

Neural Mechanisms and Cognitive-Behavioral Interventions in Body Dysmorphic Disorder: A Neurobehavioral Perspective

Abstract

Body Dysmorphic Disorder (BDD) is characterized by obsessive preoccupation with perceived physical flaws, often unnoticed by others. This preoccupation causes significant emotional distress, social withdrawal, and functional impairment. Recent advancements emphasize the importance of integrating neurobiological and behavioral perspectives in understanding BDD. This review synthesizes findings from neuroscience and behavioral psychology to examine the brain-behavior relationship in BDD and explores implications for treatment strategies. A narrative synthesis of current literature, including neuroimaging, neurochemical studies, and behavioral research, was conducted. Evidence from clinical trials on pharmacological treatments, cognitive-behavioral therapy, and neuromodulation techniques was analyzed. Neuroimaging studies reveal hyperactivity in the orbitofrontal cortex, amygdala, and fusiform gyrus, contributing to obsessive thoughts, heightened emotional responses, and distorted visual processing. Neurochemical imbalances, particularly in serotonin, dopamine, and gamma-aminobutyric acid systems, perpetuate cognitive distortions and compulsive behaviors. Cognitive patterns, including selective attention to perceived flaws, catastrophic thinking, and overestimation of others' judgments, interact with maladaptive behaviors such as mirror checking, avoidance, and reassurance-seeking. Integrated treatments, including selective serotonin reuptake inhibitors, cognitive-behavioral therapy, and transcranial magnetic stimulation, address these interconnected mechanisms effectively. BDD arises from a complex interaction between neurobiological dysfunction, cognitive distortions, and maladaptive behaviors. Effective treatment requires a multidisciplinary approach targeting these domains. Future research should focus on longitudinal brain plasticity studies, sex differences, and optimized neuromodulation protocols to enhance therapeutic outcomes and personalized interventions.

Keywords: Body Dysmorphic Disorder, Neurobehavioral Perspective, Cognitive Distortions, Neuroimaging, Treatment Approaches.

Introduction

Body Dysmorphic Disorder (BDD) is a psychiatric condition characterized by an obsessive preoccupation with perceived flaws or defects in one's physical appearance, which are often imperceptible or trivial to others. [1] This persistent concern leads to significant emotional distress and impairment in social, occupational, and daily functioning. [2] While the preoccupation can center on any part of the body, common focal points include the skin, hair, nose, and overall facial symmetry [3].

Individuals with BDD frequently engage in repetitive and ritualistic behaviors aimed at examining, hiding, or correcting the perceived defect [4]. These behaviors include excessive mirror-checking, skin-picking, grooming, seeking reassurance from others, or undergoing unnecessary cosmetic procedures [5]. Despite these efforts, relief is often temporary, and the cycle of obsession and compulsion continues, reinforcing the disorder's chronic nature [6].

BDD typically emerges during adolescence, a critical developmental period marked by heightened self-awareness and societal pressures around physical appearance [7]. If left untreated, the condition often persists into adulthood, significantly affecting quality of life

and increasing the risk of comorbidities such as depression, anxiety disorders, substance abuse, and suicidal ideation [8].

From a neurobehavioral perspective, BDD is believed to result from a complex interplay between genetic predisposition, neurobiological factors, cognitive distortions, and environmental influences [9]. Neuroimaging studies have revealed abnormalities in brain regions responsible for visual processing, emotional regulation, and executive functioning, such as the orbitofrontal cortex, amygdala, and fusiform gyrus [10]. These findings suggest that individuals with BDD may have a distorted perception of their physical appearance at both a cognitive and neural level [11].

Understanding BDD through a neurobehavioral lens allows for a deeper examination of how brain function and behavioral patterns interact to perpetuate the disorder. This perspective offers valuable insights into developing more targeted and effective therapeutic interventions, bridging the gap between neuroscience and clinical practice.

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The Growing Importance of Neurobehavioral Perspectives in Understanding BDD

In recent years, the study of BDD has shifted from purely psychological and sociocultural explanations to an integrated neurobehavioral perspective^[12]. This approach combines insights from neuroscience and behavioral psychology to offer a more comprehensive understanding of the disorder's underlying mechanisms.

Traditional views of BDD focused primarily on cognitive distortions, low self-esteem, and societal pressures related to physical appearance^[13]. While these factors remain critical, advancements in neuroimaging technologies—such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET)—have revealed structural and functional abnormalities in specific brain regions associated with visual processing, emotional regulation, and self-referential thinking^[14]. These findings suggest that individuals with BDD may perceive and process visual information, particularly related to their appearance, differently from individuals without the disorder^[15].

At a behavioral level, the repetitive and ritualistic actions often seen in BDD, such as excessive mirror-checking, reassurance-seeking, and grooming, are now understood as maladaptive coping mechanisms driven by underlying neurocognitive dysfunction^[16]. For instance, heightened activity in the orbitofrontal cortex (OFC) and amygdala has been linked to obsessive thoughts and amplified emotional responses to perceived flaws^[17]. Meanwhile, dysfunction in the fusiform gyrus, a brain region involved in facial recognition and detail processing, may explain why individuals with BDD fixate on minor imperfections^[18].

The neurobehavioral perspective also emphasizes the feedback loop between thought, emotion, and action^[19]. For example, distorted visual processing triggers obsessive thoughts, which in turn provoke anxiety and drive compulsive behaviors aimed at mitigating distress^[20]. These behaviors, however, reinforce the brain's maladaptive patterns, perpetuating the cycle of BDD^[21].

Understanding BDD through a neurobehavioral framework highlights the importance of integrated treatment approaches that address both cognitive-behavioral patterns and underlying neurobiological dysfunction. Treatments such as Cognitive-Behavioral Therapy (CBT) combined with pharmacological interventions (e.g., SSRIs) or emerging techniques like Transcranial Magnetic Stimulation (TMS) represent promising avenues rooted in this growing perspective^[22].

By bridging the gap between brain function and observable behaviors, the neurobehavioral approach not only advances scientific understanding but also opens pathways for personalized treatment strategies, ultimately improving outcomes for individuals with BDD.

Bridging Neurobiology and Behavior in BDD

The growing body of research on BDD highlights the necessity of an integrated neurobehavioral perspective to fully understand the disorder's origins, maintenance, and treatment^[23]. While earlier studies primarily focused on cognitive distortions and sociocultural influences, emerging evidence underscores the critical role of neurological mechanisms in shaping the

obsessive thoughts and compulsive behaviors characteristic of the disorder. Advances in neuroimaging techniques have revealed significant structural and functional abnormalities in brain regions responsible for visual processing, emotional regulation, and executive functioning. These insights have reshaped the understanding of how distorted body image perceptions are not merely psychological phenomena but deeply rooted in neurobiological dysfunction^[24].

At the same time, behavioral research has provided a clearer understanding of how maladaptive coping mechanisms, such as mirror checking, avoidance behaviors, and repetitive grooming, perpetuate the disorder^[25]. These behaviors are not random but represent attempts to alleviate the intense distress caused by distorted self-perceptions. However, they ultimately reinforce the brain's maladaptive patterns, creating a self-sustaining feedback loop between thought, emotion, and action. This bidirectional relationship between brain function and behavior is central to the neurobehavioral perspective, offering a more dynamic and integrative explanation of how BDD manifests and persists^[26].

Understanding BDD through this lens also highlights the limitations of isolated treatment approaches that focus solely on either psychological symptoms or biological dysfunction^[27]. Effective interventions must address both dimensions simultaneously. Cognitive-behavioral therapy, often combined with pharmacological treatments such as SSRIs, has shown promising results in targeting these interconnected mechanisms. Additionally, emerging therapies like transcranial magnetic stimulation offer new possibilities for directly addressing neural dysfunction. These treatment strategies demonstrate the potential of a neurobehavioral approach to bridge the gap between brain function and observable behaviors, ultimately improving therapeutic outcomes^[28].

