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PHARMACOTHERAPY IN MUSCLE DYSMORPHIC DISORDER: CLINICAL CONSIDERATIONS AND THE LIMITS OF EXTRAPOLATING EVIDENCE FROM BODY DYSMORPHIC DISORDER

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ABSTRACT

Background: Muscle dysmorphic disorder is a specifier of body dysmorphic disorder (BDD) characterized by a tenacious belief of insufficient muscularity. Although currently classified within BDD, muscle dysmorphism exhibits distinct clinical and behavioral features, constituting compulsive exercise, rigid dietary practices, and use of performance-enhancing substances. Aforementioned differences raise concerns regarding applicability of pharmacotherapeutic strategies derived from BDD studies to patients with muscle dysmorphism. The aim of the study is to critically evaluate the existing literature on pharmacotherapy in body dysmorphic disorder and assess its clinical applicability to muscle dysmorphic disorder, with considerations of diverging neurobiological, phenomenological, and behavioral distinction limiting direct generalizability.
Methods: A broad literature search was conducted in three major databases: PubMed, Cochrane Library, and Google Scholar, utilizing search terms ("muscle dysmorphism" or "muscle dysmorphic disorder" or "bigorexia nervosa" or "body dysmorphic disorder") and ("pharmacotherapy" or "treatment" or "medication" or "pharmaceutical"). References were screened for relevance, and a total of 20 studies published between 1996 and 2026 were included in the analysis.
Results: This review evaluates and summarizes established pharmacological strategies used in the treatment of BDD and examines their potential relevance for muscle dysmorphic disorder. Selective serotonin reuptake inhibitors (SSRIs) remain the most consistently supported pharmacological intervention, demonstrating meaningful reductions in symptom severity across several studies. Additional treatments, including clomipramine and serotonin–noradrenaline reuptake inhibitors, have shown potential benefits in selected cases. Emerging therapeutic approaches, such as glutamatergic modulators, intranasal oxytocin, and psychedelic-assisted therapy, remain experimental. However, no clinical trials have specifically examined pharmacotherapy in muscle dysmorphic disorder.
Conclusions: Pharmacological strategies developed for BDD may provide partial therapeutic benefit, important differences in clinical presentation and potential neurobiological mechanisms limit direct extrapolation to muscle dysmorphic disorder. Future research should prioritize clinically controlled randomized trials exploring targeted pharmacological treatments for this condition.

KEYWORDS

Muscle Dysmorphism, Bigorexia Nervosa, Body Dysmorphic Disorder, Exercise Addiction Disorder, Eating Disorder, Pharmacotherapy

CITATION

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Introduction:

Muscle dysmorphic disorder is a specifier within body dysmorphic disorder (BDD) characterized by a pathological conviction of having insufficient muscle mass or being too small despite pronounced muscularity. Although the condition has been primarily associated with bodybuilders, concerns arise with the increasing number of patients among adolescents. (Burmeister, 2025) In the literature, this disorder can be sometimes referred to as bigorexia nervosa, reverse anorexia or "Adonis complex". (Vasiliu, 2023) Diagnostic criteria of BDD according to DSM-5 are as follows: 1. Preoccupation with one or more perceived flaws in physical appearance, which are not of significant notice to others; 2. Repetitive behaviors; 3. The preoccupation causes significant distress or affects at least one major life area; 4. Symptoms cannot be attributed to an eating disorder. (Nicewicz et al., 2025)

However, muscle dysmorphism presents several distinguishing characteristics such as continuous rumination over insufficient muscle mass, which can convert into obsessions as well as over-exercising, substance use disorders, overindulgence in supplements and dysfunctional eating patterns. Identification of traits associated with muscle dysmorphism requires screening for comorbidities, most commonly including anabolic-androgenic steroid use disorder, behavioral addictions, substance use disorders, physical exercise addictions, mood disturbances. (Vasiliu, 2023) (Trichas et al., 2025) This BDD specifier is characterized by a significantly lower quality of life and increased suicidality. (Pope et al., 2005) Bigorexia nervosa shares many features with anorexia, e.g. functional impairment, pathological preoccupation with body shape or weight, and distress over appearance. However, current nosology classifies it within BDDs. (Vasiliu, 2023) Its etiology has not been clearly established, but theories suggest the involvement of genetic, physiological, psychological and socioenvironmental factors. (Nagata et al., 2026) Potentially problematic trends have emerged with its classification as only male disorder. Epidemiological data should be investigated in terms of gender distribution, comorbidity with substance abuse and other disorders, to further aid in establishing potential risk factors. (Vasiliu, 2023)

