

## REVIEW ARTICLE

# Pharmacological Treatment of Body Dysmorphic Disorder

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**Abstract:** Body dysmorphic disorder is a challenging disorder that manifests as erroneously perceived flaws in one's physical appearance and repetitive behaviors in response to appearance concerns. This disorder is also frequently comorbid with other psychiatric disorders, including major depressive disorder and autism spectrum disorder. It is currently understood to arise from a combination of biological, psychological, and environmental factors. Treatment of body dysmorphic disorder typically consists of a combination of pharmacotherapy and cognitive behavioral therapy. However, not all patients respond to treatment, and BDD symptoms remain even in those who do respond. This review outlines current pharmacological and neuromodulation treatments for body dysmorphic disorder and suggests directions for future studies of novel treatments such as augmentation with atypical antipsychotics and the use of intranasal oxytocin in cases of body dysmorphic disorder that show residual symptomatology even with tailored monotherapy. There is emerging evidence suggesting that non-invasive neurostimulatory techniques, such as repetitive transcranial magnetic stimulation, may be of value in treatment-resistant cases.

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## 1. CLINICAL PRESENTATION

Body dysmorphic disorder (BDD) is characterized by the belief that some aspects of one's appearance are ugly, unacceptable, or otherwise deformed, while this is in fact not the case [1]. Preoccupations regarding perceived physical flaws may consume an average of three to eight hours per day, are intrusive, and are associated with significant anxiety and distress [2-4]. These concerns can be focused on any combination of body regions, and focus on multiple perceived defects is common [5]. Although these concerns are typically focused, they can also involve general appearance, as is the case in muscle dysphoria [6]. These preoccupations are associated with low self-esteem, feelings of shame, depressive symptoms, anxiety, and guilt [7]. Patients often demonstrate heightened rejection sensitivity and believe themselves to be unacceptable. Patients are often highly sensitive and vulnerable to rejection experiences [8].

Many patients with BDD have little to no insight into the nature of their beliefs and preoccupations [9]; they overwhelmingly believe that their self-perceptions are accurate and that their concerns are appropriate [10]. Most patients do not perceive their beliefs as being due to a diagnosable mental

illness [11]. Many also demonstrate delusions of reference and believe that others take notice of their deformations [12]. Although the DSM-5 has recently reclassified BDD as being part of the obsessive-compulsive disorder (OCD) and related disorders (OCD) spectrum, it is notable that insight is lower in BDD than in OCD [13].

In response to their physical concerns, patients with BDD will follow compulsive behaviors, such as combing of hair in a specific pattern, in order to reduce the distress caused by their perceived deformations [14]. These behaviors are typically difficult to resist or control [15]. The associated feelings of guilt and shame appear to contribute to high rates of suicidal ideation and suicide attempts in BDD patients [16]. 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 [17].

Many patients are unaware of the existence of effective treatment and therapy and will hide or misrepresent symptoms because of feelings of guilt or embarrassment [18]. Patients often have difficulty in intimate relationships and broader social functioning because of their perceived deformations [19]. They also experience academic or occupational difficulties; many patients are unable to work, be in school, or complete schoolwork because of their symptoms [20]. Treatment is also a challenge because patients will often request unusual hours for appointments or otherwise poorly adhere to treatment when doing so requires that they encoun-

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ter others which risks exposing their perceived flaws to ridicule.

Comorbidity is often seen in BDD [21]. The most frequently occurring comorbidities include major depressive disorder (MDD), anxiety disorders, OCD, and substance-use disorders [22]. Overlapping symptom domains and thought patterns suggest that looking to these disorders, in which pharmacotherapy has been more extensively studied, may be of considerable value [23].

## 2. EPIDEMIOLOGY

The prevalence of BDD is estimated to be approximately 2% in the general population [24]. Prevalence may be higher in college student populations, at approximately 5%, as well as in clinical populations [25]. Outpatient adult psychiatric patient prevalence is estimated to be approximately 5.8%, and inpatient patient prevalence approximately 7.4% [26]. It is also notable that the prevalence among patients who sought non-psychiatric treatment, which includes dermatologic and cosmetic procedures, is estimated to be 11.3% in dermatology outpatient settings and 9.2% in cosmetic dermatology settings [26]. This suggests that dysmorphic concerns need to be incorporated into pre-operative and other non-psychiatric contexts.

Prevalence estimates suggest that BDD is more common in women (2.5%) in comparison to men (2.2%) [27]. The average age of onset is estimated to be 17 years [28], with an often-unremitting course; a recent study found a low cumulative probability (20%) of full remission over 4 years with treatment [29]. This low probability was further decreased by greater symptom severity at presentation [29].