This article aims to synthesize current findings from neuroscience and behavioral psychology to offer a comprehensive overview of the brain-behavior connection in BDD. By examining the interplay between neural abnormalities, cognitive distortions, and behavioral patterns, this review seeks to provide valuable insights for clinicians, researchers, and mental health professionals^[29]. A deeper understanding of these mechanisms may pave the way for more targeted and effective treatments, offering hope for improved outcomes in individuals affected by this debilitating disorder.

Bridging Brain and Behavior

Understanding BDD from a neurobehavioral perspective requires examining how structural and functional abnormalities in the brain interact with cognitive and behavioral patterns to perpetuate the disorder. Research has consistently shown irregularities in brain regions responsible for visual processing, emotional regulation, and self-referential thinking. For example, hyperactivity in the orbitofrontal cortex has been linked to obsessive preoccupations, while heightened amygdala responses suggest an exaggerated emotional reaction to perceived flaws. Additionally, dysfunction in the fusiform gyrus, a region crucial for facial recognition and detail processing, may explain why individuals with BDD fixate on minor imperfections that others would overlook^[30]. These findings indicate that the distorted self-image experienced by individuals with BDD is not solely

a product of psychological biases but is deeply rooted in neurobiological processes.

At the same time, behavioral patterns observed in BDD, such as mirror checking, skin picking, and avoidance of social situations, are closely tied to these neurobiological dysfunctions. These repetitive behaviors are often triggered by intrusive thoughts and emotional distress, providing temporary relief but ultimately reinforcing the obsessive cycle. The brain's reward pathways play a role in this reinforcement, as the brief reduction in anxiety following a compulsive behavior strengthens the neural circuits associated with these maladaptive responses^[31]. Over time, these patterns become deeply ingrained, creating a persistent feedback loop between neural activity, cognitive distortions, and behavioral responses.

The neurobehavioral perspective emphasizes that BDD cannot be fully understood without considering this interplay between brain function and behavior. This framework also highlights the importance of early intervention, as prolonged engagement in maladaptive behaviors may lead to further neurobiological changes that make treatment more challenging. Furthermore, it underscores the need for integrated therapeutic approaches that address both cognitive and neural dysfunctions simultaneously^[32]. While cognitive-behavioral therapy has been a cornerstone in treating BDD, emerging treatments such as neuromodulation techniques, including transcranial magnetic stimulation, show promise in directly targeting brain regions implicated in the disorder.

Exploring BDD through this lens not only deepens the understanding of its etiology but also paves the way for more personalized treatment approaches. By identifying specific neurobehavioral markers, clinicians may eventually be able to predict which treatments will be most effective for individual patients^[33]. This evolving perspective represents a crucial step forward in bridging the gap between neuroscience and clinical practice, offering new hope for individuals struggling with the debilitating effects of BDD.

Neurobiological Foundations of BDD

Research into the neurobiological underpinnings of BDD has revealed significant abnormalities in brain structure and function, shedding light on why individuals with the disorder experience persistent and distressing preoccupations with their appearance. Neuroimaging studies, particularly functional magnetic resonance imaging (fMRI) and positron emission tomography (PET), have been instrumental in identifying regions of the brain implicated in the disorder. These studies consistently highlight dysfunctions in areas responsible for visual processing, emotional regulation, and self-referential thinking, suggesting that distorted body image perceptions are deeply rooted in altered brain activity^[34].

One of the most well-documented findings in BDD research involves abnormalities in the orbitofrontal cortex (OFC). This region plays a critical role in decision-making, impulse control, and the evaluation of perceived threats. In individuals with BDD, hyperactivity in the OFC is associated with heightened obsessive thinking and repetitive compulsive behaviors, such as mirror checking and reassurance-seeking. The OFC's involvement suggests that individuals may struggle with appropriately filtering or dismissing intrusive thoughts about

their appearance, leading to the persistent cycle of obsession and compulsion observed in the disorder^[35].

Additionally, the amygdala, a region central to emotional processing and threat detection, exhibits heightened activity in individuals with BDD. This hyperactivation may explain the intense emotional distress and anxiety triggered by perceived flaws. Even minor physical imperfections, which would typically be disregarded or rationalized by individuals without BDD, can provoke disproportionate emotional responses, further reinforcing obsessive thoughts and compulsive behaviors^[36]. The amygdala's involvement also underscores why individuals with BDD often experience heightened social anxiety and avoid situations where they fear being scrutinized by others.

Another critical area implicated in BDD is the fusiform gyrus, a brain region responsible for processing detailed visual information, particularly related to faces. Research suggests that individuals with BDD may have heightened sensitivity to minor asymmetries or imperfections in their facial features. This abnormal processing could explain why individuals become hyper-focused on specific perceived flaws and why these features appear magnified or distorted in their self-perception. Dysfunction in the fusiform gyrus also contributes to an inability to integrate visual information holistically, causing individuals with BDD to fixate on isolated details rather than perceiving their appearance as a cohesive whole^[37].

Furthermore, disruptions in the default mode network (DMN), a network of brain regions active during self-referential thought and introspection, have been observed in individuals with BDD. Dysregulation within the DMN may contribute to the persistent self-focused attention and distorted self-image that characterize the disorder^[38]. These disruptions suggest that individuals with BDD may have difficulty shifting their attention away from their perceived flaws, further embedding their preoccupations into daily thought patterns.

Collectively, these neurobiological findings offer critical insights into the mechanisms underlying BDD. They suggest that the disorder arises from a complex interplay between heightened emotional responses, dysfunctional visual processing, and impaired self-referential thinking. This understanding underscores the importance of treatment approaches that not only address cognitive distortions and maladaptive behaviors but also target the underlying neurobiological dysfunctions. As research advances, the integration of neuroimaging biomarkers into clinical practice may pave the way for more personalized and effective treatment strategies tailored to the specific neural profiles of individuals with BDD.

Neurochemical Imbalances in BDD

Research into the neurobiological underpinnings of BDD has revealed significant abnormalities in brain structure and function, shedding light on why individuals with the disorder experience persistent and distressing preoccupations with their appearance. Neuroimaging studies, particularly functional magnetic resonance imaging and positron emission tomography, have been instrumental in identifying regions of the brain implicated in the disorder^[39]. These studies consistently highlight dysfunctions in areas responsible for visual processing, emotional regulation, and self-referential thinking, suggesting that distorted body image perceptions are deeply rooted in altered brain activity.

One of the most well-documented findings in BDD research involves abnormalities in the orbitofrontal cortex. This region plays a critical role in decision-making, impulse control, and the evaluation of perceived threats. In individuals with BDD, hyperactivity in the orbitofrontal cortex is associated with heightened obsessive thinking and repetitive compulsive behaviors, such as mirror checking and reassurance-seeking^[40]. The involvement of this region suggests that individuals may struggle with appropriately filtering or dismissing intrusive thoughts about their appearance, leading to the persistent cycle of obsession and compulsion observed in the disorder.

Additionally, the amygdala, a region central to emotional processing and threat detection, exhibits heightened activity in individuals with BDD. This hyperactivation may explain the intense emotional distress and anxiety triggered by perceived flaws. Even minor physical imperfections, which would typically be disregarded or rationalized by individuals without BDD, can provoke disproportionate emotional responses, further reinforcing obsessive thoughts and compulsive behaviors^[41]. The involvement of the amygdala also underscores why individuals with BDD often experience heightened social anxiety and avoid situations where they fear being scrutinized by others.

Another critical area implicated in BDD is the fusiform gyrus, a brain region responsible for processing detailed visual information, particularly related to faces. Research suggests that individuals with BDD may have heightened sensitivity to minor asymmetries or imperfections in their facial features. This abnormal processing could explain why individuals become hyper-focused on specific perceived flaws and why these features appear magnified or distorted in their self-perception^[42]. Dysfunction in the fusiform gyrus also contributes to an inability to integrate visual information holistically, causing individuals with BDD to fixate on isolated details rather than perceiving their appearance as a cohesive whole.