Current evidence indicates cognitive-behavioral therapy (CBT) in the treatment of muscle dysmorphism. Performed systematic reviews show the efficacy in improving body image, alleviating symptoms and meliorating global functioning. However, the study failed to demonstrate statistically significant changes in insight, depression and quality of life. (Abdalla et al., 2026) Response rates with CBT come up to 71,9%. Both in person and online delivered strategies yielded satisfactory results. However, remote treatment option can be complicated by more adverse effects and higher dropout rate. (Byrne et al., 2026) Although aforementioned meta-analyses show the efficacy of the psychotherapeutic approaches in the treatment of bigorexia nervosa, a significant portion of patients remain unresponsive. The need to establish other treatment strategies arises. So far no clinical trials testing the efficacy of pharmacotherapy in muscle dysmorphic disorder has been conducted. This significantly limits the potential therapeutical impact of clinicians' interventions. The need for further research in this area desperately arises with the notably increasing prevalence. Bigorexia nervosa deviates from BDD. Differences stem from male predominance, over-exercising, substance abuse, possible reward-system involvement, greater symptom overlap with addiction disorders and OCD. However, its obsessive preoccupation, compulsive pattern of behaviors classifies it nosologically with body dysmorphic disorder. Pharmacological trials have been conducted in the treatment of latter. Hence, following manuscript evaluates and summarizes established pharmacological strategies and their efficacy in body dysmorphic disorder, compares clinical and behavioral phenotype of muscle dysmorphic disorder with classical BDD, examines the neurobiological rationale for various treatment options and identifies limitations in extrapolating date derived from BDD trials to muscle dysmorphic disorder, accentuating priorities for future research.

Methods:

A large literature search was conducted with the utilization of 3 major databases: Pubmed https://pubmed.ncbi.nlm.nih.gov/(accessed on: 12th of February 2026), Cochrane https://www.cochrane.org/ (accessed on: 12th of February 2026) and Google scholar https://scholar.google.com/ (accessed on: 12th of February 2026). The following keywords were included in the search ("muscle dysmorphic disorder" OR "muscle dysmorphism" OR "bigorexia" OR "body dysmorphic disorder") AND ("pharmacotherapy" OR "treatment" OR "medication" OR "pharmaceutical"). References were manually screened for relevance. 20 studies published between 1996 and 2026 have been included in the following review.

Results:

Selective Serotonin Reuptake Inhibitor

Fluoxetine

Fluoxetine is a selective serotonin reuptake inhibitor with a broad variety of uses within psychiatric conditions. It should be noted that its half-life is significantly longer than most within that group, clinicians should exhibit caution when discontinuing or starting new medications. Hepatic insufficiency decreases elimination of both fluoxetine and its active metabolite norfluoxetine, warranting a less frequent or smaller dosing. (Sohel AJ et al., 2024) Maximum dose should not exceed 120mg/day. (Castle et al., 2021)

Double-blind placebo-controlled trial tested the effect of fluoxetine in the treatment of BDD. Over a 12 week period patients were randomly allocated into two groups – one receiving the medication and the other placebo. Participants were initially given a dose of 20 mg/day and after 10 days a dose was gradually increased by 20 mg/day to a maximum of 80 mg/day. Depending on the personal tolerance, a dose could be decreased by 10 mg/day or 20 mg/day as clinically indicated. Outcomes were scrutinized with the use of BDD-YBOCS scale. Fluoxetine yielded a much higher response rate (53%) in comparison to placebo (18%), measured by a decrease in BDD-YBOCS by at least 30%. It proved effective in alleviating symptoms in both delusional and non-delusional variants. Most common side effects included: insomnia, headache, abdominal discomfort, nausea, tremor, decreased libido and memory impairment. (Phillips et al., 2002) (Phillipou et al., 2016)

