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What Causes BDD: Research Findings and a Proposed Model

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The etiology and pathophysiology of body dysmorphic disorder (BDD), as with most psychiatric disorders, is likely complex. Because research is limited, the causative and contributory factors to the development of BDD are unclear. Yet there is emerging evidence from research studies of various factors that may contribute to the development and maintenance of BDD symptoms.

This review of the etiology and pathophysiology of BDD explores what has been elucidated thus far from research on developmental, psychosocial, cognitive and behavioral, neuropsychological, and neurobiological (visual processing, neuroanatomical, genetic, and neurochemical) factors. We explore "submodels" of BDD within these domains, and we synthesize these to generate an overarching, yet preliminary, model of the etiological and pathophysiological processes that appear to contribute to the development and maintenance of BDD. This model involves a context of likely biological and genetic susceptibility upon which adverse life events interact with cognitive distortions and subsequent learned behavior to result in BDD symptoms. Given the limited research base, this model – while reflecting available research evidence – serves primarily a heuristic function. We hope nonetheless that this model informs clinicians' thinking about how to understand patients with BDD.

DEVELOPMENTAL FACTORS

Developmental factors in BDD are under-researched relative to other disorders of similar prevalence, and hence remain largely unknown. Nevertheless, there is preliminary evidence that sexual, emotional, and physical abuse in childhood may be associated with BDD. One study found that 38% of 50 BDD patients reported some form of abuse during childhood (28% emotional abuse; 22% sexual abuse; 14% physical abuse) as compared to 14% of a comparison group of 50 OCD patients¹. In a study of 75 BDD subjects who completed the Childhood Trauma Questionnaire, 78.7% reported a history of childhood maltreatment (68.0% emotional neglect; 56.0% emotional abuse; 34.7% physical abuse; 33.3% physical neglect; and 28.0% sexual abuse)². However, because these two studies are cross-sectional, they cannot establish a causative role for childhood trauma in the development of BDD.

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BDD most often begins in adolescence³, a period in development of accelerated changes in physical appearance as well as hormonal milieu; these factors may potentially contribute to, or trigger, the development of BDD, but have yet to be studied directly.

SOCIAL FACTORS

Social interactions can be a significant source of childhood adversity for some. Teasing related to physical stigmata, such as acne, may have a long-term impact on an individual, negatively affecting one's thoughts and emotions. Once the negative association is made between the appearance feature and others' reactions to it, everyday interactions and experiences can continue to imprint these thoughts and emotions, which may be theorized to contribute to symptoms of BDD⁴. In one study, individuals with BDD reported more incidences of teasing, especially about appearance and competency, than mentally healthy controls⁵.

While childhood adversity as a risk factor is relatively nonspecific, heightened aesthetic sensitivity may be more specific to BDD. Aesthetic sensitivity refers to awareness and appreciation of beauty and harmony and includes symmetry, averageness, secondary sexual characteristics, and general attractiveness⁶. There is evidence that individuals with BDD have heightened aesthetic sensitivity⁷, However, it is unclear whether this is the result of specific social influences, a trait that predisposes to BDD, or the result of having BDD symptoms.

CULTURAL INFLUENCES

Cases of BDD have been reported in various countries, including the US, Canada, Australia, China, Japan, South America, and the former Soviet Union among others⁸. Bohne et al. (2002) conducted the only cross cultural study published to date and found that BDD prevalence rates are fairly similar between American (4%) and Germans students (5.3%)⁹. Cultural factors may play a role in which body parts are of concern, and how other BDD symptoms are expressed, as different cultures (as well as races or ethnicities) may have variations in aesthetic standards of beauty¹⁰, but this has not yet been studied in relation to BDD. On the other hand, there is evidence of invariant standards of beauty such as averageness, symmetry, and sexual dimorphism; these appear to cross culture, gender, and age, and emerge early in development before individuals experience the influence of culture and the media¹¹.

COGNITIVE-BEHAVIORAL AND LEARNING MODELS OF BDD

Cognitive-behavioral and learning models can provide a formulation for how biological, cultural, and social factors lead to the development and maintenance of BDD, and are based on classical and operant conditioning¹². These models mainly serve a heuristic function, as research is needed to test conditioning effects in individuals with BDD.

Operant conditioning (based on reinforcement) coupled with social learning results in the development of values and beliefs about attractiveness, including a sense of value conditioned on body image¹³. Early experiences that positively reinforce an individual for appearance may play an important role in BDD development, but have yet to be studied directly.

In the framework of classical conditioning, negative events involving one's physical appearance (e.g. teasing at the onset of puberty) may serve as unconditioned stimuli and cause an unconditioned negative emotional response (e.g. anxiety, disgust, or shame). Anything paired with this is also evaluated as negative, such as words or images of the body

part. Higher order conditioning may account for additional appearance concerns secondary to the patient's primary concern. As a result of higher-order conditioning the negative emotional reaction triggered by exposure to the primary body part of concern may generalize to other body parts that are noticed during this negative emotional state¹³.