Furthermore, disruptions in the default mode network, a network of brain regions active during self-referential thought and introspection, have been observed in individuals with BDD. Dysregulation within this network may contribute to the persistent self-focused attention and distorted self-image that characterize the disorder. These disruptions suggest that individuals with BDD may have difficulty shifting their attention away from their perceived flaws, further embedding their preoccupations into daily thought patterns^[43].

Collectively, these neurobiological findings offer critical insights into the mechanisms underlying BDD. They suggest that the disorder arises from a complex interplay between heightened emotional responses, dysfunctional visual processing, and impaired self-referential thinking. This understanding underscores the importance of treatment approaches that not only address cognitive distortions and maladaptive behaviors but also target the underlying neurobiological dysfunctions. As research advances, the integration of neuroimaging biomarkers into clinical practice may pave the way for more personalized and effective treatment strategies tailored to the specific neural profiles of individuals with BDD^[44].

The role of neurochemical imbalances in BDD further complements this neurobiological perspective. Neurotransmitters such as serotonin, dopamine, and gamma-aminobutyric acid have been implicated in the regulation of mood, anxiety, and

repetitive behaviors, all of which are central features of BDD. Dysregulation of these neurotransmitters may exacerbate obsessive thinking, emotional dysregulation, and compulsive behaviors, creating a neurochemical backdrop that reinforces the brain-behavior feedback loop seen in the disorder^[45].

Serotonin, in particular, has been extensively studied in relation to BDD. Reduced serotonin availability or receptor dysfunction is thought to contribute to the obsessive-compulsive features commonly seen in individuals with the disorder. This is supported by the efficacy of selective serotonin reuptake inhibitors in reducing symptoms of BDD. These medications are believed to work by increasing serotonin levels in the synaptic cleft, thereby modulating mood and reducing obsessive thinking. However, not all individuals respond equally to serotonin-based treatments, suggesting that other neurochemical pathways may also play a role in the disorder^[46].

Dopamine, another key neurotransmitter, is associated with reward processing, motivation, and pleasure. Abnormal dopamine signaling may contribute to the compulsive behaviors observed in BDD, as repetitive actions such as mirror checking or grooming may temporarily alleviate anxiety, creating a reward loop that reinforces these behaviors. Additionally, disruptions in the dopamine system could contribute to the heightened focus on specific physical details and the inability to shift attention away from perceived flaws^[47].

Gamma-aminobutyric acid, the brain's primary inhibitory neurotransmitter, is also thought to play a role in BDD. Dysregulation in gamma-aminobutyric acid signaling may contribute to heightened anxiety and emotional arousal, which are often observed in individuals with the disorder. This imbalance may further impair the brain's ability to regulate intrusive thoughts and compulsive urges, amplifying the cycle of obsession and behavior^[48].

Understanding these neurochemical imbalances provides a clearer picture of how brain function influences the psychological and behavioral manifestations of BDD. It also highlights the importance of pharmacological treatments that target these pathways, either alone or in combination with behavioral therapies. Moving forward, research focusing on the interaction between neurotransmitter systems and brain networks implicated in BDD will be essential in refining treatment approaches and improving outcomes for affected individuals.

Neurobiological and Neurochemical Mechanisms

The interaction between neurobiological and neurochemical mechanisms in BDD forms the foundation for understanding the cognitive and behavioral patterns that characterize the disorder. Abnormalities in brain regions such as the orbitofrontal cortex, amygdala, and fusiform gyrus, combined with dysregulation in neurotransmitters like serotonin, dopamine, and gamma-aminobutyric acid, create a feedback loop where distorted perceptions, obsessive thoughts, and maladaptive behaviors reinforce one another^[49]. This dynamic relationship between brain function and behavior not only perpetuates the symptoms of BDD but also makes the disorder particularly resistant to change without targeted interventions.

At a cognitive level, individuals with BDD often exhibit selective attention to perceived flaws in their appearance. This hyper-focus on minor imperfections is driven by altered

activity in visual processing areas and emotional regulatory centers. Instead of perceiving their physical features holistically, individuals fixate on isolated details, magnifying their perceived defects. These cognitive distortions are further reinforced by heightened emotional responses mediated by the amygdala, creating a cycle where thoughts about appearance become intrusive and difficult to dismiss^[50]. Over time, these cognitive distortions solidify into rigid beliefs, such as the conviction that others are constantly noticing or judging their perceived flaws.

Behavioral patterns in BDD emerge as attempts to manage the distress caused by these cognitive distortions and emotional responses. Repetitive behaviors such as mirror checking, grooming, skin-picking, and reassurance-seeking are often performed in an effort to reduce anxiety or correct perceived imperfections. However, these behaviors provide only temporary relief, and their repetitive nature reinforces both cognitive and emotional dysfunctions. For example, mirror checking may briefly reduce anxiety, but it also strengthens the brain's association between visual self-scrutiny and emotional relief, perpetuating the compulsion to repeat the behavior^[51]. Avoidance behaviors, such as withdrawing from social situations or covering up perceived flaws with clothing or makeup, further limit opportunities for corrective experiences that might challenge these distorted beliefs.

Emotionally, individuals with BDD often experience intense anxiety, shame, and self-disgust when confronted with their perceived flaws. These emotional responses are not proportionate to the actual physical characteristics in question but are instead amplified by hyperactivity in the amygdala and altered neurotransmitter signaling^[52]. This heightened emotional reactivity makes it difficult for individuals to rationalize their thoughts or gain perspective on their appearance. Furthermore, the emotional toll of these experiences often leads to secondary symptoms, including depression, social isolation, and, in severe cases, suicidal ideation.

From a neurobehavioral perspective, the relationship between cognition, behavior, and brain function can be understood as a self-reinforcing loop. Intrusive thoughts about appearance activate emotional distress, which drives compulsive behaviors aimed at alleviating that distress. These behaviors, in turn, strengthen the neural circuits associated with obsession and compulsion, making the symptoms of BDD more entrenched over time^[53]. This cyclical relationship highlights why interventions that target only one aspect of the disorder—whether cognitive, behavioral, or neurobiological—often fall short in producing lasting change.

Integrated treatment approaches that address both the neurobiological and behavioral dimensions of BDD have shown promise in breaking this cycle. Cognitive-behavioral therapy, particularly exposure and response prevention, aims to disrupt maladaptive behavioral patterns while challenging distorted cognitive beliefs^[54]. Pharmacological treatments, such as selective serotonin reuptake inhibitors, target the underlying neurochemical imbalances, reducing obsessive thoughts and emotional distress. Emerging therapies, such as transcranial magnetic stimulation, offer the potential to directly modulate brain activity in regions implicated in BDD, addressing dysfunction at its source.

Understanding the interaction between brain function, cognitive

distortions, and behavioral patterns in BDD underscores the need for a multifaceted approach to treatment. Future research focused on mapping these relationships in greater detail may pave the way for more targeted and personalized interventions. By bridging the gap between neuroscience and clinical practice, the neurobehavioral perspective offers a promising framework for improving outcomes and reducing the burden of this debilitating disorder.

Cognitive and Behavioral Mechanisms

Cognitive and behavioral mechanisms play a central role in the onset, maintenance, and persistence of BDD. These mechanisms are deeply intertwined with the neurobiological abnormalities observed in individuals with the disorder, creating a feedback loop that reinforces distorted perceptions, obsessive thoughts, and maladaptive behaviors^[55]. Understanding these mechanisms is essential for developing targeted interventions that address both the psychological and neurobiological dimensions of BDD.