It should also be noted that 5-HT2C antagonism might contribute towards anxiety, agitation and sleeplessness. Due to CYP2D6 inhibition it affects the metabolism of many psychotropic medications, hence augmentation should be performed with utmost care. (Sohel AJ et al., 2024)

Fluvoxamine

Fluvoxamine can be indicated in obsessive-compulsive disorders, anxiety, depression, PTSD. Out of all SSRIs it has a higher affinity for sigma 1 (σ-1) receptor, associated with psychosis and aggression. Its stimulation might reduce cytokine production, hence decreasing inflammation. Hepatic impairment require a decrease in dosing, whilst renal insufficiency does not seem to affect its pharmacokinetics. Some of most common adverse effects include: insomnia, headache, asthenia, and diarrhea. Less frequent but clinically significant constitute platelet dysfunction and hyponatremia. Fluvoxamine interacts with several cytochrome P450 enzymes with a potent inhibition of CYP1A2 and CYP2C19, moderate inhibition of CYP3A4, and a weak inhibition of CYP2C9 and CYP2D6. (Hoffman R & Parmar M, 2025) Maximum dose should not exceed 450mg/day. (Castle et al., 2021) Fluvoxamine has shown a positive effect in reducing BDD symptoms in 30 patients with both non- and delusional variant. Participants were assessed with BDD-YBOCS initially and after 16 weeks. Scores dropped from the baseline 31.1± 5.4 to 16.9±11.8 at the end. The mean dose assigned measures 238 mg/day±85 mg/day, and an average time to respond equals 6.1(±3.7) weeks. (Phillips et al., 1998). In one study 400 mg fluvoxamine augmented with 10 mg aripiprazole and proved effective in treatment-resistant BDD. (Uzun & Özdemir, 2010)

Escitalopram

Escitalopram has been tested in both short term and long term treatment of BDD. In weeks 1-3 participants received a dose of 10 mg/day, in weeks 4-6 20 mg/day and in weeks 7-14 30 mg/day. In case of an intolerance dosing was adjusted. Mental state of patients was assessed in regular intervals, weekly during the first month and biweekly thereafter. A reduction of ≥30% in BDD-YBOCS score was achieved by 67% of participants. The mean time to respond measured 7.9 weeks and the mean escitalopram dose at the end of 14 weeks was 26.2±7.2 mg/day with a range of 5-30 mg/day. The experiment continued for another 6 months, with responders randomly allocated into two groups - first continuing escitalopram monotherapy and second gradually discontinuing it and substituting with the placebo. In the control group medication was slowly tapered by 10 mg/day a week to avoid potential side effects. Biweekly psychiatric assessments took place to monitor patients. Relapse occurred in 40% of patients receiving the placebo and only 18% taking escitalopram. In the group with prolonged escitalopram therapy BDD-YBOCS scores decreased further by an average of 4.1 points. The mean escitalopram dose at the end of 40 weeks equaled 28.7 ± 4.6 mg/day with a range of 7.5–30.0 mg/day. (Phillips et al., 2016) One of the identified predictor for escitalopram response in the therapy of BDD is baseline neuroticism.(Fang A. et al., 2019) Hence, patient exhibiting such personality trait should be potentially considered for a different treatment option. It should be brough to our attention that the maximum dose should not exceed 60 mg/day. Current recommendation is to acquire an ECG when a dose surpasses 20 mg/day. (Castle et al., 2021) Common side effects of escitalopram included: fatigue, nausea, sexual dysfunction, dry mouth, headache, appetite changes, sleeplessness, hypersomnia, agitation, sweating. No serious adverse reaction were noted during the course of the study. Findings of this study mimic those from the literature published thus far with SSRI proving effective in the treatment of BDD. Protracted-therapy seems beneficial in relapse-prevention. (Phillips et al., 2016)