## 3. ETIOLOGY

BDD is understood to arise from a combination of biological, psychological, and environmental factors [30]. 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 [31, 32]. A survey of patients with BDD found high rates of emotional neglect (68.0%), emotional abuse (56.0%), physical neglect (33.3%), physical abuse (34.7%), and sexual abuse (28.0%) [33]. These findings are in line with the current understanding of BDD as being associated with a low quality of life and with increased suicidality. Neuroimaging studies in BDD have implicated abnormal structure and function of occipitotemporal and frontolimbic regions; cortical gray matter thinning has been demonstrated within the left temporal and left inferior parietal regions [34]. Functional MRI (fMRI) studies have found that patients interpret visual information that is holistic in nature *via* neural pathways meant for detailed, focused information [35-38]. These studies also demonstrated hypoactive structural connectivity and poor information transfer between the primary and secondary occipital regions [37, 38]. Another fMRI study, comparing visual processing networks in BDD and in anorexia nervosa (AN), found similar patterns of higher-order connectivity between the right fusiform face area, the precuneus, and the posterior cingulate cortex [39]. Connec-

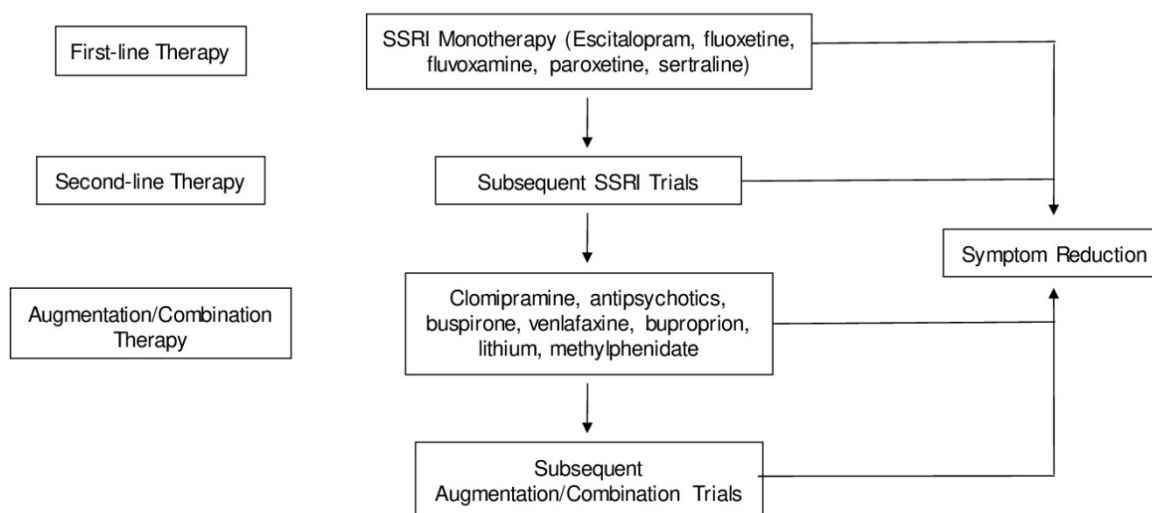
tivity between the insula and the central opercular cortex was also found to be reduced [39]. These results suggest reduced introspection in both BDD and in AN, and a tendency to assign inappropriate importance to visual information as processed [35, 39]. Additionally, reduced striatal dopamine D2/D3 receptor availability is observed in BDD, suggesting pathophysiology involving dopaminergic pathways and potential therapeutic targets [40]. Neuropsychological tests indicate that BDD patients experience deficits in the interpretation of facial emotional expressions, particularly as they relate to disgust and anger. Imaging studies have shown an increase in total white matter volume and caudate volume asymmetry in BDD patients, which is consistent with the recent classification of BDD as falling within the obsessive-compulsive spectrum [41]. Other studies have found a positive correlation between right amygdala volume and BDD symptom severity, suggesting engagement of neuroanatomical structures in pathological face processing, fear neurocircuitry, hypervigilance, and heightened social sensitivity [41, 42].

