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Body Dysmorphic Disorder: Neurobiological Features and an Updated Model

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Abstract

Body Dysmorphic Disorder (BDD) affects approximately 2% of the population and involves misperceived defects of appearance along with obsessive preoccupation and compulsive behaviors. There is evidence of neurobiological abnormalities associated with symptoms in BDD, although research to date is still limited. This review covers the latest neuropsychological, genetic, neurochemical, psychophysical, and neuroimaging studies and synthesizes these findings into an updated (yet still preliminary) neurobiological model of the pathophysiology of BDD. We propose a model in which visual perceptual abnormalities, along with frontostriatal and limbic system dysfunction, may combine to contribute to the symptoms of impaired insight and obsessive thoughts and compulsive behaviors expressed in BDD. Further research is necessary to gain a greater understanding of the etiological formation of BDD symptoms and their evolution over time.

Keywords

BDD; neurobiology; pathophysiology; etiology; model

Introduction

Body dysmorphic disorder (BDD) is an often severe psychiatric disorder in which individuals are preoccupied with imagined defects in their appearance, which are not noticeable or appear slight to others (American Psychiatric Association., 2000). They subsequently experience significant distress, disability, and functional impairment, often accompanied by depression and suicidality (Phillips et al., 2005). In addition, they are often delusional in their beliefs (Eisen, Phillips, Coles, & Rasmussen, 2004), and tend to present to plastic surgeons and dermatologists more often than mental health clinicians (Phillips, Didie, Feusner, & Wilhelm, 2008).

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BDD affects approximately 1–2% of the population (Bienvenu et al., 2000; Faravelli et al., 1997; Otto, Wilhelm, Cohen, & Harlow, 2001; Rief, Buhlmann, Wilhelm, Borkenhagen, & Brahler, 2006), yet is still under-studied and under-recognized. Attesting to this, a recent Pubmed search for 'body dysmorphic disorder' generated 842 hits, whereas a search for 'obsessive compulsive disorder' generated 13532 hits. BDD has several important phenomenological features including obsessive thoughts and compulsive behaviors, distorted perception, poor insight, and difficulty engaging in treatments (Phillips, 2005). The pathophysiology behind BDD is complex and likely involves interactions between a variety of factors, some of which may contribute to development and others to the maintenance of BDD symptoms. However, the field of neurobiological research in BDD is still young, and there are many gaps in our knowledge of how BDD symptoms are formed and evolve over time.

This review explores the research findings regarding neurobiological abnormalities that may be associated with the etiology and pathophysiology of BDD. These include neurobiological factors that pertain to neurocognitive functioning, neurochemistry, brain activation relative to visual processing, morphometry, and genetic factors that have been associated with BDD. We attempt to synthesize the evidence from these various domains to generate a preliminary, heuristic model that integrates research findings to date.

Neurocognitive Functioning

Early research done in BDD to assess neurobiological functioning involved tests of neurocognitive functioning. Three different studies have been performed in BDD using neuropsychological measures to investigate domains of memory, executive functioning, motor functioning, and/or visuospatial functioning (Deckersbach et al., 2000; Dunai, Labuschagne, Castle, Kyrios, & Rossell, 2010; Hanes, 1998).

Hanes (1998) found that individuals with BDD and those with OCD performed poorly relative to healthy controls on tests of executive function, including response inhibition and planning. However, they performed normally on measures of verbal memory (Rey Auditory Verbal Learning Task), visuospatial construction and memory (Rey Osterreith Complex Figure Test – RCFT), verbal fluency, and motor function (Hanes, 1998).

Contrary to this, a later study found that individuals with BDD performed poorer than controls on the RCFT, as well as the California Verbal Learning Test (Deckersbach et al., 2000). On the RCFT, group differences in free recall were mediated by deficits in organizational strategies; the BDD group selectively recalled details instead of larger organizational design features. The authors interpreted this deficit in memory organization strategy to be most likely attributed to abnormalities in executive functioning. However, as this task involves viewing and encoding a complex visual figure, it is also possible that earlier perceptual abnormalities in global and/or local visual processing and/or differences in selective attention may have contributed to poor performance. The findings in this study may have clinical implications, as individuals with BDD tend to focus on details of their appearance at the expense of global aspects. These abnormalities, which may implicate

organizational difficulties, abnormal selective attention, and/or aberrant perception, may contribute to, or be involved in the maintenance of, BDD symptoms.

Dunai et al. (2010) performed a battery of executive functioning tests in BDD (Dunai et al., 2010). They found that BDD participants made significantly more between-search errors on the Spatial Working Memory Task and had slower subsequent thinking times in a Stockings of Cambridge test, used to probe deficits in planning.