Citalopram

Citalopram is a SSRI mainly indicated in the treatment of depression. It has been found to attenuate the severity of BDD. Patients received a mean dose of 51.3±16.9 mg/day. Their symptoms were regularly assessed with BDD-YBOCS over a course of 12 weeks. Initial and final scores measured subsequently 30.7±4.9 and 15.3±10.6. Mean time to respond equaled 4.6±2.6 weeks. (Phillips & Najjar, 2003)

Maximum dose should not exceed 40mg/day. (Castle et al., 2021) Concern arise that increasing the dose warrants a risk of QT prolongation. Obtaining an electrocardiogram and examining electrolyte levels prior to implementing therapy is recommended. Moreover, dose adjustment should be considered in patients experiencing hepatic impairment. Paradoxically, initial response to treatment can be worsening of depressive symptoms and suicidality. Monitoring patient's wellbeing on the regular might prove beneficial.(Sharbaf Shoar N et al., 2023)

Paroxetine

No clinically controlled trials have been conducted researching the efficacy of paroxetine on body dysmorphic disorder. Basing on aforementioned research showing positive results with SSRIs, it could be assumed that it may prove beneficial. One case report disclose the lack of improvement upon implementing 40 mg/day monotherapy in a 26-year old woman with a 10 year history of BDD. (Nakaaki et al., 2008) However, systematic reviews and meta analyses prove its effectiveness in OCD. (Varigonda et al., 2016) Starting dose begins at 20 mg/day daily with weekly increases up to 10 mg/day depending on tolerance. Dosing should not exceed 60 mg/day. Side effects profile is very similar to that of other SSRIs. However, discontinuation syndrome is more common and more severe in paroxetine with withdrawal characterized by fever, chills, lethargy, nausea and vomiting, anxiety, dyskinesia and depersonalization among others. (Shrestha P et al., 2023) Hence caution should be implemented when dosing and discontinuing paroxetine. Nevertheless, more studies need to be conducted in order to establish its effectiveness in treating body dysmorphism.

Tricyclic Antidepressants

Clomipramine

Clomipramine is a tricyclic antidepressant (TCA) characterized by a potent serotonin reuptake inhibition. Treatment indications include OCD, with some evidence suggesting its beneficial role in depression, anxiety, chronic pain and other disorders. (Wilson M & Tripp J, 2024)

One study examined the effect of intravenous pulses in delusional variant of BDD. Clomipramine was subsequently dosed 150 mg on day 1 and 200 mg on day 2, and thereafter an oral treatment was continued over the course of 2 months. 2 patients exhibited a reduction of symptoms, noted in the decline in BDD-YBOCS scores only 4,5 days after an initial dose. Authors theorize that intravenous pulse-dosing may prove beneficial in cases where immediate treatment is required (Pallanti & Koran, 1996).

A double-blind control trial has been conducted on 40 individuals with BDD, in which researches compared the efficacy of clomipramine and desipramine (selective noradrenaline reuptake inhibitor) over 16 weeks. Doses of clomipramine were carefully titrated from an initial 25mg/day to a maximum of 250mg/day, considering tolerance and severity of symptoms. Clomipramine resulted in a higher reduction in repetitive behaviors and perceived defects. Curiously, it yielded better outcomes in patients with delusional variant. (Hollander et al., 1999)

Clomipramine exhibits antihistaminic and antimuscarinic side effects, such as dry-mouth, weight gain, sleepiness and orthostatic hypotension. Low therapeutic index and an increasing risk seizure prevents from increasing the dose. (Castle et al., 2021) Physicians should utilize caution, considering potential cardiac complications such as QT prolongation, tachycardia, arrythmias. Electrocardiogram is recommended both prior to initiating the treatment and after reaching a desired dose. Vigilant monitoring of patient should be conducted as clomipramine is also linked to increased risk of suicidality. (Wilson M & Tripp J, 2024)