## 4. DIAGNOSIS

Diagnosis of BDD begins with an initial clinical evaluation that incorporates a mental status examination, a psychiatric history, and a medical history. Suicidality, delusional-ity, psychiatric comorbidity, and a history of dermatological and cosmetic surgical procedures are significant features potentially present in the patient presenting with BDD [43]. Per the DSM-5, BDD is diagnosed according to the following criteria: a preoccupation with one or more perceived defects or flaws in physical appearance that are either slight or not present, the presence of repetitive behaviors in response to these concerns, the preoccupations causing clinically significant distress and/or impairment in functioning, and these preoccupations failing to be better explained by an eating disorder [1]. BDD often goes undiagnosed in clinical practice [22]. Patients often decline to disclose symptoms out of feelings of shame and guilt or because they do not know that their symptoms can be treated clinically [22]. It is important to screen for and specifically treat BDD, as pharmacological treatment is informed by its many comorbidities and is also distinct from many disorders with which it shares symptomatology [44]. There is a demonstrated need for specific screening techniques in inpatient, substance abuse, dermatological, cosmetic surgery, and psychiatric settings; BDD is often present and comorbid in such contexts and may worsen outcomes if not identified and treated explicitly [30]. Awareness of the prevalence of BDD on the part of the clinician is of paramount importance because BDD is characterized by feelings of guilt and shame leading patients to mask their symptoms, and because of typically poor insight presenting challenges in establishing therapeutic relationships and ensuring treatment adherence.

## 5. THE ROLE OF PHARMACOTHERAPY

Treatment mainstays of BDD typically involve some combination of cognitive behavioral therapy (CBT) modalities and pharmacotherapy [45]. Antidepressant medications have been found to improve the core symptoms of BDD, suicidality, and quality of life, particularly in more severely ill and suicidal patients [2]. Medications currently used in the



**Fig. (1).** Step-wise pharmacotherapeutic approach to body dysmorphic disorder.

treatment of BDD are used in a range of contexts including as monotherapy, augmentation therapy, and combination therapy; this is shown in Fig. (1). Specific techniques and challenges in CBT have been well-documented elsewhere; this review will focus on current approaches to psychopharmacology in the treatment of BDD. 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. First-line pharmacological treatment of BDD is centered around the use of selective serotonin reuptake inhibitors (SSRIs), with the incorporation of clomipramine when necessary [46]. 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). 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. A 12-week randomized trial compared fluoxetine with placebo in 67 subjects with BDD [47]. 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%) [47]. Delusionality did not influence response to treatment, and treatment response was independent of symptom severity, duration, and the presence of any comorbidities [47]. Fluoxetine was also 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 [48].

Medication dosing presents an issue in the effective treatment of BDD. The therapeutically effective dosages required are often significantly higher than the doses used to effectively treat other psychiatric disorders, which presents challenges in drug tolerance and suitability in the treatment of pediatric populations [43]. This is an issue in administering clomipramine as well, as TCAs are known to have a more severe side effect profile than SSRIs. Medication is

typically titrated up, monitoring for patient tolerance and improvement, eventually reaching the manufacturer-recommended maximum dosage by week 5 to 9 [49]. Response to SSRI typically occurs gradually and requires 12 to 16 weeks to fully determine medication response. The manufacturer-recommended maximum dosage can be cautiously increased, depending on patient response and drug tolerance.

If patients do not demonstrate symptom improvement, monotherapy with a different SSRI is indicated. There is limited evidence supporting the addition of another SSRI [46], but buspirone, clomipramine, venlafaxine, and second-generation antipsychotics may prove to be viable options for medication augmentation [46, 50]. Augmentation therapy is usually prescribed for 6 to 8 weeks before drawing conclusions as to efficacy [50]. If SSRI trials are ineffective, the SSRI is discontinued in favor of clomipramine [49]. In patients that achieve symptom improvement with medication, maintenance is strongly indicated; discontinuation is strongly associated with relapse [29]. 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 [51, 52]. 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 [43].

## 6. DISCUSSION, CONCLUSION, FUTURE DIRECTIONS

Pharmacotherapy with antidepressants remains a foundational element in the treatment of BDD. In concert with CBT, response rates vary from about 50 to 80 percent [43]. Medications utilized will typically involve SSRI monotherapy with the possibility of augmentation with a second medication such as buspirone, clomipramine, second-generation antipsychotics [53], and venlafaxine. In the case of failure to improve symptoms, another SSRI can be tested, or SSRIs can be discontinued altogether in favor of clomipramine. The mean response time is about 4 to 9 weeks, and treatment for up to 16 weeks may be necessary to achieve

symptom improvement [43]. Approximately 20 percent of patients will recover from BDD, but 40 percent of those recovered will subsequently experience relapse [29]. It is important to note that the presence of and severity of delusional beliefs did not affect the probability of recovery or recurrence [29]. The risk of relapse was found to be associated with an earlier age of onset of BDD, and with the severity of symptoms at presentation [29]. Maintenance therapy is indicated, as the duration of remission and time to relapse were found to be extended with continued maintenance therapy.