Other neuropsychological studies in BDD have focused on processing of emotional or otherwise highly salient stimuli. Individuals with BDD appear to have deficits in facial emotion recognition. One study found that in self-referent situations, BDD patients were more likely to misinterpret neutral faces as angry or contemptuous compared to controls (Buhlmann, Etcoff, & Wilhelm, 2006). This may implicate brain regions and systems involved in facial emotion perception such as the inferior frontal cortex, right parietal cortex, occipito-temporal cortex, insula, striatum, and/or amygdala, as potentially dysfunctional in BDD (Adolphs, Demasio, Tranel, & Demasio, 1996; Gur, Skolnik, & Gur, 1994; Sprengelmeyer, Rausch, Eysel, & Przuntek, 1998). However, functional neuroimaging studies have yet to be conducted to investigate the role of these regions in emotion regulation in BDD. These findings are also consonant with the clinical observation that individuals with BDD often have self-referential delusions or overvalued ideations in regards to other people's emotional response to their perceived flaws. In another study, subjects were asked to interpret various ambiguous scenarios. BDD subjects were more likely than controls to report general situations, social situations, and body-focused situations as threatening, (Buhlmann, Wilhelm, et al., 2002). In an emotional Stroop task, BDD subjects had greater Stroop interference (more delayed response) for words related to their disorder and appearance ideal, i.e. 'beauty' or 'attractive' (Buhlmann, McNally, Wilhelm, & Florin, 2002). However, a study of negative priming found no difference between BDD and healthy control subjects in terms of cognitive inhibition as measured by response latencies, for appearance-related "threating" words (Wilhelm, Buhlmann, & McNally, 2003).

In sum, most studies of neurocognition in BDD have found evidence of abnormal executive functioning (specifically, planning, organization and response inhibition). There is also evidence of abnormalities in facial emotional recognition, and possibly heightened sensitivity to appearance-related stimuli and perceived social threats.

Neurochemistry

There is a small body of evidence for the role of serotonin in BDD. Marazziti et al. found decreased serotonin transporter binding density in OCD-related disorders, including BDD (Marazziti, Dell'Osso, & Presta, 1999). There is evidence from both controlled and uncontrolled studies that serotonin reuptake inhibitor (SRI) medications are effective treatments for BDD (see (Ipser, 2010; Phillips & Hollander, 2008) for review). Treatment with medications that have serotonin reuptake inhibition often result in less frequent and intense preoccupations, better control over impulsivities, and decreased BDD-related distress (Allen et al., 2008; Phillips & Hollander, 2008). Other evidence for the involvement

of serotonin in BDD includes a case study in which BDD symptoms were exacerbated during dietary depletion of tryptophan (a serotonin precursor) (Barr, Goodman, & Price, 1992). Another case study found a serotonin agonist, psilocybin, led to decreased BDD symptoms (Hanes, 1996).

The role of serotonin in the pathogenesis of BDD as of yet is uncertain. The strongest evidence of an association with serotonin is that BDD symptoms are often improved through treatment with SRIs. However, all of the aforementioned studies only provide indirect evidence of a relationship between serotonergic systems and BDD, and do not prove serotonergic abnormalities underlie BDD pathophysiology. An investigation of serotonergic abnormalities using PET, similar to what has already been performed in individuals with OCD (Berney et al. 2011; Perani et al. 2008) could provide more definitive evidence of the role of serotonin in BDD.

Genetics and Heritability

Thus far there have been limited studies investigating genetic factors underlying BDD. Nevertheless, heredity and genetic factors do appear to contribute to BDD; for example, 8% of individuals with BDD have a family member also diagnosed with BDD, a statistic 4–8 times the prevalence in the general population (Bienvenu et al., 2000). A twin study in females that utilized self-report measures of dysmorphic concerns and concerns about body odor and body malfunction from a UK twin registry found genetic factors accounted for approximately 44% of the variance of dysmorphic concerns (Monzani et al., 2011). The same group found in another twin study that up to 64% of the covariation between body dysmorphic and obsessive-compulsive traits was accounted for by common genetic factors (Monzani et al., 2012).

Additional evidence for a heritable connection with OCD comes from family studies. In one study, 7% of BDD patients had a first-degree relative with OCD (Phillips, Gunderson, Mallya, McElroy, & Carter, 1998). There is a six times higher lifetime prevalence of BDD in first-degree relatives of OCD probands, compared to relatives of controls (Bienvenu et al., 2000).