Serotonin Noradrenaline Reuptake Inhibitor

Venlafaxine

Venlafaxine increases both serotonin and norepinephrine available in the synaptic cleft. Its effectiveness in the therapy of BDD has been examined over a course of 12 to 16 weeks. Severity of symptoms was surveyed at baseline, every 2 weeks over the first month, and monthly thereafter. Yale Brown Obsessive Compulsive Scale Modified for BDD (BDD-YBOCS) was utilized in the scoring system. Participants begun the trial with a dose of 37.5 mg/day, which was increased generally over a month to at least 150 mg/day, and subsequently maintained for 8 weeks. Dosage was carefully customized to each patient with consideration of the presence of symptoms and medication side effects, with the final doses ranging from 150 to 225 mg/day and mean ending dose of 164±30.34 mg/day. In 11 participants who completed the study severity of symptoms lessened.

In psychiatrist's evaluation BDD-YBOCS scores decreased from baseline 34.64±5.09 to endpoint 26.55+11.28 (p=0.01). Findings show the response rate of 64%, with 7 out of 11 completers reporting an alleviation of symptoms. No significant adverse effects of the medication has been noted in this study. It should be noted that the use of venlafaxine carries a lesser risk and more favorable side effect profile in comparison to clomipramine. As it comes with no anticholinergic, alfa-adrenergic and antihistaminic effects. Small sample size with a majority of female participants may not make the outcome applicable in the therapy of muscle dysmorphism. To establish the effectiveness of venlafaxine in its treatment more trials should be conducted. (Allen et al., 2008)

Venlafaxine presents with a lower incidence of sedation, anticholinergic and cardiovascular side effects as compared to TCAs. However, sleep impairment, sexual dysfunction and gastrointestinal disturbances are more common. Appropriate monitoring should be performed as its use can be associated with suicidal behaviors. (Singh D & Saadabadi A, 2024)

Duloxetine

Another representative of serotonin-norepinephrine reuptake inhibitor group is duloxetine. As of 2026 no randomized trials have been conducted utilizing it in the treatment of body dysmorphic disorder. However, considering the effectiveness of venlafaxine in cases of SSRI resistance or intolerance, duloxetine could also be implemented, especially in cases of patients with co-existing neuropathy, fibromyalgia, musculoskeletal pain, depression or anxiety. (Jaberpreet S. Dhaliwal et al., 2023)

Psychedelics

Psilocybin

Partial serotonin 1A and 2A agonist (5-HT1A, 5-HT2A) with known psychedelic properties has been shown some promise in the therapy of some psychiatric maladies such as obsessive-compulsive disorder and depression. (Yao et al., 2024) Schneier et al. (2023) investigated the use of psilocybin in the treatment of moderate to severe body dysmorphic disorder (BDD). (Schneier et al., 2023) Direct therapeutic pathway is not fully identified as of yet. However, studies have shown post-administration changes in both the severity of symptoms and neural imagining. Participants diagnosed with moderate to severe non-delusional body dysmorphic disorder for over 6 months priorly resistant to SSRI, SNRI or clomipramine, measuring or equal to ≥20 mg/day fluoxetine for at least 2 months were given a single dose of 25 mg psilocybin orally and provided with psychological support. Symptoms were evaluated on the Yale Brown Obsessive Compulsive Scale Modified for BDD (BDD-YBOCS) a day before the dosing, at the end of the day (0 day), one day after the dosing (day 1), and post-dosing weeks 1, 2, 3, 6, 9, and 12. Significant decline in symptoms has been observed from the baseline to week 12 with the response rate of 7 (58.33%) out of 12 participants exhibiting >30% decrease in BDD-YBOCS score or Clinical Global Impression (CGI) Change scale of 1 (very much improved) or 2 (much improved). Zhu et al. (2025) replicated the study with an identical design with all 12 participants presenting the decline in BDD-YBOCS scores in week 1 and week 12 assessments. Those results imply psilocybin's effectiveness in diminishing symptoms of BDD. Findings of these studies should be interpreted with an appropriate reservations as the small sample size and the lack of control group limit its generalizability. Future research should investigate the effects of different doses and focus on expanding upon the single-dose design to further our comprehension of the mechanisms in which psilocybin takes effect and its long-term durability. (Schneier et al., 2023) (Zhu et al., 2024) However, the incidence of adverse reactions should be noted. A minority of participants reported mild complications such as fatigue, headache, nausea, light-headedness, insomnia, spaciness have occurred and resolved within 2 days. One participant detailed severe adverse reactions in the form of decreased libido that started in the 1st week and persisted for 2 weeks, mood disturbances in the 2nd and 3rd week after dosing, 4 episodes of visual hallucinations, 2 in 3rd week and 2 occurring in 7th week post-dosing. Patient admitted that hallucinations lasted from 5 to 45 minutes, participant's judgement of their illusory nature was preserved and episodes were not considered as overly distressing. (Schneier et al., 2023) Overall, single-dose psilocybin appears effective and relatively safe in the treatment of BDD. Although its efficacy in lesser to that of SRI (70-80%) in uncontrolled trials. (Castle et al., 2021) Utilization of 5-HT1A and 5-HT2A agonists should be therefore considered in cases of SRI- and SNI-intolerance of resistance.