Future directions in the study of BDD pharmacotherapy should also include placebo-controlled studies comparing various SSRIs; comparing SSRIs individually and as a class to other medication classes; further studies on medication discontinuation, maintenance, and relapse prevention; and regimens that incorporate augmentation medications, symptomatology and medication response in pediatric populations. Preliminary findings suggest that the demonstrated efficacy of SSRI monotherapy can be improved with augmentation co-medications, but there are few studies that investigate the promising medications thus far proposed as monotherapy. Dosage studies are required as well since the high dosages currently used to treat BDD lend themselves to higher rates of side effects and the associated potential risk of nonadherence. Additionally, there is a precedent for the investigation of both endogenous oxytocin in BDD and in the use of exogenous oxytocin formulations in the modulation of BDD symptomatology. Oxytocin administration has been associated with hyper-grooming behaviors as a model of compulsive behavior in obsessive-compulsive disorders [54]. Oxytocin receptor gene polymorphisms have been suggested as modulators in the age of onset in obsessive-compulsive disorder [55]. Prior clinical experience demonstrates overlapping symptom domains and frequent comorbidity with ASD, a condition in which the role of oxytocin has been previously established [23, 56]. Such associations are important to pursue, both to explore the role of oxytocin in BDD pathophysiology and to clarify the role and importance of comorbid conditions so often found in BDD.

Neurostimulation techniques represent novel directions for research based on the pathophysiological basis of BDD as an OCD. Recent studies of treatment regimens incorporating techniques such as repetitive transcranial magnetic stimulation (rTMS) and bilateral deep brain stimulation show promise in the treatment of complex, treatment-resistant BDD even in the context of profound comorbidities [57-60]. rTMS is currently FDA-approved for treatment of major depression and is promising in the treatment of OCD and related disorders, including BDD. In a single-blind randomized clinical trial, rTMS reduced both self-reported and provider-reported symptom severity in OCD [61]. Randomized controlled trials have found that rTMS is effective in cases of SSRI-resistant BDD [62]. These findings present interesting future directions in the treatment of BDD; however, it must be noted that these options are typically only utilized in refractory and treatment-resistant cases. An integrative neurostimulative and pharmacological approach can dramatically improve quality of life in patients and demonstrate the necessity for more clinical research and a deeper understanding of mechanisms involved in BDD.

## CONSENT FOR PUBLICATION

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## CONFLICT OF INTEREST

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## REFERENCES

- [1] American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)*, 5th ed.; American Psychiatric Association: Arlington, VA, **2013**.
- [2] Phillips, K.A. *The Broken Mirror: Understanding and Treating Body Dysmorphic Disorder (Revised and Expanded Edition)*. New York: Oxford University Press, **2005**.
- [3] Phillips, K.A. Body dysmorphic disorder: clinical aspects and treatment strategies. *Bull. Menninger Clin.*, **1998**, *62*(4)(Suppl. A), A33-A48. [PMID: 9810776]
- [4] Fang, A.; Wilhelm, S. Clinical features, cognitive biases, and treatment of body dysmorphic disorder. *Annu. Rev. Clin. Psychol.*, **2015**, *11*, 187-212. [http://dx.doi.org/10.1146/annurev-clinpsy-032814-112849] [PMID: 25581240]
- [5] Kaplan, R.A.; Rossell, S.L.; Enticott, P.G.; Castle, D.J. Own-body perception in body dysmorphic disorder. *Cogn. Neuropsychiatry*, **2013**, *18*(6), 594-614. [http://dx.doi.org/10.1080/13546805.2012.758878] [PMID: 23441837]
- [6] Phillipou, A.; Castle, D. Body dysmorphic disorder in men. *Aust. Fam. Phys.*, **2015**, *44*(11), 798-801. [PMID: 26590618]
- [7] Weingarden, H.; Renshaw, K.D. Shame in the obsessive compulsive related disorders: a conceptual review. *J. Affect. Disord.*, **2015**, *171*, 74-84. [http://dx.doi.org/10.1016/j.jad.2014.09.010] [PMID: 25299438]
- [8] Kelly, M.M.; Didie, E.R.; Phillips, K.A. Personal and appearance-based rejection sensitivity in body dysmorphic disorder. *Body Image*, **2014**, *11*(3), 260-265. [http://dx.doi.org/10.1016/j.bodyim.2014.03.004] [PMID: 24958661]
- [9] Phillips, K.A.; Pinto, A.; Hart, A.S.; Coles, M.E.; Eisen, J.L.; Menard, W.; Rasmussen, S.A. A comparison of insight in body dysmorphic disorder and obsessive-compulsive disorder. *J. Psychiatr. Res.*, **2012**, *46*(10), 1293-1299. [http://dx.doi.org/10.1016/j.jpsychires.2012.05.016] [PMID: 22819678]
- [10] Phillips, K.A.; Hart, A.S.; Simpson, H.B.; Stein, D.J. Delusional versus nondelusional body dysmorphic disorder: recommendations for DSM-5. *CNS Spectr.*, **2014**, *19*(1), 10-20. [http://dx.doi.org/10.1017/S1092852913000266] [PMID: 23659348]
- [11] Hartmann, A.S.; Thomas, J.J.; Wilson, A.C.; Wilhelm, S. Insight impairment in body image disorders: delusionality and overvalued ideas in anorexia nervosa versus body dysmorphic disorder. *Psychiatry Res.*, **2013**, *210*(3), 1129-1135. [http://dx.doi.org/10.1016/j.psychres.2013.08.010] [PMID: 23992792]
- [12] Phillips, K.A.; Kim, J.M.; Hudson, J.I. Body image disturbance in body dysmorphic disorder and eating disorders. Obsessions or delusions? *Psychiatr. Clin. North Am.*, **1995**, *18*(2), 317-334. [PMID: 7659601]
- [13] Phillips, K.A.; Pinto, A.; Menard, W.; Eisen, J.L.; Mancebo, M.; Rasmussen, S.A. Obsessive-compulsive disorder versus body dysmorphic disorder: a comparison study of two possibly related disorders. *Depress. Anxiety*, **2007**, *24*(6), 399-409. [http://dx.doi.org/10.1002/da.20232] [PMID: 17041935]
- [14] Hollander, E.; Wong, C.M. Body dysmorphic disorder, pathological gambling, and sexual compulsions. *J. Clin. Psychiatry*, **1995**, *56*(Suppl. 4), 7-12. [PMID: 7713866]
- [15] Greenberg, J.L.; Mothi, S.S.; Wilhelm, S. Cognitive-behavioral therapy for body dysmorphic disorder by proxy. *Behav. Ther.*,