Once BDD symptoms have been established and appearance-related beliefs, assumptions, and values developed, it has been hypothesized that secondary operant conditioning in the form of negative reinforcement serves to maintain the maladaptive behaviors and cognitions^{13, 14}. More specifically, BDD individuals' compulsive behaviors serve to reduce short-term distress by alleviating the negative emotional reaction triggered by either an intrusive thought about perceived appearance flaws or contact with the perceived flaw. For example, BDD patients often avoid looking into mirrors, which may result in relief as a result of not coming in contact with their image. This relief represents a negative reinforcer in that it increases the probability that the avoidance behavior will be used again when in a similar situation. However, this example of mirror use is complicated; mirror-checking is a common behavior in BDD and may also represent negative reinforcement since the patient's negative emotional reaction may be temporarily relieved when they perceive (at times) that they look less defective.

NEUROCOGNITIVE FUNCTIONING

Researchers have begun to examine neurocognitive functioning in BDD, which may shed light on the etiology and maintenance of BDD's key symptoms. When presented with a complex visuospatial task (Rey-Osterrieth Complex Figure Test, ROCF) and the instruction to draw the figure, individuals with BDD tended to overfocus on details, which interfered with their ability to encode and ultimately recall the figure¹⁵. In a recent study female BDD subjects, relative to women with or without a disfiguring dermatological condition, were significantly more accurate at rating the degree of distortion of faces in which a feature (e.g., nose) had been changed¹⁶. Thus, BDD subjects appear to be more likely than others to notice minor deviations from beauty standards. These neuropsychological deficits related to an excessive focus on detail (versus global) processing, and sensitivity to perceived flaws may be related to one of the core clinical features of BDD. For example, when gazing in the mirror, BDD patients often selectively focus on minor flaws while ignoring global aspects of their appearance. This appears to trigger shame, anxiety and increased appearance preoccupations.

Recent research has also examined emotion recognition in BDD¹⁷. Subjects were presented with photographs of faces varying in emotional expressions¹⁸ and answered two sets of questions: one including self-referent scenarios (e.g.,, "Imagine that the bank teller is looking at you. What is his facial expression like?") and one with other-referent scenarios (e.g., "Imagine that the bank teller is looking at a friend of yours...."). Subjects then were instructed to identify the emotional expression of the face. BDD subjects misinterpreted significantly more neutral expressions as contemptuous and angry when compared to healthy controls. This bias was found only in self-referent scenarios but not in other-referent scenarios. These results are consistent with prior research on emotion recognition in individuals with BDD¹⁹ and with the ideas and delusions of reference often experienced by individuals with BDD (e.g., thoughts that others laugh about or stare at the perceived flaw). Moreover, in a study in which BDD patients were asked to interpret various ambiguous scenarios, they reported threatening interpretations for general situations, social situations, and body-focused situations, whereas healthy control subjects showed no bias and OCD subjects had a bias toward threatening interpretations only for general situations²⁰. It is possible that biases to interpret situations as threatening and faces as contemptuous might exacerbate social anxiety and social avoidance in BDD.

In an emotional Stroop study, BDD subjects and healthy control subjects were presented with words varying in color and emotional valence. Individuals with BDD selectively attended to words like "beauty" and "attractive," delaying their responses to these words²¹. This finding is consistent with clinical observations that they are preoccupied with their appearance ideal. In another recent study individuals with BDD, individuals with subclinical BDD symptoms, and healthy control participants participated in the Implicit Association Test (IAT)²². The IAT requires the rapid categorization of various stimuli, and easier pairings (and faster responses) are interpreted as being more strongly associated in memory. In comparison to control participants, BDD participants had significantly lower implicit selfesteem (automatic beliefs about one's self that occur outside of conscious awareness or control), and the subclinical BDD participants were intermediate between these groups²³. BDD participants also had significantly stronger implicit associations between attractive and competent than the other groups. Attractive Competent and Self-Esteem scores were significantly associated with BDD symptom severity, distress, and avoidance during a mirror exposure task, suggesting that maladaptive behaviors in BDD might be fueled by automatic processes outside of conscious control.

Furthermore, when rating the attractiveness of various photographs of faces, including their own, individuals with BDD underestimated their own attractiveness and overestimated the attractiveness of beautiful faces of others²⁴. BDD subjects also endorsed high levels of perfectionistic thinking. (In terms of other personality traits, individuals with BDD have also shown high levels of neuroticism and low extroversion)²⁵.