Anticonvulsants

Levetiracepam

Levetiracetam is an antiepileptic drug ordinarily used in the adjunctive treatment of focal, myoclonic and primary generalized seizures. It's mechanism of action is assigned to binding to a synaptic vesicle protein 2A (SV2A), a part of secretory vesicle membrane responsible for calcium-dependent neurotransmitter release. As the result vesicular release decreases. (Kumar A et al., 2023) It also enhances GABA-ergic transmission via negative allosteric zinc and βcarbolines antagonism on GABA-A receptors. In one trial it appeared effective in the symptoms of social anxiety, which are often present within individuals burdened with BDD. 17 patients received an initial dose of 250 mg/day in the first week, in the second week 250 mg/day was given two times a day, further the dosing was increased by 500mg/day each week to a maximum of 1500 mg twice a day. Participants were evaluated with the BDD-YBOCS at the beginning, in 1 week increments for one month, and biweekly thereafter for a cumulative time of 12 weeks. In 11 patients who finished a full course of treatment BDD-YBOCS scores decreased from 32.9 ± 5.1 to 19.6 ± 11.8. Mild adverse effects which can perhaps be attributed to the implemented treatment in approximately 10% of participants and those included: fatigue, irritability, sleep disturbances, headache, anxiety, nausea and dizziness. 1 patient experienced severe adverse reaction in the form of increased suicidality requiring hospitalization. Notably, for a variety of reasons, high dropout rate has been observed in this study. BDD patients' response to levetiracetam appears to be lower than that of SSRIs in previously aforementioned studies. However, caution should be implemented when comparing studies due to small sample sizes utilized and the fact that 16 out of 17 participants have undergone SRI trial in the past with minimal effects, marking them as treatment-resistant. The lack of placebo control makes it impossible to eliminate placebo effect. Placebo-controlled trials should be further performed in order to thoroughly examine the effectiveness of levetiracetam in BDD. (Phillips & Menard, 2009)

Oxitocin

One trial examined the impact of intranasal oxytocin to brain functionality in body dysmorphic disorder. Single intranasal dose of 24 IU was administered. Researchers reveled the functional changes in the central nervous system in the areas that could theoretically contribute to diminishing body-repetitive behaviors and aiding in the shift from pathological focus on 'self' to 'other'. (Grace et al., 2019) However, randomized controlled trials with a broader sample evaluating the appropriate dosing regimen, impact on the quality of life, protracted therapy and relapse potential need to be performed.