- 2016, 47(4), 515-526. [http://dx.doi.org/10.1016/j.beth.2016.01.002] [PMID: 27423167]
- [16] Angelakis, I.; Gooding, P.A.; Panagioti, M. Suicidality in body dysmorphic disorder (BDD): A systematic review with meta-analysis. *Clin. Psychol. Rev.*, **2016**, *49*, 55-66. [http://dx.doi.org/10.1016/j.cpr.2016.08.002] [PMID: 27607741]
- [17] Phillips, K.A.; Coles, M.E.; Menard, W.; Yen, S.; Fay, C.; Weisberg, R.B. Suicidal ideation and suicide attempts in body dysmorphic disorder. *J. Clin. Psychiatry*, **2005**, *66*(6), 717-725. [http://dx.doi.org/10.4088/JCP.v66n0607] [PMID: 15960564]
- [18] Marques, L.; Weingarden, H.M.; Leblanc, N.J.; Wilhelm, S. Treatment utilization and barriers to treatment engagement among people with body dysmorphic symptoms. *J. Psychosom. Res.*, **2011**, *70*(3), 286-293. [http://dx.doi.org/10.1016/j.jpsychores.2010.10.002] [PMID: 21334500]
- [19] IsHak, W.W.; Bolton, M.A.; Bensoussan, J.C.; Dous, G.V.; Nguyen, T.T.; Powell-Hicks, A.L.; Gardner, J.E.; Ponton, K.M. Quality of life in body dysmorphic disorder. *CNS Spectr.*, **2012**, *17*(4), 167-175. [http://dx.doi.org/10.1017/S1092852912000624] [PMID: 22939280]
- [20] Weingarden, H.; Renshaw, K.D.; Wilhelm, S.; Tangney, J.P.; DiMauro, J. Anxiety and shame as risk factors for depression, suicidality, and functional impairment in body dysmorphic disorder and obsessive compulsive disorder. *J. Nerv. Ment. Dis.*, **2016**, *204*(11), 832-839. [http://dx.doi.org/10.1097/NMD.0000000000000498] [PMID: 26998694]
- [21] Gunstad, J.; Phillips, K.A.; Axis, I. Axis I comorbidity in body dysmorphic disorder. *Compr. Psychiatry*, **2003**, *44*(4), 270-276. [http://dx.doi.org/10.1016/S0010-440X(03)00088-9] [PMID: 12923704]
- [22] Mufaddel, A.; Osman, O.T.; Almgaddam, F.; Jafferany, M. A review of body dysmorphic disorder and its presentation in different clinical settings. *Prim. Care Companion CNS Disord.*, **2013**, *15*(4), 15. [PMID: 24392251]
- [23] Vasudeva, S.B.; Hollander, E. Body dysmorphic disorder in patients with autism spectrum disorder: A reflection of increased local processing and self-focus. *Am. J. Psychiatry*, **2017**, *174*(4), 313-316. [http://dx.doi.org/10.1176/appi.ajp.2016.16050559] [PMID: 28366095]
- [24] Veale, D.; Akyüz, E.U.; Hodsoll, J. Prevalence of body dysmorphic disorder on a psychiatric inpatient ward and the value of a screening question. *Psychiatry Res.*, **2015**, *230*(2), 383-386. [http://dx.doi.org/10.1016/j.psychres.2015.09.023] [PMID: 26404769]
- [25] Bohne, A.; Wilhelm, S.; Keuthen, N.J.; Florin, I.; Baer, L.; Jenike, M.A. Prevalence of body dysmorphic disorder in a German college student sample. *Psychiatry Res.*, **2002**, *109*(1), 101-104. [http://dx.doi.org/10.1016/S0165-1781(01)00363-8] [PMID: 11850057]
- [26] Veale, D.; Gledhill, L.J.; Christodoulou, P.; Hodsoll, J. Body dysmorphic disorder in different settings: A systematic review and estimated weighted prevalence. *Body Image*, **2016**, *18*, 168-186. [http://dx.doi.org/10.1016/j.bodyim.2016.07.003] [PMID: 27498379]