Cognitive distortions are a hallmark of BDD, shaping how individuals perceive and interpret their physical appearance. One of the most prominent distortions is selective attention, where individuals hyper-focus on perceived flaws while disregarding or minimizing their overall appearance. This heightened focus often leads to an exaggerated perception of minor imperfections, making them appear far more prominent and distressing than they are in reality^[56]. Another common cognitive distortion is catastrophic thinking, where individuals anticipate the worst possible outcomes related to their appearance. For example, they may believe that a small blemish will lead to widespread social rejection or that others will ridicule them because of a perceived asymmetry in their facial features. Overestimation of others' judgments further compounds these fears, as individuals often believe that others notice and scrutinize their perceived flaws to the same extent they do themselves.

These cognitive distortions give rise to a range of maladaptive behavioral patterns aimed at reducing anxiety and distress. Repetitive behaviors are among the most commonly observed, including mirror checking, skin-picking, excessive grooming, and frequent reassurance-seeking from others. These behaviors serve as temporary coping mechanisms, offering brief moments of relief from the anxiety caused by obsessive thoughts. However, the relief is fleeting, and the repetitive nature of these actions reinforces the underlying cognitive distortions and emotional distress^[57]. Over time, these behaviors become deeply ingrained, creating a self-sustaining cycle that is difficult to break. In addition to repetitive behaviors, avoidance behaviors are also prevalent in BDD. Individuals may withdraw from social situations, avoid bright lighting, or use excessive makeup and clothing to hide their perceived flaws. While these avoidance strategies may temporarily reduce anxiety, they also reinforce negative self-perceptions and prevent individuals from gaining corrective feedback from others.

The cognitive-behavioral model of BDD provides a framework for understanding how these mechanisms interact to maintain the disorder. Obsessive thoughts about appearance trigger intense emotional distress, prompting compulsive behaviors or avoidance strategies as an attempt to reduce anxiety^[58]. These behaviors may offer temporary relief, but they simultaneously reinforce the obsessive thought patterns and maladaptive beliefs

that initiated them. For instance, an individual who repeatedly checks their appearance in a mirror may experience brief reassurance, but this act also strengthens the brain's association between mirror checking and anxiety relief. Over time, this cycle becomes self-perpetuating, trapping individuals in a loop of obsession, compulsion, and temporary relief that prevents meaningful progress toward recovery.

This cyclical pattern highlights why interventions targeting both cognitive distortions and maladaptive behaviors are essential in treating BDD. Cognitive-behavioral therapy remains one of the most effective approaches, as it aims to challenge distorted beliefs, reduce compulsive behaviors, and break the reinforcement cycle that maintains the disorder. Techniques such as exposure and response prevention are particularly valuable in helping individuals confront their fears without resorting to compulsive rituals. By addressing both the cognitive and behavioral dimensions of BDD, therapeutic interventions can disrupt the vicious cycle and pave the way for more sustainable improvements in mental health and quality of life.

Interaction Between Neurobiology and Behavior

The interaction between neurobiological dysfunction and behavioral patterns in BDD creates a self-reinforcing cycle that perpetuates obsessive thoughts, distorted perceptions, and maladaptive behaviors. This interplay underscores how structural and functional abnormalities in the brain translate into cognitive distortions and repetitive behavioral patterns, which, in turn, reinforce underlying neural dysfunctions^[59]. Understanding this brain-behavior feedback loop is essential for developing integrated treatment approaches that address both dimensions simultaneously.

The brain-behavior feedback loop in BDD highlights how hyperactivity in certain brain regions sustains distorted cognitive and emotional patterns. Abnormal activity in visual processing areas, particularly the fusiform gyrus, leads individuals to fixate on minute details of their appearance while neglecting the overall holistic perception of their physical features^[60]. This heightened sensitivity contributes to the exaggerated perception of minor flaws, which fuels obsessive thoughts. At the same time, hyperactivity in the amygdala amplifies emotional responses to these perceived imperfections, creating intense anxiety, shame, and fear of social judgment. These emotional reactions further reinforce maladaptive behavioral responses, such as mirror checking or avoidance behaviors, which temporarily reduce anxiety but ultimately strengthen the neural circuits associated with obsession and compulsion. Over time, this loop solidifies distorted cognitive patterns, emotional dysregulation, and repetitive behaviors, making BDD highly resistant to change without targeted intervention.

Stress and trauma also play a significant role in shaping the neurobiological and behavioral patterns observed in BDD. Early-life trauma, including emotional neglect, bullying, or exposure to highly critical environments, can have long-lasting effects on brain development and plasticity. Chronic exposure to stress during critical developmental periods can alter the structure and function of brain regions involved in emotional regulation and threat detection, such as the amygdala, prefrontal cortex, and hippocampus^[61]. These alterations may predispose individuals to heightened emotional sensitivity and an increased likelihood

of developing maladaptive coping mechanisms. Additionally, early traumatic experiences can shape core beliefs about self-worth, appearance, and social acceptance, laying the cognitive foundation for the distorted self-perceptions seen in BDD.

Self-referential thinking patterns further exacerbate the interaction between neurobiology and behavior in BDD. The default mode network, a brain network active during introspection, self-focused thought, and daydreaming, often exhibits dysregulation in individuals with the disorder. Instead of engaging in balanced self-reflection, individuals with BDD become trapped in repetitive and intrusive thoughts about their perceived flaws^[62]. This hyperactivity in self-referential thinking prevents cognitive flexibility, making it challenging to shift focus away from obsessive concerns. Additionally, the dysfunction of this network contributes to heightened self-consciousness and a distorted internal narrative about physical appearance.

Together, these factors form a complex interaction where neurobiological abnormalities drive cognitive distortions, which, in turn, manifest as maladaptive behaviors. These behaviors then reinforce neural activity patterns, further entrenching the disorder. This cyclical relationship explains why BDD can be so resistant to treatment when interventions target only one dimension of the disorder. Integrated therapeutic approaches that address both brain and behavior are necessary to break this cycle. Cognitive-behavioral therapy helps individuals challenge distorted beliefs and reduce compulsive behaviors, while pharmacological interventions and neuromodulation techniques directly target the neurobiological underpinnings of the disorder.

Understanding the interaction between neurobiology and behavior in BDD offers valuable insights into why certain treatments are more effective for some individuals than others. Future research focusing on identifying neurobehavioral biomarkers could help predict treatment outcomes and guide clinicians in tailoring interventions to the unique neural profiles of their patients. This evolving perspective not only deepens our understanding of BDD but also represents a significant step toward more effective, personalized therapeutic approaches.

Neurobehavioral Insights into Treatment Approaches

Treatment approaches for BDD have evolved to address both the cognitive-behavioral and neurobiological dimensions of the disorder. Understanding how brain function and behavior interact has allowed for the development of interventions that target the underlying neurochemical imbalances, maladaptive cognitive patterns, and repetitive behaviors that sustain BDD. Effective treatments often combine pharmacological, psychotherapeutic, and neuromodulation techniques to address the disorder holistically^[63].

Pharmacological interventions play a central role in managing the neurochemical dysregulation observed in BDD. Selective serotonin reuptake inhibitors, or SSRIs, are currently the first-line pharmacological treatment for BDD. These medications work by increasing serotonin availability in the brain, helping to regulate mood, reduce obsessive thoughts, and alleviate anxiety^[64]. The effectiveness of SSRIs supports the hypothesis that serotonin dysregulation contributes to the obsessive-compulsive features of the disorder. However, not all individuals respond equally to SSRIs, highlighting the need for alternative pharmacological

strategies. Emerging treatments, such as glutamatergic agents, are being investigated for their potential to address treatment-resistant cases^[65]. These agents target the glutamate system, which plays a role in synaptic plasticity and neural excitability, and may offer additional therapeutic benefits for individuals who do not respond to traditional serotonin-based medications.