Sylimarin

Sylimarin otherwise known as milk thistle extract is theorized to affect OCD-related disorders. Novel trial is reported to examine its effects in body dysmorphic disorder. As of march 2026 the results of that study remain unpublished. (Dong et al., 2019)

Memantine

Memantine is an anti-glutamatergic drug usually utilized in the treatment of Alzheimer's disease. NMDA antagonism with consequent increase in striatal dopamine, could prove beneficial in body dysmorphism. (Dong et al., 2019) One study examined the effects of daily dose of 10-40 mg/day in 5 female patients over a 12 week period. BDD-YBOCS was utilized from baseline do the end of the trial. With the initial scores equal to 57.67+/-0.58 and final results being 48.67+/-6.51. It shall be noted that only 1 participant finished a full 12 week course of treatment, 2 people withdrew, 1 did not participate in the follow-up and 1 exhibited lack of efficacy. No serious adverse reactions were noted: 1 participant reported mild reactions in the form of headache, cough, eye-soreness and constipation over the course of the trial. Small sample size make it impossible to generalize the findings. More trials with a broader and larger group examined need to be conducted. (James I. Hudson, 2015)

Discussion:

Pharmacotherapy of body dysmorphic disorder focuses on influencing serotonergic neurotransmission, mainly via selective serotonin reuptake inhibitors (SSRIs). Across published literature this class of medication proves effective in reducing symptoms such as repetitive appearance-related behaviors. obsessions over perceived flaws and associated distress. Documented response rates range between 53% to 73% approximately, with several trials proving effective in both delusional and non-delusional variants of BDD. (Phillips et al., 1998)(Phillips et al., 2002) (Phillips & Najjar, 2003) (Castle et al., 2021) Moreover, protracted treatment might be crucial in relapse prevention, highlighting the need for long-term serotonergic modulation in symptom control. (Phillips et al., 2016)

In the SSRIs group fluoxetine, fluvoxamine, escitalopram and citalopram are some amongst the most studied in the therapy of BDD. Significant symptom reduction has been observed, particularly in controlled open-label trials involving fluoxetine and fluvoxamine. (Phillips et al., 1998) (Phillips et al., 2002) Escitalopram appears promising in long-term relapse prevention with extended-period pharmacotherapy. (Phillips et al., 2016) Evidence regarding paroxetine remains limited with its potential benefit being extrapolated from research showing its efficacy in obsessive-compulsive disorder rather than direct clinical studies in patients with BDD. (Nakaaki et al., 2008) (Varigonda et al., 2016)

Clomipramine, a representative of tricyclic antidepressant group appears to be effective in reducing repetitive behaviors and obsessive thoughts associated with BDD. Its efficacy is shown even in delusional variants. (Hollander et al., 1999) (Pallanti & Koran, 1996) Its less favorable adverse effect profile with an increased risk of anticholinergic and cardiovascular complications. Therefore, clomipramine is ordinarily considered as a second line of treatment, whenever SSRIs prove ineffective or patient does not tolerate them. (Castle et al., 2021) (Wilson M & Tripp J, 2024)

Serotonin-noradrenaline reuptake inhibitors (SNRIs) can also be regarded as an alternative to SSRIs. Trials examining venlafaxine, report significant reduction in symptom severity, yet small sample size limit the generalizability of the evidence. (Allen et al., 2008) Duloxetine has not been studied in patients with BDD. It can be theorized that in cases of comorbid depression, anxiety, or chronic pain conditions in might prove beneficial. (Jaberpreet S. Dhaliwal et al., 2023)

Recently, several experimental pharmacological trials have been conducted. Use of psychedelic substances such as psilocybin started garnering increasing attention in psychiatry due to potential effects on cognitive flexibility, emotional processing, and neural activity. (Yao et al., 2024) Pilot studies investigated psilocybin in moderate to severe BDD, reporting reduction of symptom severity after a single dose followed with supplementary psychological support.(Schneier et al., 2023) (Zhu et al., 2024) Findings of these studies should be interpreted cautiously, considering lack of placebo control, small sample size and limited duration of follow-up.

