

Review article

The pathophysiology of body dysmorphic disorder

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Abstract

Body dysmorphic disorder (BDD) is an often severe and disabling condition, affecting up to 2% of the population. Despite its prevalence and clinical significance, very little is known about the pathophysiology of BDD. However, clues to its possible neurobiological substrates and abnormalities in information processing are starting to emerge. This article reviews findings from genetic, brain lesion, neuroimaging, neuropsychological, and psychopharmacological studies that have allowed us to develop a tentative model of the functional neuroanatomy of BDD. There is likely a complex interplay of dysfunctions in several brain networks underlying the pathophysiology of BDD. A combination of dysfunctions in frontal-subcortical circuits, temporal, parietal, and limbic structures, and possibly involving hemispheric imbalances in information processing, may produce both the characteristic symptoms and neurocognitive deficits seen in BDD. An improved understanding of the pathophysiology of BDD will be crucial to guide the development of better treatments.

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Introduction

Body dysmorphic disorder (BDD) is defined by the *Diagnostic and Statistical Manual of Mental Disorders, 4th ed.* (DSM-IV-TR) (American Psychiatric Association, 2000) as a preoccupation with an imagined defect in physical appearance or excessive concern about a slight physical anomaly that causes significant impairment or distress. It is believed to affect close to 2% of the general population (Rief, Buhlmann, Wilhelm, Borkenhagen, & Braehler, 2006), and up to 13% in psychiatric settings (Grant, Kim, & Crow, 2001; Phillips, Nierenberg, Brendel, & Fava, 1996; Wilhelm,

Otto, Zucker, & Pollack, 1997). BDD is an under-recognized disorder that causes significant suffering, disability, and functional impairment (Phillips, 2000; Veale et al., 1996).

Very little is known about the etiology or pathophysiology of BDD, as few studies have addressed this directly. This review of the pathophysiology of BDD explores what has been elucidated thus far from research on the genetics, neuroanatomy, neuropsychology, and psychopharmacology of BDD, as well as secondary BDD symptoms resulting from brain damage and medical illnesses. In addition, the brain networks that mediate body image distortion, self-recognition, and emotional reactions to visual stimuli are reviewed. This information is synthesized to produce preliminary hypotheses of the pathophysiological processes most likely to mediate the symptoms of BDD, in the interest of stimulating further research in this area.

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Genetics of BDD

Genetic factors are likely to play an important role in the etiology of BDD, as evidenced by its pattern of heritability (Bienvenu et al., 2000). Eight percent of individuals with BDD have a family member with a lifetime diagnosis of BDD, which is four to eight times the prevalence in the general population. BDD shares heredity with obsessive-compulsive disorder (OCD), as family studies have shown that 7% of BDD patients were found to have a first-degree relative with OCD (Phillips, Gunderson, Mallya, McElroy, & Carter, 1998), and first-degree relatives of OCD probands have a six times higher lifetime prevalence of BDD than do relatives of controls (Bienvenu et al., 2000).

There has been scant research on molecular genetic data to inform our understanding of the etiology of BDD. Only one preliminary genetic association study has been performed thus far. Richter et al. (2004) found an association between the gamma-aminobutyric acid (GABA) A - γ 2 gene and BDD and comorbid BDD + OCD, but not with OCD alone. There was also a trend toward association with the serotonin transporter promoter polymorphism (5-HTTLPR) short allele (Richter et al., 2004). This area of research clearly needs to expand.

Neuroimaging studies in BDD

Brain imaging studies can be extremely useful in identifying structural and functional brain abnormalities, and can be designed to parse out specific cognitive and emotional processes that may contribute to symptomatology in patients with a given disorder. Functional brain imaging research has led to a greater understanding of the neurobiological mediation of OCD, for example, indicating that OCD symptoms are mediated by overactivity along a neural circuit connecting the orbitofrontal cortex, basal ganglia, and thalamus (Saxena, Bota, & Brody, 2001; Saxena, Brody, Schwartz, & Baxter, 1998).

Unfortunately, only two brain imaging studies of BDD have been published thus far. A preliminary volumetric magnetic resonance imaging (MRI) study found leftward shift in caudate volume asymmetry and greater total white matter volume in eight women with BDD than in eight female controls (Rauch et al., 2003). This study implicates similar regions or networks in BDD as in OCD, although previous findings in OCD are a rightward shift in caudate asymmetry and lesser total white matter (Saxena et al., 2001). A small functional imaging study of six BDD patients, using single photon

emission computed tomography (SPECT), showed variable, discrepant findings—relative perfusion deficits in bilateral anterior-medial temporal and occipital regions and asymmetric perfusion in parietal lobes (Carey, Seedat, Warwick, van Heerden, & Stein, 2004). This study, however, had no control or comparison group and did not make any quantitative measurements of regional brain activity. Moreover, two of the six BDD patients had comorbid major depression, and one had comorbid OCD, making it difficult to know whether the perfusion abnormalities were associated specifically with BDD or due to the comorbid conditions.

Recently, the first functional imaging study to compare BDD patients to controls examined visual information processing of faces, with respect to spatial frequency (Feusner, Townsend, Bystritsky, & Bookheimer, 2006). Twelve BDD patients and 12 healthy controls underwent functional magnetic resonance imaging (fMRI) while matching photographs of faces. Some of the faces were digitally altered to remove the high or low spatial frequencies, which created images that contained configural or detail information, respectively. BDD participants showed greater left hemisphere activity relative to controls for all face tasks, particularly in lateral aspects of the prefrontal cortex and the temporal lobe. They also activated dorsal anterior cingulate gyrus for the low spatial frequency (LSF) face task. Controls, on the other hand, activated left-sided prefrontal cortex and dorsal anterior cingulate gyrus only for the high spatial frequency (HSF) face task. Greater left-sided activity for LSF and normal faces suggests a predominance of detail encoding and analysis, a pattern evident in controls only for HSF faces. This suggests that BDD patients may process faces in a piecemeal manner, while healthy controls' perception of faces may be more configural and holistic. These laterality patterns in the BDD participants suggest a bias for local, or detail-oriented, processing of faces over global processing.

Another finding in the BDD group was abnormal activation of amygdalae for the LSF and HSF face tasks. In contrast, the controls showed activation of the amygdalae for the NSF task, but reduced activity or deactivation for the LSF and HSF tasks. Amygdala activation did not correlate with any behavioral measures. This suggests an abnormal hyper-responsivity of the amygdala that appears specific to LSF and HSF visual information.

The results from this fMRI study suggest that BDD participants show fundamental differences from controls in visual processing, with different laterality of activation patterns in areas representing an extended visual processing network, and abnormal amygdala

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