Cognitive-behavioral therapy remains the most well-established psychotherapeutic intervention for BDD, with a strong neurobehavioral rationale supporting its effectiveness. CBT helps individuals identify and challenge distorted beliefs about their appearance while reducing maladaptive behaviors such as mirror checking, reassurance seeking, and avoidance. Exposure and response prevention, a key component of CBT, encourages individuals to confront their fears without engaging in compulsive behaviors, gradually breaking the obsessive-compulsive cycle^[66]. From a neurobehavioral perspective, these techniques are thought to modulate neural pathways associated with obsession, anxiety, and reward processing, facilitating long-term changes in brain function and behavior.

Neuromodulation techniques have emerged as promising interventions for treatment-resistant cases of BDD. Transcranial magnetic stimulation, or TMS, is a non-invasive procedure that uses magnetic fields to stimulate specific brain regions involved in emotional regulation and obsessive thinking, such as the orbitofrontal cortex^[67]. Research suggests that TMS can reduce symptom severity by normalizing abnormal brain activity patterns. Deep brain stimulation, or DBS, represents a more invasive approach, where electrodes are implanted in specific brain regions to regulate dysfunctional neural circuits^[68]. Although still considered experimental, DBS has shown potential in severe, treatment-resistant cases of BDD, offering hope for individuals who have not responded to other interventions.

Integrated neurobehavioral interventions combine pharmacotherapy with behavioral therapy to address both the neurochemical and cognitive-behavioral aspects of BDD simultaneously. This approach recognizes that treating one dimension of the disorder in isolation is often insufficient^[69]. Pharmacological treatments can reduce symptom intensity, making individuals more receptive to the cognitive and behavioral strategies employed in therapy. Similarly, behavioral interventions can reinforce neurobiological changes initiated by medications, creating a synergistic effect that enhances treatment outcomes.

The growing integration of neuroscience into clinical practice represents a significant step forward in improving treatment strategies for BDD. Future research focused on identifying biomarkers for treatment response, optimizing neuromodulation protocols, and exploring novel pharmacological agents will be essential in refining these approaches. By addressing both the brain and behavior, neurobehavioral treatment strategies offer a more comprehensive and personalized approach to managing the complexities of BDD.

Future Research Directions

Future research into BDD must address critical gaps in our understanding of the disorder's neurobehavioral underpinnings, with a focus on refining treatment strategies and enhancing personalized care. While significant progress has been made in

identifying brain regions and neurochemical pathways involved in BDD, several areas warrant further investigation to deepen our understanding and improve clinical outcomes.

One promising avenue for future research is the investigation of sex differences in neurobehavioral patterns in BDD. Epidemiological studies suggest that while the prevalence of BDD is relatively similar across genders, the presentation of symptoms, comorbidities, and coping mechanisms can vary significantly between men and women^[70]. Neuroimaging studies could explore whether these differences are reflected in distinct patterns of brain activity, neurotransmitter function, or behavioral responses. Understanding these variations may allow for more tailored treatment approaches that account for gender-specific neurobiological and psychological factors, ultimately improving intervention efficacy.

Longitudinal studies examining brain plasticity before and after treatment are another essential direction for future research. Current neuroimaging studies offer valuable insights into structural and functional abnormalities in individuals with BDD, but most are cross-sectional, capturing a single point in time^[71]. Longitudinal designs would allow researchers to observe how brain networks and neurochemical pathways evolve during and after treatment. For example, examining changes in the orbitofrontal cortex, amygdala, and fusiform gyrus following cognitive-behavioral therapy, pharmacological treatment, or neuromodulation could provide evidence of neuroplasticity and identify biomarkers associated with treatment response. These findings may help refine therapeutic interventions and improve long-term outcomes.

The development of targeted neuromodulation therapies represents another critical frontier in BDD research. Techniques such as transcranial magnetic stimulation and deep brain stimulation have shown promise in modulating neural activity in treatment-resistant cases^[72]. However, these interventions are still in their early stages of application for BDD, and their mechanisms of action remain incompletely understood. Future research could focus on identifying the most effective brain regions for stimulation, optimizing stimulation protocols, and determining which patient profiles are most likely to benefit from these treatments. Additionally, exploring non-invasive neuromodulation techniques, such as transcranial direct current stimulation, may offer alternative options with fewer risks and side effects.

Finally, integrating genetic and epigenetic research into the neurobehavioral study of BDD could uncover predispositions to the disorder and provide insights into how environmental factors, such as trauma or chronic stress, influence gene expression and brain function. Understanding these interactions may lead to early identification of at-risk individuals and the development of preventative interventions.

Conclusion

The neurobehavioral perspective on BDD offers a comprehensive framework for understanding the intricate interplay between brain function, cognitive distortions, emotional regulation, and maladaptive behaviors that define the disorder. Over the past two decades, significant advancements in neuroimaging techniques, such as functional magnetic resonance imaging and positron emission tomography, have allowed researchers

to map the neural correlates of BDD with increasing precision. These studies have consistently highlighted abnormalities in key brain regions, including the orbitofrontal cortex, amygdala, fusiform gyrus, and default mode network. Hyperactivity in these regions has been linked to obsessive thoughts, heightened emotional reactivity, and distorted visual processing, all of which contribute to the persistent preoccupation with perceived physical flaws. Similarly, dysregulation in neurotransmitters such as serotonin, dopamine, and gamma-aminobutyric acid has shed light on the neurochemical imbalances that underlie the obsessive-compulsive patterns, emotional distress, and reinforcement of maladaptive behaviors commonly observed in individuals with BDD.

At the cognitive level, BDD is marked by pervasive distortions in self-perception, including selective attention to perceived flaws, catastrophic thinking, and an overestimation of others' judgments. These cognitive distortions are not isolated phenomena but are closely intertwined with underlying neurobiological dysfunctions. For instance, hyperactivity in the fusiform gyrus contributes to an exaggerated focus on minute details of one's appearance, while heightened amygdala activity amplifies emotional distress when confronted with perceived flaws. These cognitive processes create fertile ground for maladaptive behavioral patterns, such as mirror checking, skin picking, avoidance of social situations, and repetitive reassurance-seeking. These behaviors, while initially intended to reduce anxiety, serve to reinforce obsessive thought patterns, further entrenching the disorder in a self-sustaining feedback loop.

The neurobehavioral model underscores the need for a multidisciplinary approach to treatment—one that addresses both the psychological and biological dimensions of BDD. Cognitive-behavioral therapy, particularly exposure and response prevention, has emerged as a cornerstone in the psychological treatment of BDD. This approach targets cognitive distortions and maladaptive behaviors by encouraging individuals to confront their fears without resorting to compulsive rituals. At the same time, pharmacological interventions, especially selective serotonin reuptake inhibitors, have proven effective in modulating the neurochemical imbalances associated with obsessive thinking and emotional dysregulation. Emerging treatments, including glutamatergic agents and other novel pharmacotherapies, offer additional avenues for addressing treatment-resistant cases.

Neuromodulation techniques, such as transcranial magnetic stimulation and deep brain stimulation, represent promising interventions for individuals who do not respond to conventional therapies. These methods directly target the neural circuits implicated in BDD, offering the potential to modulate dysfunctional brain activity in regions such as the orbitofrontal cortex and amygdala. While these interventions are still in their experimental stages, early findings suggest significant promise for reducing symptom severity and improving overall functioning in individuals with severe BDD.

The integration of these treatment modalities into cohesive neurobehavioral intervention plans reflects a growing acknowledgment that BDD cannot be fully addressed by focusing on either the brain or behavior in isolation. Instead, successful treatment must consider the dynamic interplay

between neurobiological factors and cognitive-behavioral mechanisms, tailoring interventions to the unique profiles of individual patients.

Looking forward, future research must continue to refine our understanding of these complex interactions. Longitudinal studies are essential to map changes in brain plasticity before, during, and after treatment, offering insights into how therapeutic interventions can reshape dysfunctional neural pathways over time. Investigating sex differences in neurobehavioral patterns may uncover critical variations in symptom presentation, treatment response, and neural activity, ultimately allowing for more targeted and gender-sensitive interventions. Additionally, advancements in neuromodulation technologies, including non-invasive approaches like transcranial direct current stimulation, hold the potential to expand therapeutic options for treatment-resistant cases.