In terms of specific genes there has only been one preliminary study published to-date in BDD, using candidate genes. Richter et al. (2004) found an association between the GABA (A)-gamma-2 1(A) allele and BDD, as well as comorbid BDD-OCD (Richter et al., 2004). The same study demonstrated an association of BDD with the serotonin transporter promoter polymorphism short allele.

Overall, these studies show that susceptibility for BDD, among other factors, may be heritable. Moreover, there may be shared genetic traits between BDD and OCD.

Behavioral and Neuroimaging Evidence of Abnormal Visual Processing

Abnormal visual information processing is a phenotype that may contribute to the phenomenology of BDD. Clinically, individuals with BDD experience distortions of self-perception of appearance. This likely causes or contributes to preoccupation with physical

defects, the conviction of disfigurement and ugliness, and subsequently to poor insight or delusionality. These phenomenological observations, as well as the neuropsychological study demonstrating impaired performance on the RCFT mediated by greater reproduction of detailed relative to global design features (Deckersbach et al., 2000) suggest possible disturbances in visual perception and/or visuospatial information processing.

The first functional neuroimaging study to investigate visual perception in BDD examined visual processing of others' faces (Feusner, Townsend, Bystritsky, & Bookheimer, 2007). Twelve BDD subjects and 13 healthy controls underwent functional magnetic resonance imaging (fMRI) while matching photographs of others' faces. Some of the faces were digitally altered to remove the high or low spatial frequencies, to create images that contained configural or detail information, respectively. This study found left hemisphere hyperactivity in an extended face-processing network for normal and low spatial frequency images. This pattern, in contrast to the generally right hemisphere-dominant pattern for healthy controls (Haxby et al., 1994), suggests greater detail encoding and analysis relative to holistic and configural processing, even for face images that contain low level of detail. Abnormal interhemispheric sharing of information may also be involved. Another interesting finding in the study was abnormally high activation of amygdalae in the BDD group for the low- and high spatial frequency images. In contrast, the control group showed normal activation of the amygdalae for the NSF task, and reduced activity for the low- and high spatial frequency images. This suggests an abnormal hyper-responsivity of the amygdala in BDD relative to controls for images that contain low and high levels of detail.

Another investigation of other-face processing, this one a psychophysical study, showed that individuals with BDD have abnormalities in identity recognition for faces with emotional expressions (Feusner, Bystritsky, Hellemann, & Bookheimer, 2010). The poor performance in the BDD group did not depend on the type of emotional expression. This suggests general abnormalities in visual information processing of faces, which may be more pronounced when a face, in general, has an emotional expression.

Feusner et al. (2010) conducted an fMRI study of own-face processing in 17 individuals with BDD and 16 healthy controls. They found abnormal hypoactivity in the BDD group in visual cortex (striate and extrastriate regions) for low spatial frequency images, and hyperactivity in frontostriatal systems (orbitofrontal cortex and caudate) for normal images (Feusner, Moody, et al., 2010). In addition, BDD symptom severity as measured by the BDD version of the Yale–Brown Obsessive–Compulsive Disorder Scale (BDD-YBOCS) (a measure of severity and impairment from obsessive thoughts, compulsive and avoidant behaviors, and insight) (Phillips et al., 1997), was correlated with frontostriatal activity and activity in extrastriate visual cortex. Despite the BDD group rating the viewing of their face as being highly aversive, they did not demonstrate greater amygdala or insula activity. This study provides preliminary evidence of similar aberrant orbitofrontal-striatal circuit activity in BDD and OCD (Rotge et al., 2008), which may be associated with obsessive thoughts and compulsive behaviors in both cases.

The same group conducted another fMRI experiment in order to investigate abnormalities in visual processing in BDD for non-appearance related stimuli (Feusner, Hembacher, Moller,

& Moody, 2011). Fourteen BDD subjects and 14 healthy controls were scanned while they matched photographs of houses that were normal, or contained only high- or low spatial frequency information. The BDD group relative to the control group showed abnormal hypoactivity in secondary visual processing systems for low spatial frequency images. This provides evidence of abnormal global and holistic processing (as this type of information is conveyed by low spatial frequency images), for non-appearance related stimuli, suggesting more general abnormalities in visual processing.