These findings require replication, and they do not necessarily indicate that these factors are causal mechanisms in BDD, as the studies are cross-sectional. In other words, they do not tell us whether the above processes contribute to BDD, result from BDD, both, or neither. Nonetheless, this research informs our conceptualization of this disorder and forms the basis of several proposed models of the development and maintenance of BDD^{4, 26, 27}. Taken together, these findings indicate that individuals with BDD appear to selectively attend to details and are particularly sensitive to appearance flaws. They may also falsely interpret others' facial expressions as negative, and may misinterpret every-day social situations as threatening. BDD sufferers appear to be preoccupied with their beauty ideal and strongly associate being attractive with being competent. They appear to overestimate the attractiveness of beautiful others and underestimate their own attractiveness. These information-processing biases likely lead to feelings of shame, depression and anxiety.

NEUROBIOLOGICAL FACTORS

Visual Processing

Clinical observation suggests that individuals with BDD may have perceptual distortions, as they perceive defects in their appearance that are not observable or appear minor to others. These observations, in addition to above-mentioned neuropsychological findings of aberrant visuospatial processing¹⁵, suggests possible disturbances in visual perception and/or visuospatial processing.

The first neuroimaging study to investigate this in BDD examined visual processing of others' faces²⁸. Twelve BDD subjects and thirteen healthy controls were scanned with functional magnetic resonance imaging (fMRI) while matching photographs of faces that were unaltered or altered to contain primarily low detail (conveying configural/holistic information) or high-detail information. BDD subjects demonstrated greater left hemisphere activity relative to controls in an extended face-processing network for all image types (particularly lateral prefrontal cortex and temporal lobe), which was most prominent for the

low-detail faces. This imbalance in laterality suggests greater detailed and piecemeal face processing and lesser configural and holistic processing.

A subsequent fMRI study in BDD examined *own*-face processing. Seventeen BDD subjects and sixteen healthy controls²⁹ viewed photographs of their own face and a familiar face that were similarly unaltered or altered to include only high- or low-detail visual information. BDD subjects demonstrated relative hypoactivity in primary and secondary visual cortical systems for low-detail images of their own and familiar faces, suggesting abnormal brain activity for configural and holistic elements. In addition, BDD subjects demonstrated hyperactivity in left orbitofrontal cortex and bilateral head of the caudate when viewing unaltered photos of their own face compared to a familiar face. Aversiveness ratings were associated with lesser activity in occipital regions for low-detail faces. Moreover, frontostriatal activation correlated with severity of BDD-related obsessive thoughts and compulsive behaviors.

To better understand whether individuals with BDD have more general abnormalities in visual processing, the same group performed an fMRI experiment in which 14 BDD subjects and 14 healthy controls matched photographs of houses²⁹. The BDD group demonstrated abnormal relative hypoactivity in left visual association areas, including the parahippocampal place area, for low-detail images and abnormal relative hyperactivity of prefrontal systems for high-detail images. Controlling for depression symptom severity resulted in relative hypoactivity in dorsal visual stream (used for configural and holistic as well as motion processing) for all stimulus types. Severity of BDD symptoms also correlated with decreased activity in dorsal visual stream. This abnormal hypoactivity of visual systems for configural and holistic processing in non-symptom-related stimuli suggests general abnormalities in lower- and higher-order visual processing in BDD.

Other evidence suggesting an imbalance in local (detail) vs. global (holistic) processing comes from a study of inverted faces³⁰. Eighteen BDD subjects and 17 healthy controls viewed sets of others' faces that were upright or inverted. Inverted faces resulted in less slowing of response time in BDD subjects relative to healthy controls. This finding suggests a greater reliance on part decomposition and detail processing, for which inversion has less of an effect than holistic processing (due to the fact that humans have a holistic template for upright faces)³¹. However, for the subset of short-duration stimuli, which only allow time for configural/holistic processing, the inversion effect was normal. This suggests that the imbalance of local over global processing emerges only after longer viewing times.

Taken together, these studies, while requiring replication, provide converging evidence of abnormal visual processing in BDD. Specifically, visual systems responsible for configural and holistic processing demonstrate relative hypoactivity for a variety of stimuli, including one's own and others' faces, as well as non-face objects. This suggests a model of distorted visual processing in which details are not contextualized or integrated into a whole percept. This may be associated with patients' propensity to focus on details of their appearance while seeming less able to view themselves in an integrated, holistic manner. The inverted faces study additionally suggests that this global vs. local imbalance may depend on viewing duration, such that at viewing durations of several seconds to several minutes there is a greater propensity for local/detailed processing. There also appears to be abnormal hyperactivity in fronto-striatal systems specifically for own-face viewing, which may be associated with tendency for obsessive thoughts and compulsive BDD behaviors (see Didie et al in this issue). A similar pattern of fronto-striatal hyperactivity with symptom provocation has been found in studies of OCD, particularly for checking subtypes³².