Equally important is the development of predictive biomarkers that can identify individuals at higher risk for developing BDD or those who are more likely to respond to specific treatments. The integration of genetic and epigenetic research into the neurobehavioral study of BDD may further clarify the hereditary and environmental contributions to the disorder, opening up possibilities for early identification and intervention.

In conclusion, the neurobehavioral perspective represents a transformative shift in how BDD is understood, diagnosed, and treated. By bridging the gap between neuroscience and clinical psychology, this framework allows for a more nuanced understanding of the disorder's etiology while informing innovative treatment strategies. As research continues to unravel the brain-behavior connection in BDD, the promise of more effective, personalized interventions becomes increasingly tangible. This evolving understanding offers hope not only for improved clinical outcomes but also for reducing the profound psychological distress and functional impairment faced by individuals living with this debilitating disorder.

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REFERENCES

1. Phillips, K. A. (2000). Quality of life for patients with body dysmorphic disorder. *The Journal of Nervous and Mental Disease*, 188(3), 170-175.

2. Kelly, M. M., Brault, M. E., & Didie, E. R. (2017). Psychosocial functioning and quality of life in body dysmorphic disorder. *Body dysmorphic disorder: Advances in research and clinical practice*, 139-154.
3. Reese, H. E., McNally, R. J., & Wilhelm, S. (2010). Facial asymmetry detection in patients with body dysmorphic disorder. *Behaviour research and therapy*, 48(9), 936-940. DOI: [10.1016/j.brat.2010.05.021](https://doi.org/10.1016/j.brat.2010.05.021)
4. Simmons, R. A., & Phillips, K. A. (2017). Core clinical features of body dysmorphic disorder: appearance preoccupations, negative emotions, core beliefs, and repetitive and avoidance behaviors. *Body dysmorphic disorder: Advances in research and clinical practice*, 61-80.
5. Phillips, K. A. (2005). *The broken mirror: understanding and treating body dysmorphic disorder*. Oxford University Press, USA.
6. Phillips, K. A. (2009). *Understanding body dysmorphic disorder*. Oxford University Press.
7. Saeed, B., Sheikh, N. M., Shahzadi, N., & Khan, Z. H. (2023). Shaping Self-Perception: The Intricate Relationship Between Self-Concept Self-Image And Body Dysmorphic Disorder. *Journal of Positive School Psychology*, 7(5), 1043-1059.
8. Rück, C., Mataix-Cols, D., Feusner, J. D., Shavitt, R. G., Veale, D., Krebs, G., & Fernández de la Cruz, L. (2024). Body dysmorphic disorder. *Nature Reviews Disease Primers*, 10(1), 1-15. DOI: [10.1038/s41572-024-00577-z](https://doi.org/10.1038/s41572-024-00577-z)
9. Veale, D., & Gilbert, P. (2014). Body dysmorphic disorder: The functional and evolutionary context in phenomenology and a compassionate mind. *Journal of Obsessive-Compulsive and Related Disorders*, 3(2), 150-160. DOI: [10.1016/j.jocrd.2013.11.005](https://doi.org/10.1016/j.jocrd.2013.11.005)
10. Grace, S. A., Labuschagne, I., Kaplan, R. A., & Rossell, S. L. (2017). The neurobiology of body dysmorphic disorder: A systematic review and theoretical model. *Neuroscience & Biobehavioral Reviews*, 83, 83-96. DOI: [10.1016/j.neubiorev.2017.10.003](https://doi.org/10.1016/j.neubiorev.2017.10.003)
11. Rossell, S. L., Harrison, B. J., & Castle, D. (2015). Can understanding the neurobiology of body dysmorphic disorder (BDD) inform treatment?. *Australasian Psychiatry*, 23(4), 361-364. DOI: [10.1177/1039856215591327](https://doi.org/10.1177/1039856215591327)
12. Landi, P., Marazziti, D., Rutigliano, G., & Dell'Osso, L. (2016). Insight in psychiatry and neurology: state of the art, and hypotheses. *Harvard Review of Psychiatry*, 24(3), 214-228. DOI: [10.1097/HRP.0000000000000083](https://doi.org/10.1097/HRP.0000000000000083)
13. Ahmadpanah, M., Arji, M., Arji, J., Haghighi, M., Jahangard, L., Sadeghi Bahmani, D., & Brand, S. (2019). Sociocultural attitudes towards appearance, self-esteem and symptoms of body-dysmorphic disorders among young adults. *International journal of environmental research and public health*, 16(21), 4236. DOI: [10.3390/ijerph16214236](https://doi.org/10.3390/ijerph16214236)
14. Machremi, E., Bakirtzis, C., Karakasi, M. V., Boziki, M. K., Siokas, V., Aloizou, A. M., ... & Grigoriadis, N. (2022). What scans see when patients see defects: neuroimaging findings in body dysmorphic disorder. *Journal of Integrative Neuroscience*, 21(2), 45. DOI: [10.31083/j.jin2102045](https://doi.org/10.31083/j.jin2102045)
15. Virgili, G., Neill, E., Enticott, P., Castle, D., & Rossell, S. L. (2024). A systematic review of visual processing in body dysmorphic disorder (BDD). *Psychiatry Research*, 116013. DOI: [10.1016/j.psychres.2024.116013](https://doi.org/10.1016/j.psychres.2024.116013)
16. Jefferies-Sewell, K., Chamberlain, S. R., Fineberg, N. A., & Laws, K. R. (2017). Cognitive dysfunction in body dysmorphic disorder: new implications for nosological systems and neurobiological models. *CNS spectrums*, 22(1), 51-60. DOI: [10.1017/S1092852916000468](https://doi.org/10.1017/S1092852916000468)
17. Rangaprakash, D., Bohon, C., Lawrence, K. E., Moody, T., Morfini, F., Khalsa, S. S., ... & Feusner, J. D. (2018). Aberrant dynamic connectivity for fear processing in anorexia nervosa and body dysmorphic disorder. *Frontiers in psychiatry*, 9, 273. DOI: [10.3389/fpsyt.2018.00273](https://doi.org/10.3389/fpsyt.2018.00273)
18. Borgers, T., Kürten, M., Kappelhoff, A., Enneking, V., Möllmann, A., Schulte, J., ... & Redlich, R. (2022). Brain functional correlates of emotional face processing in body dysmorphic disorder. *Journal of Psychiatric Research*, 147, 103-110.
19. Khemlani-Patel, S., & Neziroglu, F. (2022). *Body dysmorphic disorder*. Hogrefe Publishing GmbH.
20. López-Martín, O., Dores, A. R., Peixoto, M., & Marques, A. (2024). Effectiveness of Interventions for Cognitive Processing in Body Dysmorphic Disorder and Body Dissatisfaction: Systematic Review. *Cognitive Therapy and Research*, 1-19. DOI: [10.1007/s10608-024-10499-5](https://doi.org/10.1007/s10608-024-10499-5)
21. Morgan-Sowada, H. M. (2018). *Relationships and attachment in individuals with body dysmorphic disorder: A qualitative study* (Doctoral dissertation, The University of Iowa).
22. Cororve, M. B., & Gleaves, D. H. (2022). Body dysmorphic disorder: a review of conceptualizations, assessment, and treatment strategies. *Obsessive-Compulsive Disorder and Tourette's Syndrome*, 13-34.
23. Çınaroğlu, M. (2024). Hormonal Catalysts in the Addiction Cycle of Muscle Dysmorphia: A Neuroendocrine Perspective. *The Journal of Neurobehavioral Sciences*, 11(1), 1-9. DOI: [10.4103/jnbs.jnbs_19_23](https://doi.org/10.4103/jnbs.jnbs_19_23)
24. Arienzo, D., Leow, A., Brown, J. A., Zhan, L., GadElkarim, J., Hovav, S., & Feusner, J. D. (2013). Abnormal brain network organization in body dysmorphic disorder. *Neuropsychopharmacology*, 38(6), 1130-1139. DOI: [10.1038/npp.2013.18](https://doi.org/10.1038/npp.2013.18)
25. Feusner, J. D., Neziroglu, F., Wilhelm, S., Mancusi, L., & Bohon, C. (2010). What causes BDD: Research findings and a proposed model. *Psychiatric annals*, 40(7), 349-355. DOI: [10.3928/00485713-20100701-08](https://doi.org/10.3928/00485713-20100701-08)
26. Allen, L. M., Roberts, C., Zimmer-Gembeck, M. J., & Farrell, L. J. (2020). Exploring the relationship between self-compassion and body dysmorphic symptoms in adolescents. *Journal of Obsessive-Compulsive and Related Disorders*, 25, 100535. DOI: [10.1016/j.jocrd.2020.100535](https://doi.org/10.1016/j.jocrd.2020.100535)
27. Pavan, C., Simonato, P., Marini, M., Mazzoleni, F., Pavan, L., & Vindigni, V. (2008). Psychopathologic aspects of body dysmorphic disorder: a literature review. *Aesthetic Plastic Surgery*, 32, 473-484. DOI: [10.1007/s00266-008-9113-2](https://doi.org/10.1007/s00266-008-9113-2)
28. Hong, K., Nezgovorova, V., Uzunova, G., Schlussek, D., & Hollander, E. (2019). Pharmacological treatment of body dysmorphic disorder. *Current Neuropharmacology*, 17(8), 697-702. DOI: [10.2174/1570159X16666180426153940](https://doi.org/10.2174/1570159X16666180426153940)
29. Li, W., Lai, T. M., Loo, S. K., Strober, M., Mohammad-Rezazadeh, I., Khalsa, S., & Feusner, J. (2015). Aberrant early visual neural activity and brain-behavior relationships in anorexia nervosa and body dysmorphic disorder. *Frontiers in human neuroscience*, 9, 301. DOI: [10.3389/fnhum.2015.00301](https://doi.org/10.3389/fnhum.2015.00301)
30. Diaz-Fong, J. P., & Feusner, J. D. (2024). Visual Perceptual Processing Abnormalities in Body Dysmorphic Disorder. DOI: [10.1007/7854_2024_472](https://doi.org/10.1007/7854_2024_472)
31. Phillips, K. A., Rodriguez, C. I., Harding, K. J., Fallon, B. A., & Stein, D. J. (2024). Obsessive-Compulsive Disorder Related Disorders: Hypochondriasis, Hoarding Disorder, Olfactory Reference Disorder, Body Dysmorphic Disorder, Trichotillomania, Excoriation Disorder. In *Tasman's Psychiatry* (pp. 2153-2196). Cham: Springer International Publishing. DOI: [10.1007/978-3-030-51366-5_24](https://doi.org/10.1007/978-3-030-51366-5_24)
32. Iannazzi, E. M., Grennan, G., Zhao, Y., Crane, J., & Fang, A. (2024). CBT Model and Overview of Core Components of CBT for BDD. In *Optimizing Evidence-Based Treatment for Body Dysmorphic Disorder* (pp. 75-96). Cham: Springer Nature Switzerland. DOI: [10.1007/978-3-031-65339-1_5](https://doi.org/10.1007/978-3-031-65339-1_5)
33. Greenberg, J. L., Weingarden, H., Hoepfner, S. S., Berger-Gutierrez,