An imbalance between local (detail) and global (holistic) processing in BDD was also found in a psychophysical study of inverted faces (Feusner, Moller, et al., 2010). Eighteen BDD subjects and 17 healthy controls performed a face recognition task with sets of upright and inverted (upside-down) faces. Normally, recognition of inverted faces is less accurate and slower relative to upright faces, attributed to the absence of a holistic template for inverted faces (Farah, Tanaka, & Drain, 1995); this is termed the “face inversion effect.” Results from this study indicated that the inversion effect for response time was smaller in BDD subjects than controls during the long duration stimuli (due to faster processing of inverted faces than controls), but was not significantly different during the short duration stimuli. This suggests that BDD individuals may have a propensity to engage in highly detailed processing of faces, whether upright or inverted. Controls, on the other hand may primarily engage holistic processing for upright faces, yet have to rely on detailed processing for inverted faces. If so, this may have conferred the BDD group’s advantage in speed of responses for inverted faces. This was only observed for the long viewing duration condition, likely because this condition allowed sufficient time for encoding of details. For short-duration stimuli, on the other hand, there was likely insufficient time to process details, only allowing for holistic processing. The fact that the inversion effect was normal in BDD subjects for short viewing durations therefore suggests that an imbalance in detail vs. holistic processing may be a dynamic phenomenon that emerges only in situations in which viewing durations are long. Clinically, this occurs on a daily basis in most individuals with BDD, as they often spend many minutes or even hours at a time viewing themselves in mirrors and reflective surfaces (Phillips, 2005).

The ability to detect aberrancies in facial features or asymmetry is another aspect of visual processing that has been investigated in BDD. Evidence that BDD may involve perceptual distortions for own-face processing comes from a study in which BDD subjects perceived distortions of digital images of their faces that were not actually present (Yaryura-Tobias et al., 2002). Another study investigated the ability to detect asymmetry in BDD (Reese, McNally, & Wilhelm, 2010). This has clinical relevance, as some individuals with BDD perceive defects of their appearance related to asymmetry. In addition, a theory of symptom formation in BDD relates to the possibility that individuals have enhanced aesthetic sensitivity (Veale, 2009), which could include noticing imperfections including asymmetry that are not noticed by others. To investigate this, Reese et al. (2010) enrolled 20 BDD subjects, 20 OCD patients and 20 healthy controls who viewed sets of others’ faces that were altered in symmetry (Reese et al., 2010). Individuals with BDD were not significantly more accurate or faster than healthy controls in detecting differences in facial symmetry. Another study (Stangier, Adam-Schwebe, Muller, & Wolter, 2008) found that the BDD group was more accurate in detecting changes in aesthetic features of others’ faces. An

important difference between the Stangier et al. (2008) study and the Reese et al. (2010) study is that in the former the view time was limited to 200 ms and in the latter the view time was unlimited and subjects were not told to respond as quickly as possible. In addition, in the Stangier et al. (2008) study the subjects were asked to identify changes in facial *details* (with the exception of distance between the eyes, which is more of a configural judgment). In the Reese et al. (2010) study, judging facial symmetry would most likely engage configural processing. Thus, enhanced detail processing in BDD may explain performance advantages relative to controls for inverted faces as well as for change detection for facial features of others' faces. The difficulty in synthesizing the results from these experiments comes from the fact that they used different tasks, time durations, own or others' face stimuli, and endpoint measures (accuracy or reaction time), all of which may affect outcome.

In summary, there is evidence of abnormal visual processing in BDD. The findings in the functional neuroimaging studies suggest imbalances in detailed vs. global/configural processing marked by abnormalities in primary and/or secondary visual cortical, temporal, and prefrontal systems. Moreover, this overall pattern is evident for own-face, other-face, and inanimate object stimuli. The behavioral (psychophysical) studies provide evidence for enhanced detail processing, with reduced face inversion effect and enhanced ability to detect changes in facial features.

Morphometric Studies

There have only been three small studies in BDD that have investigated volumetric brain morphometry. A study of females with BDD compared to healthy controls found greater total white matter and a relative leftward shift in caudate asymmetry (Rauch et al., 2003). A study of males also found greater total white matter, as well as smaller anterior cingulate and orbitofrontal cortex and a trend for larger thalamic volumes (Atmaca et al., 2010). Both studies provide evidence for abnormalities in frontostriatal systems. The third (Feusner et al., 2009) did not find volumetric differences between groups, but found that symptom severity as measured by the BDD-YBOCS correlated significantly with volumes of the left inferior frontal gyrus (IFG) and the right amygdala.

Neurobiological Model for Pathophysiology of BDD

Here we attempt to integrate the presented findings into a preliminary model for understanding the pathophysiology of BDD as it pertains to neurobiological abnormalities. As with most psychiatric disorders, the governing pathophysiology for BDD is complex and unlikely to be encompassed by a single domain. Importantly, neurobiological models alone are also likely to be insufficient in explaining such a complex disorder; interpersonal, cognitive-behavioral, psychodynamic, and cultural contributions are also critical factors to consider.