- [27] Koran, L.M.; Abujaoude, E.; Large, M.D.; Serpe, R.T. The prevalence of body dysmorphic disorder in the United States adult population. *CNS Spectr.*, **2008**, *13*(4), 316-322. [http://dx.doi.org/10.1017/S1092852900016436] [PMID: 18408651]
- [28] Bjornsson, A.S.; Didie, E.R.; Grant, J.E.; Menard, W.; Stalker, E.; Phillips, K.A. Age at onset and clinical correlates in body dysmorphic disorder. *Compr. Psychiatry*, **2013**, *54*(7), 893-903. [http://dx.doi.org/10.1016/j.comppsy.2013.03.019] [PMID: 23643073]
- [29] Phillips, K.A.; Menard, W.; Quinn, E.; Didie, E.R.; Stout, R.L.A. A 4-year prospective observational follow-up study of course and predictors of course in body dysmorphic disorder. *Psychol. Med.*, **2013**, *43*(5), 1109-1117. [http://dx.doi.org/10.1017/S0033291712001730] [PMID: 23171833]
- [30] Krebs, G.; Fernández de la Cruz, L.; Mataix-Cols, D. Recent advances in understanding and managing body dysmorphic disorder. *Evid. Based Ment. Health*, **2017**, ebmental-2017-102702. [PMID: 28729345]
- [31] Buhlmann, U.; Marques, L.M.; Wilhelm, S. Traumatic experiences in individuals with body dysmorphic disorder. *J. Nerv. Ment. Dis.*, **2012**, *200*(1), 95-98. [http://dx.doi.org/10.1097/NMD.0b013e31823f6775] [PMID: 22210370]
- [32] Buhlmann, U.; Cook, L.M.; Fama, J.M.; Wilhelm, S. Perceived teasing experiences in body dysmorphic disorder. *Body Image*, **2007**, *4*(4), 381-385. [http://dx.doi.org/10.1016/j.bodyim.2007.06.004] [PMID: 18089284]
- [33] Didie, E.R.; Tortolani, C.C.; Pope, C.G.; Menard, W.; Fay, C.; Phillips, K.A. Childhood abuse and neglect in body dysmorphic disorder. *Child Abuse Negl.*, **2006**, *30*(10), 1105-1115. [http://dx.doi.org/10.1016/j.chiabu.2006.03.007] [PMID: 17005251]
- [34] Grace, S.A.; Buchanan, B.G.; Maller, J.J.; Toh, W.L.; Castle, D.J.; Rossell, S.L. Reduced cortical thickness in body dysmorphic disorder. *Psychiatry Res. Neuroimaging*, **2017**, *259*, 25-28. [http://dx.doi.org/10.1016/j.pscychres.2016.11.004] [PMID: 27918913]
- [35] Grace, S.A.; Labuschagne, I.; Kaplan, R.A.; Rossell, S.L. The neurobiology of body dysmorphic disorder: A systematic review and theoretical model. *Neurosci. Biobehav. Rev.*, **2017**, *83*, 83-96. [http://dx.doi.org/10.1016/j.neubiorev.2017.10.003] [PMID: 29017915]
- [36] Feusner, J.D.; Townsend, J.; Bystritsky, A.; Bookheimer, S. Visual information processing of faces in body dysmorphic disorder. *Arch. Gen. Psychiatry*, **2007**, *64*(12), 1417-1425. [http://dx.doi.org/10.1001/archpsyc.64.12.1417] [PMID: 18056550]
- [37] Feusner, J.D.; Moody, T.; Hembacher, E.; Townsend, J.; McKinley, M.; Moller, H.; Bookheimer, S. Abnormalities of visual processing and frontostriatal systems in body dysmorphic disorder. *Arch. Gen. Psychiatry*, **2010**, *67*(2), 197-205. [http://dx.doi.org/10.1001/archgenpsychiatry.2009.190] [PMID: 20124119]