- R. M., Klare, D., Snorrason, I., ... & Wilhelm, S. (2024). Predicting response to a smartphone-based cognitive-behavioral therapy for body dysmorphic disorder. *Journal of Affective Disorders*, 355, 106-114. DOI: 10.1016/j.jad.2024.03.044
34. Thorpe, F., & Bourne, P. A. (2024). An Examination of Psychopathology: Body Dysmorphic Disorder (BDD).
35. Fineberg, N. A., Chamberlain, S. R., Goudriaan, A. E., Stein, D. J., Vanderschuren, L. J., Gillan, C. M., ... & Potenza, M. N. (2014). New developments in human neurocognition: clinical, genetic, and brain imaging correlates of impulsivity and compulsivity. *CNS spectrums*, 19(1), 69-89. DOI: 10.1017/S1092852913000801
36. Buchanan, B. (2013). *Brain structure and circuitry in body dysmorphic disorder (BDD) patients: a multimodal neuroimaging study* (Doctoral dissertation, Monash University).
37. Feusner, J. D., Townsend, J., Bystritsky, A., & Bookheimer, S. (2007). Visual information processing of faces in body dysmorphic disorder. *Archives of general psychiatry*, 64(12), 1417-1425. DOI: 10.1001/archpsyc.64.12.1417
38. Fang, A., Baran, B., Feusner, J. D., Phan, K. L., Beatty, C. C., Crane, J., ... & Wilhelm, S. (2024). Self-focused brain predictors of cognitive behavioral therapy response in a transdiagnostic sample. *Journal of psychiatric research*, 171, 108-115. DOI: 10.1016/j.jpsychires.2024.01.018
39. Feusner, J. D., Yaryura-Tobias, J., & Saxena, S. (2008). The pathophysiology of body dysmorphic disorder. *Body image*, 5(1), 3-12. DOI: 10.1016/j.bodyim.2007.11.002
40. Atmaca, M., Bingol, I., Aydin, A., Yildirim, H., Okur, I., Yildirim, M. A., ... & Gurok, M. G. (2010). Brain morphology of patients with body dysmorphic disorder. *Journal of affective disorders*, 123(1-3), 258-263. DOI: 10.1016/j.jad.2009.08.012
41. Borgers, T., Kürten, M., Kappelhoff, A., Enneking, V., Möllmann, A., Schulte, J., ... & Redlich, R. (2022). Brain functional correlates of emotional face processing in body dysmorphic disorder. *Journal of Psychiatric Research*, 147, 103-110. DOI: 10.1016/j.jpsychires.2022.01.007
42. Buchanan, B., Rossell, S., Maller, J. J., Toh, W. L., Brennan, S., & Castle, D. (2014). Regional brain volumes in body dysmorphic disorder compared to controls. *Australian & New Zealand Journal of Psychiatry*, 48(7), 654-662. DOI: 10.1177/0004867413520253
43. Ho, J. T., Preller, K. H., & Lenggenhager, B. (2020). Neuropharmacological modulation of the aberrant bodily self through psychedelics. *Neuroscience & Biobehavioral Reviews*, 108, 526-541. DOI: 10.1016/j.neubiorev.2019.12.006
44. Malcolm, A. C. (2020). *Psychology, neurocognition, and neurobiology in body dysmorphic disorder: similarities and differences with obsessive-compulsive disorder* (Doctoral dissertation, Australian Catholic University).
45. Çelik, F. G. H., & Hocaoglu, Ç. Role of Glutamatergic Modulators in the Treatment of Obsessive Compulsive and Related Disorders. *Psikiyatri Guncel Yaklaşımlar*, 16(3), 383-400. DOI: 10.18863/pgy.1279927
46. Castle, D., Beilharz, F., Phillips, K. A., Brakoulias, V., Drummond, L. M., Hollander, E., ... & Fineberg, N. A. (2021). Body dysmorphic disorder: a treatment synthesis and consensus on behalf of the International College of Obsessive-Compulsive Spectrum Disorders and the Obsessive Compulsive and Related Disorders Network of the European College of Neuropsychopharmacology. *International Clinical Psychopharmacology*, 36(2), 61-75. DOI: 10.1097/YIC.0000000000000342
47. Baluchová, S., Brycht, M., Taylor, A., Mortet, V., Krůšek, J., Dittert, I., ... & Schwarzová-Pecková, K. (2021). Enhancing electroanalytical performance of porous boron-doped diamond electrodes by increasing thickness for dopamine detection. *Analytica Chimica Acta*, 1182, 338949. DOI: 10.1016/j.aca.2021.338949
48. Grados, M. A., Atkins, E. B., Kovacikova, G. I., & McVicar, E. (2015). A selective review of glutamate pharmacological therapy in obsessive-compulsive and related disorders. *Psychology research and behavior management*, 115-131. DOI: 10.2147/PRBM.S58601
49. McCurdy-McKinnon, D., & Feusner, J. D. (2017). Neurobiology of body dysmorphic disorder: heritability/genetics, brain circuitry, and visual processing. *Body dysmorphic disorder: Advances in research and clinical practice*, 253.
50. Grochowski, A., Kliem, S., & Heinrichs, N. (2012). Selective attention to imagined facial ugliness is specific to body dysmorphic disorder. *Body image*, 9(2), 261-269.
51. Oakes, A., Collison, J., & Milne-Home, J. (2017). Repetitive, safe, and automatic: the experience of Appearance-Related behaviours in body dysmorphic disorder. *Australian Psychologist*, 52(6), 433-441. DOI: 10.1111/ap.12247
52. Buhlmann, U., Etcoff, N. L., & Wilhelm, S. (2006). Emotion recognition bias for contempt and anger in body dysmorphic disorder. *Journal of psychiatric research*, 40(2), 105-111. DOI: 10.1016/j.jpsychires.2005.03.006
53. Pineda, D. A., Ardila, A., Rosselli, M., Puerta, I. C., Mejia, S., & Toro, M. C. (2000). Neurobehavioral characteristics of adolescents with behavioral dysregulation disorder. *International journal of neuroscience*, 101(1-4), 133-155. DOI: 10.3109/00207450008986497
54. Çınaroğlu, M. Intersecting Realities: Body Dysmorphic Disorder and Substance Abuse in Women—A Holistic Treatment Approach. *Sakarya Üniversitesi Kadın Araştırmaları Dergisi*, 3(1), 1-16. DOI: 10.61158/saukad.1437131
55. Veale, D. (2004). Advances in a cognitive behavioural model of body dysmorphic disorder. *Body image*, 1(1), 113-125. DOI: 10.1016/S1740-1445(03)00009-3
56. Kollei, I., Horndasch, S., Erim, Y., & Martin, A. (2017). Visual selective attention in body dysmorphic disorder, bulimia nervosa and healthy controls. *Journal of psychosomatic research*, 92, 26-33. DOI: 10.1016/j.jpsychores.2016.11.008
57. Phillips, K. A., & Kelly, M. M. (2020). Body dysmorphic disorder: clinical overview and relationship to obsessive-compulsive disorder. *Focus*, 19(4), 413-419. DOI: 10.1176/appi.focus.20210012
58. Neziroglu, F., Khemlani-Patel, S., & Veale, D. (2008). Social learning theory and cognitive behavioral models of body dysmorphic disorder. *Body image*, 5(1), 28-38. DOI: 10.1016/j.bodyim.2008.01.002
59. Didie, E. R., Reinecke, M. A., & Phillips, K. A. (2010). Case conceptualization and treatment of comorbid body dysmorphic disorder and bulimia nervosa. *Cognitive and Behavioral Practice*, 17(3), 259-269. DOI: 10.1016/j.cbpra.2010.02.003
60. Grace, S. A., Labuschagne, I., Castle, D. J., & Rossell, S. L. (2019). Intranasal oxytocin alters amygdala-temporal resting-state functional connectivity in body dysmorphic disorder: a double-blind placebo-controlled randomized trial. *Psychoneuroendocrinology*, 107, 179-186. DOI: 10.1016/j.psyneuen.2019.05.022
61. Goodarzi, M., Noori, M., Aslakerlighvan, M., & Abasi, I. (2022). The Relationship Between Childhood Traumas with Social Appearance Anxiety and Symptoms of Body Dysmorphic Disorder: The Mediating Role of Sociocultural Attitudes Toward Appearance. *Iranian Journal of Psychiatry and Behavioral Sciences*, 16(1). DOI: 10.5812/ijpbs.113064
62. Moody, T. D., Sasaki, M. A., Bohon, C., Strober, M. A., Bookheimer, S. Y., Sheen, C. L., & Feusner, J. D. (2015). Functional connectivity for face processing in individuals with body dysmorphic disorder and anorexia nervosa. *Psychological medicine*, 45(16), 3491-3503. DOI: 10.1017/S0033291715001397
63. Hossain, R., Sinyor, M., Nestor, S., Richter, M. A., Lipsman, N., Hamani, C., & Giacobbe, P. (2023). Mapping the future of interventional psychiatry for the obsessive-compulsive related

