016) Body dysmorphic disorder in different settings: a systematic review and weighted prevalence. Body Image 18: 168-186

Veale D, Phillips KA, Neziroglu F (2017). Challenges in assessing and treating patients with body dysmorphic disorder and recommended approaches. In: *Body Dysmorphic Disorder: Advances in Research and Clinical Practice.* Phillips KA, editor. New York, NY: Oxford University Press

Weingarden H, Mothi SS, Ladis I, et al (2019) D-cycloserine-augmented behavior therapy for body dysmorphic disorder: A Preliminary Efficacy Trial. Cognitive Therapy and Research 43: 937–947

Wilhelm S (2006). Feeling good about the way you look: A program for overcoming body image problems. New York, NY: Guilford Press

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

Wilhelm S, Phillips KA, Didie E, et al (2014) Modular cognitive-behavioural therapy for body dysmorphic disorder: a randomised controlled trial. Behaviour Research and Therapy 45: 314-327

Wilhelm S, Phillips KA, Greenberg JL, et al (2019) Efficacy and posttreatment effects of therapist-delivered cognitive behavioural therapy vs supportive psychotherapy for adults with body dysmorphic disorder: a randomised clinical trial. JAMA Psychiatry 2019; 76: **