- [38] Feusner, J.D.; Hembacher, E.; Moller, H.; Moody, T.D. Abnormalities of object visual processing in body dysmorphic disorder. *Psychol. Med.*, **2011**, *41*(11), 2385-2397. [http://dx.doi.org/10.1017/S0033291711000572] [PMID: 21557897]
- [39] Moody, T.D.; Sasaki, M.A.; Bohon, C.; Strober, M.A.; Bookheimer, S.Y.; Sheen, C.L.; Feusner, J.D. Functional connectivity for face processing in individuals with body dysmorphic disorder and anorexia nervosa. *Psychol. Med.*, **2015**, *45*(16), 3491-3503. [http://dx.doi.org/10.1017/S0033291715001397] [PMID: 26219399]
- [40] Vulink, N.C.; Planting, R.S.; Figees, M.; Booij, J.; Denys, D. Reduced striatal dopamine D2/3 receptor availability in Body Dysmorphic Disorder. *Eur. Neuropsychopharmacol.*, **2016**, *26*(2), 350-356. [http://dx.doi.org/10.1016/j.euroneuro.2015.11.018] [PMID: 26711686]
- [41] Tasios, K.; Michopoulos, I. Body dysmorphic disorder: Latest neuroanatomical and neuropsychological findings. *Psychiatriki*, **2017**, *28*(3), 242-250. [http://dx.doi.org/10.22365/jpsych.2017.283.242] [PMID: 29072188]
- [42] Grant, J.E.; Fineberg, N.; van Ameringen, M.; Cath, D.; Visser, H.; Carmi, L.; Pallanti, S.; Hollander, E.; van Balkom, A.J.L.M. New treatment models for compulsive disorders. *Eur. Neuropsychopharmacol.*, **2016**, *26*(5), 877-884. [http://dx.doi.org/10.1016/j.euroneuro.2015.11.008] [PMID: 26621260]
- [43] Phillips, K.A. Body Dysmorphic Disorder: Advances in Research and Clinical Practice. Oxford, New York: Oxford University Press, **2017**. [http://dx.doi.org/10.1093/med/9780190254131.001.0001]
- [44] Frías, A.; Palma, C.; Fariols, N.; González, L. Comorbidity between obsessive-compulsive disorder and body dysmorphic disorder: prevalence, explanatory theories, and clinical characterization. *Neuropsychiatr. Dis. Treat.*, **2015**, *11*, 2233-2244. [http://dx.doi.org/10.2147/NDT.S67636] [PMID: 26345330]
- [45] Phillips, K.A. Understanding Body Dysmorphic Disorder. Oxford, New York: Oxford University Press, **2009**.
- [46] Phillips, K.A.; Albertini, R.S.; Siniscalchi, J.M.; Khan, A.; Robinson, M. Effectiveness of pharmacotherapy for body dysmorphic disorder: a chart-review study. *J. Clin. Psychiatry*, **2001**, *62*(9), 721-727. [http://dx.doi.org/10.4088/JCP.v62n0910] [PMID: 11681769]
- [47] Phillips, K.A.; Albertini, R.S.; Rasmussen, S.A. A randomized placebo-controlled trial of fluoxetine in body dysmorphic disorder. *Arch. Gen. Psychiatry*, **2002**, *59*(4), 381-388. [http://dx.doi.org/10.1001/archpsyc.59.4.381] [PMID: 11926939]
- [48] Phillips, K.A.; Kelly, M.M. Suicidality in a placebo-controlled fluoxetine study of body dysmorphic disorder. *Int. Clin. Psychopharmacol.*, **2009**, *24*(1), 26-28. [http://dx.doi.org/10.1097/YIC.0b013e32831db2e9] [PMID: 19060721]
- [49] Phillips, K.A. Pharmacotherapy for body dysmorphic disorder. *Psychiatr. Ann.*, **2010**, *40*(7), 325-332. [http://dx.doi.org/10.3928/00485713-20100701-05] [PMID: 27761054]
- [50] Phillips, K.A.; Hollander, E. Treating body dysmorphic disorder with medication: evidence, misconceptions, and a suggested ap-