- disorders: a scoping review. *Psychiatry Research*, 319, 115007. DOI: 10.1016/j.psychres.2022.115007
64. Phillipou, A., Rossell, S. L., Wilding, H. E., & Castle, D. J. (2016). Randomised controlled trials of psychological & pharmacological treatments for body dysmorphic disorder: A systematic review. *Psychiatry research*, 245, 179-185. DOI: 10.1016/j.psychres.2016.05.062
 65. Laoutidis, Z. G., Lekka, G. E., & Kioulos, K. T. (2016). Glutamatergic agents as add-on medication for the treatment of obsessive-compulsive disorder: a systematic review and meta-analysis. *The Journal of clinical psychiatry*, 77(12), 7839.
 66. Folke, F., Von Bahr, M., Assadi-Talaremi, V., & Ramnero, J. (2012). Exposure and response prevention in the treatment of body dysmorphic disorder: A case series. *Pragmatic Case Studies in Psychotherapy*, 8(4), 255-287. DOI: 10.14713/pcsp.v8i4.1799
 67. van Paridon, M. W., Neuteboom, D., Denys, D. A., Gilbers, R. N., Vulink, N. C., & Scheepstra, K. W. (2022). Effects of repetitive transcranial magnetic stimulation (rTMS) in patients with body dysmorphic disorder (BDD). *Brain Stimulation: Basic, Translational, and Clinical Research in Neuromodulation*, 15(6), 1495-1497.
 68. Baldermann, J. C., Kohl, S., Visser-Vandewalle, V., Klehr, M., Huys, D., & Kuhn, J. (2016). Deep brain stimulation of the ventral capsule/ventral striatum reproducibly improves symptoms of body dysmorphic disorder. *Brain Stimulation: Basic, Translational, and Clinical Research in Neuromodulation*, 9(6), 957-959.
 69. Satyal, M. K., Basso, J. C., Tegge, A. N., Metpally, A. R., & Bickel, W. K. (2021). A novel model of obesity prediction: Neurobehaviors as targets for treatment. *Behavioral neuroscience*, 135(3), 426. DOI: 10.1037/bne0000385
 70. Phillips, K. A., Menard, W., & Fay, C. (2006). Gender similarities and differences in 200 individuals with body dysmorphic disorder. *Comprehensive psychiatry*, 47(2), 77-87. DOI: 10.1016/j.comppsy.2005.07.002
 71. Rossetti, M. G., Delvecchio, G., Calati, R., Perlini, C., Bellani, M., & Brambilla, P. (2021). Structural neuroimaging of somatoform disorders: A systematic review. *Neuroscience & Biobehavioral Reviews*, 122, 66-78. DOI: 10.1016/j.neubiorev.2020.12.017
 72. Di Ponzio, M., & Pallanti, S. (2023). Deep Transcranial Magnetic Stimulation in combination with symptom provocation: An effective treatment in patients with treatment-resistant Body Dysmorphic Disorder?. *Neuroscience Applied*, 2, 101137. DOI: 10.1016/j.nsa.2023.101137