In BDD, aberrant interactions between networks and regions as well as neurotransmitter and neurochemical systems may have etiological roles, or else may represent secondary sequelae of the illness. Up to this point the limited research into this disorder, particularly due to the fact that most studies have involved small number of subjects and have not been replicated,

precludes drawing firm conclusions about the neurobiology of BDD and hampers the development of a well-supported model. However, from the extant research there are patterns that have begun to emerge across studies. One pattern is that of abnormalities in frontostriatal systems, as evidenced by neurocognitive (impaired executive functioning) and functional and structural neuroimaging studies (Feusner et al. 2010). Similar patterns of frontostriatal hyperactivity and dysfunction are evident in many studies of OCD (for reviews see (Whiteside, Port, & Abramowitz, 2004) and (Menzies et al., 2008)), although it is important to note that other regions of abnormalities have been identified, such as in the parietal and visual cortices. In addition, improvement of BDD symptoms through modulation of the serotonergic system with SRIs, could be the result of mechanistic action at the level of frontostriatal circuits or the limbic system (Blier, Habib, & Flament, 2006; Furmark et al., 2002). Another pattern that has emerged involves abnormalities in visual processing for appearance and non-appearance related stimuli, as evidenced by findings in functional neuroimaging and psychophysical studies. These appear to follow a pattern of enhanced detail and/or impaired global and configural processing. In addition, there is evidence of abnormal emotional processing, as evidenced by impairments in facial emotional recognition and abnormal patterns of amygdala activity. There is also emerging evidence that there may be heritable genetic factors involved in BDD, and possibly a heritable link between BDD and OCD.

A preliminary neurobiological model of BDD symptoms thus needs to take into account abnormalities that span domains of perception, emotional processing, planning, organization, response inhibition, and patterns of obsessive thoughts and compulsive behaviors. Abnormalities in visual perception, originating in primary and secondary visual processing systems, may represent a sensory deficit that provides an initial distorted percept. This may subsequently be modulated by impaired emotional processing and aberrant frontostriatal systems (including impaired response inhibition and visuospatial organization), which could give rise to inability to inhibit obsessive thought patterns and concomitant urges to perform compulsive and avoidant behaviors. A distorted perception, further erroneously validated by impaired perception of others' emotional reactions towards them (thus contributing to poor insight and delusionality) would then be maintained and perpetuated over time by an inability to control thoughts and behavioral patterns.

Future Research Directions

To help test and refine this and other models, further neurobiological research in BDD is imperative. In particular, as most studies thus far have been cross-sectional, it is not possible to discern if the pathophysiological deficits are etiological or secondary to other aspects of the illness that have developed over time. To elucidate this, longitudinal studies of children at risk will be useful, particularly in relation to developmental changes that occur during adolescence, when the disorder typically onsets. In addition, studies of unaffected first-degree relatives will help identify likely inherited phenotypes and endophenotypes. A better integration of brain imaging modalities such as electroencephalography (EEG), magnetoencephalography (MEG), fMRI, and structural (DTI and volumetric/cortical thickness) imaging could uncover important structure-function relationships as well as develop a better understanding of the time-course and dynamics of brain activity. Future

imaging studies employing functional and effective connectivity methods could further elucidate abnormalities in corticostriatal systems, visual processing systems, and interactions between the two. Once dysfunctional systems have been identified (e.g. specific circuits within frontostriatal systems, and/or subsystems of visual processing networks such as dorsal or ventral visual stream), further neurochemical investigations of neurotransmitter systems that may mediate abnormalities would be useful. In addition, because of the evidence of shared heredity and overlap in phenomenology and comorbidity, direct comparisons of individuals with BDD to those with OCD could elucidate specific neurobiological factors that may represent these shared traits or susceptibility factors. Similar investigations should also be conducted across other possibly related disorders such as eating disorders and social phobia.

Conclusions

BDD appears to be a complex disorder in which heritable factors related to dysmorphic concerns and obsessive-compulsive traits, as well as other biological susceptibilities may combine in certain individuals with family, interpersonal, and cultural experiences to lead to the development of BDD. Neurobiological dysfunctions span several domains that have been studied thus far, including neurocognitive, neurochemical, visual and emotional processing systems, and genetics. We propose a neurobiological model for BDD that includes visual and emotional processing abnormalities and frontostriatal and limbic system dysfunction. These may combine to contribute to the symptoms of perceptual distortions and impaired insight, as well as obsessive thoughts and compulsive behaviors. Further research is necessary to gain a greater understanding of the etiological formation of BDD symptoms and their evolution over time, and will be important in aiding intervention strategies and the development of improved treatments.

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