- proach. *Body Image*, **2008**, 5(1), 13-27. [http://dx.doi.org/10.1016/j.bodyim.2007.12.003] [PMID: 18325859]
- [51] Phillips, K.A.; Keshaviah, A.; Dougherty, D.D.; Stout, R.L.; Menard, W.; Wilhelm, S. Pharmacotherapy relapse prevention in body dysmorphic disorder: A double-blind, placebo-controlled trial. *Am. J. Psychiatry*, **2016**, 173(9), 887-895. [http://dx.doi.org/10.1176/appi.ajp.2016.15091243] [PMID: 27056606]
- [52] Hollander, E.; Hong, K. Relapse prevention and the need for ongoing pharmacological treatment in body dysmorphic disorder. *Am. J. Psychiatry*, **2016**, 173(9), 857-859. [http://dx.doi.org/10.1176/appi.ajp.2016.16050624] [PMID: 27581691]
- [53] Rashid, H.; Khan, A.A.; Fineberg, N.A. Adjunctive antipsychotic in the treatment of body dysmorphic disorder - A retrospective naturalistic case note study. *Int. J. Psychiatry Clin. Pract.*, **2015**, 19(2), 84-89. [http://dx.doi.org/10.3109/13651501.2014.981546] [PMID: 25363200]
- [54] Marroni, S.S.; Nakano, F.N.; Gati, C.D.C.; Oliveira, J.A.; Antunes-Rodrigues, J.; Garcia-Cairasco, N. Neuroanatomical and cellular substrates of hypergrooming induced by microinjection of oxytocin in central nucleus of amygdala, an experimental model of compulsive behavior. *Mol. Psychiatry*, **2007**, 12(12), 1103-1117. [http://dx.doi.org/10.1038/sj.mp.4002015] [PMID: 17505467]
- [55] Kang, J.I.; Kim, H.W.; Kim, C.H.; Hwang, E.H.; Kim, S.J. Oxytocin receptor gene polymorphisms exert a modulating effect on the onset age in patients with obsessive-compulsive disorder. *Psychoneuroendocrinology*, **2017**, 86, 45-52. [http://dx.doi.org/10.1016/j.psneuen.2017.09.011] [PMID: 28915380]
- [56] Hollander, E. Social synchrony and oxytocin: from behavior to genes to therapeutics. *Am. J. Psychiatry*, **2013**, 170(10), 1086-1089. [http://dx.doi.org/10.1176/appi.ajp.2013.13070848] [PMID: 24084810]
- [57] Park, H.R.; Kim, I.H.; Kang, H.; Lee, D.S.; Kim, B-N.; Kim, D.G.; Paek, S.H. Nucleus accumbens deep brain stimulation for a patient with self-injurious behavior and autism spectrum disorder: functional and structural changes of the brain: report of a case and review of literature. *Acta Neurochir. (Wien)*, **2017**, 159(1), 137-143. [http://dx.doi.org/10.1007/s00701-016-3002-2] [PMID: 27807672]
- [58] Mahato, R.S.; San, G.M.C.P.; Longshore, C.T.; Schnur, D.B. A case of treatment-resistant depression and body dysmorphic disorder: The role of electroconvulsive therapy revisited. *Innov. Clin. Neurosci.*, **2016**, 13(7-8), 37-40. [PMID: 27672487]
- [59] Kumar, S.; Singh, S.; Chadda, R.K.; Verma, R.; Kumar, N. The effect of low-frequency repetitive transcranial magnetic stimulation at orbitofrontal cortex in the treatment of patients with medication-refractory obsessive-compulsive disorder: A retrospective open study. *J. ECT*, **2017**. [http://dx.doi.org/10.1097/YCT.0000000000000462] [PMID: 29053484]
- [60] Carmi, L.; Alyagon, U.; Barnea-Ygael, N.; Zohar, J.; Dar, R.; Zangen, A. Clinical and electrophysiological outcomes of deep TMS over the medial prefrontal and anterior cingulate cortices in OCD patients. *Brain Stimul.*, **2018**, 11(1), 158-165. [http://dx.doi.org/10.1016/j.brs.2017.09.004] [PMID: 28927961]
- [61] Haghghi, M.; Shayganfard, M.; Jahangard, L.; Ahmadpanah, M.; Bajoghli, H.; Pirdehghan, A.; Holsboer-Trachsler, E.; Brand, S. Repetitive Transcranial Magnetic Stimulation (rTMS) improves symptoms and reduces clinical illness in patients suffering from OCD--Results from a single-blind, randomized clinical trial with sham cross-over condition. *J. Psychiatr. Res.*, **2015**, 68, 238-244. [http://dx.doi.org/10.1016/j.jpsychires.2015.06.020] [PMID: 26228425]
- [62] Ma, Z-R.; Shi, L-J. Repetitive transcranial magnetic stimulation (rTMS) augmentation of selective serotonin reuptake inhibitors (SSRIs) for SSRI-resistant obsessive-compulsive disorder (OCD): a meta-analysis of randomized controlled trials. *Int. J. Clin. Exp. Med.*, **2014**, 7(12), 4897-4905. [PMID: 25663986]