Brain Morphometry in BDD

Only three small morphometric neuroimaging studies have been performed in BDD. A study of females found a leftward shift in caudate volume asymmetry and greater white matter volume in BDD than healthy control subjects³³. A study of males similarly found greater white matter volume in BDD relative to healthy control subjects, as well as smaller orbitofrontal cortex and anterior cingulate and a trend for larger thalamic volumes³⁴. These studies' results are consistent with abnormalities in fronto-striatal systems. However, a third study of males and females found no significant volumetric differences in BDD vs healthy controls³⁵.

Genetics

Susceptibility for BDD may be heritable. This is evidenced by the observation that 8% of individuals with BDD have a first-degree family member with a lifetime diagnosis of BDD³⁶, which is about 3–8 times the prevalence in the general population. Seven percent of BDD patients also have a first-degree relative with OCD³⁷, and first-degree relatives of OCD patients have six times the lifetime prevalence of BDD than controls³⁶, showing shared heritability between BDD and OCD. Despite limited research on specific gene involvement in BDD, in a preliminary candidate gene study of 57 BDD subjects and 58 healthy controls matched for ethnicity and gender, association was demonstrated for GABA_A- γ 2 (5q31.1-q33.2) (p=.032), with the 1 (A) allele occurring more frequently in BDD subjects than controls³⁸.

Neurochemistry of BDD

There is some preliminary evidence suggesting a role for serotonin function in BDD. One study found decreased platelet serotonin transporter binding density in OCD and OCD-related disorders, including BDD³⁹. In a case study dietary depletion of tryptophan (a serotonin precursor amino acid) led to exacerbation of BDD but not OCD symptoms in a patient with BDD and OCD⁴⁰. In other case reports, the serotonin receptor partial agonist psilocybin led to decreased BDD symptoms⁴¹, and a serotonin antagonist led to the onset of BDD symptoms in a patient⁴². On the other hand, a mixed serotonin agonist led to increased preoccupation with perceived body defects in BDD in another case⁴³, suggesting that the role of serotonin in BDD is likely to be complex, and not fully understood. Additionally, SRIs have been shown to be efficacious in treating BDD (see article by Phillips in this issue). This suggests possible involvement of the serotonin system in the disorder, but it does not prove that serotonergic abnormalities are part of BDD's underlying pathophysiology.

Neuroethology

Excessive grooming behaviors, for example, excessive face washing, plucking hairs, or skinpicking, are common in BDD⁴⁴. Pathological grooming conditions in animals have long been observed, and may provide analogous models for these behaviors in BDD⁴⁵.

Preliminary Neurobiological Model of the Pathophysiology of BDD

Attempts to integrate these findings result in a complex proposed model that implicates brain dysfunction and neurochemistry in the development of BDD. Right hemisphere dysfunction may be involved in the development of BDD, given the right dominance related to body image⁴⁶, as well as suggestions from case reports that BDD may be associated with right temporal lobe lesions^{47, 48}. Thus, one hypothesis is that right hemisphere dysfunction could put an individual at risk for developing BDD, which could be exacerbated by dysfunction in frontal-striatal circuits, contributing to BDD-related obsessive thoughts and compulsions. Dysfunction in specific areas of the brain such as visual cortical systems could

lead to distorted perception of body and face, and to an inability to correct this distortion or inhibit fronto-striatal responses to aversive stimuli related to the body. Possible dysfunction in the frontal-occipital-temporal circuit (i.e., top-down modulation) could lead to an imbalance in detail vs. holistic visual processing. Finally, serotonin may modulate BDD symptoms in frontal-subcortical circuitry and/or the limbic system, although little is known about its involvement in BDD.

CONCLUSIONS

The contribution of these different putative etiological and pathophysiologic factors to BDD's development and maintenance, and others yet to be identified, remains unclear. A preliminary, hypothetical model involves a complex interplay in which genetic and biological susceptibilities interact with environmental events, such as teasing or abuse. These more "distal" genetic and environmental causative factors in turn may lead to more proximal contributing factors, such as neurobiologically based imbalances in global vs. local visual processing that may contribute to perceptual distortions and other information processing biases, as well as fronto-striatal abnormalities that may be associated with BDDrelated obsessive-thoughts and repetitive behaviors. In individuals with such susceptibilities, adverse events such as abuse and teasing may be more likely to have influential effects. The subsequent development of distorted cognitions and reinforcing repetitive and avoidance behaviors may then serve to maintain the symptoms of the disorder. However, many questions remain; just one example is whether the above-noted brain abnormalities are inherited phenotypes or develop as a result of the disorder. Further development and testing of submodels and integrated etiological models for BDD will be important to aid in early identification and intervention, and will be crucial to guide the development of improved treatments.

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