Other novel approaches consider anticonvulsants such as levetiracetam, neuropeptide interventions via intranasal oxytocin, and glutamatergic modulation via memantine. Available evidence in support of these strategies remains preliminary and is based on small pilot studies. Therefore, its role and clinical application is uncertain. (Phillips & Menard, 2009) (Grace et al., 2019) (James I. Hudson, 2015)

Although pharmacotherapy has been demonstrated as an effective strategy in the treatment of body dysmorphic disorder, applying the same findings to muscle dysmorphic disorder pose several challenges. While current diagnostic framework categorize muscle dysmorphism as a specifier within BDD, its clinical presentation varies significantly. Behaviors commonly exhibited by patients include rigid dietary control, compulsive exercise, and use of performance-enhancing substances, commonly anabolic-androgenic steroids. (Vasiliu, 2023) (Trichas et al., 2025) Those might imply a stronger overlap with addictive and compulsive behavioral disorders than classical appearance-focused BDD.

Aforementioned distinctions might indicate potentially differing underlying mechanisms from a neurobiological standpoint. Traditionally BDD reflects serotonergic dysregulation and abnormal processing within frontostriatal circuits associated with compulsive behaviors and obsessive thoughts. (Castle et al., 2021) Conversely, positive-reinforcement and addiction-like behaviors might indicate the involvement of additional dopaminergic pathways and dysregulation of reward-related neural systems. (Nagata et al., 2026)

Direct extrapolation of pharmacotherapeutic findings from BDD to muscle dysmorphism is complicated by unique demographic composition of existing research samples. Many BDD trials involved predominantly female participants, whilst muscle dysmorphism is most commonly reported among males, especially within athletic and bodybuilding groups. (Vasiliu, 2023) (Burmeister, 2025) Masculine body ideals within sociocultural pressures may influence both the development and potential treatment response.

Additionally, muscle dysmorphism is commonly comorbid with other psychiatric and behavioral issues, such as disordered pattern of eating, substance use disorders, mood disturbances. (Vasiliu, 2023) (Trichas et al., 2025) Existing comorbidities might affect treatment outcomes and therefore suggest that holistic treatment strategies addressing several psychopathologies might prove necessary.

Overall, the current literature suggests that medications involved in serotonergic modulation might alleviate certain symptoms, such as obsessive rumination and anxiety. However, treatment strategies should not be directly inferred from only BDD studies. As of 2026, no randomized controlled trials investigating pharmacotherapy in patients with muscle dysmorphic disorder have been performed.

Future research should prioritize clinical trials focusing specifically on muscle dysmorphism. Strategies examined should encompass both serotonergic modulation and affecting reward-related neural pathways, especially in patients exhibiting compulsion to exercise and substance abuse. Further examining the neurobiological mechanisms involved in muscle dysmorphic disorder may ultimately facilitate developing targeted and effective therapeutic strategies.

Conclusions:

Muscle dysmorphic disorder constitutes a psychiatric condition associated with clinically significant impairment in functioning, substantial psychological distress and increased risk of suicidality. (Pope et al., 2005) Although the current classification marks it as a specifier within body dysmorphic disorder, notable differences in behavioral presentation, demographic composition, and potential neurobiological mechanism distinguish it from classical BDD. (Vasiliu, 2023) (Nagata et al., 2026)

Pharmacotherapy of body dysmorphic disorder explored a vast group of medications affecting serotonergic transmission, particularly selective serotonin reuptake inhibitors, which are approved as a first line therapeutic option. (Phillips et al., 2002) (Phillips et al., 2016) (Castle et al., 2021) In selected cases, additional treatment options such as clomipramine and serotonin-noradrenaline reuptake inhibitors can be considered. (Allen et al., 2008) Novel approaches including use of psychedelic therapy remains experimental. (Zhu et al., 2024) (Schneier et al., 2023)

The lack of clinical trials targeting pharmacotherapy in muscle dysmorphic disorder directly present a significant gap in the existing literature. Considering distinctive behavioral patterns associated with muscle dysmorphism, e.g. compulsive exercise, rigid dietary practices, frequent substance abuse, treatment strategies derived from BDD trials may not fully encompass the complexity of this disorder. (Vasiliu, 2023) (Trichas et al., 2025)

Future research should investigate evidence-based pharmacotherapeutic interventions addressing muscle dysmorphic disorder specifically. Further understanding of neurobiological mechanism and clinical consequences will be crucial in the development of targeted treatment strategies and improving outcomes for affected individuals.

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