The Relationship of Body Dysmorphic Disorder and Eating Disorders to Obsessive-Compulsive Disorder

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Abstract

Body dysmorphic disorder (BDD) and eating disorders are body image disorders that have long been hypothesized to be related to obsessive-compulsive disorder (OCD). Available data suggest that BDD and eating disorders are often comorbid with OCD. Data from a variety of domains suggest that both BDD and eating disorders have many similarities with OCD and seem related to OCD. However, these disorders also differ from OCD in some ways. Additional research is needed on the relationship of BDD and eating disorders to OCD, including studies that directly compare them to OCD in a variety of domains, including phenomenology, family history, neurobiology, and etiology.

Introduction

Body dysmorphic disorder (BDD) and eating disorders have long been hypothesized to be related to obsessive-compulsive disorder (OCD). More recently, since the advent of the obsessive-compulsive spectrum disorders (OCSDs) concept, they have been considered candidates for inclusion in this grouping of disorders.\(^1\)\(^-\)\(^8\) Their relationship to OCD has been examined empirically and discussed in the literature, and is the focus of this review. It should be noted, however, that BDD has also been hypothesized to be related to other disorders, such as social phobia and major depressive disorder, and eating disorders have been hypothesized to be related to major depression, anxiety disorders, or substance abuse.\(^7\)\(^,\)\(^9\)\(^-\)\(^13\) These possible relationships, however, have received little empirical investigation. Furthermore, BDD and eating disorders themselves seem to have some shared features, such as disturbance in body image. However, their relationship has received virtually no empirical attention and is not discussed in this article. The relationships among all of these disorders have important theoretical and clinical implications and are ripe for empirical study. It is possible that a number of these disorders (eg, BDD, OCD, and eating disorders) may share some genetic and environmental risk factors as well as pathophysiologic mechanisms.

Body Dysmorphic Disorder

Body dysmorphic disorder (BDD) has been considered closely related to OCD for more than a century, and it is widely conceptualized as an OCSD.\(^1\)\(^-\)\(^17\)\(^-\)\(^14\) BDD’s similarities with social phobia have also been noted; the Japanese conceptualization of *taijin kyofusho*, which is similar to social phobia, includes BDD (*shubo-kyofu*, or “the phobia of a deformed body”).\(^9\)\(^,\)\(^10\) During the development of the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, consideration was given to classifying BDD as an anxiety disorder, but this change was not made because there were insufficient data.\(^15\) Since then, research on BDD has substantially increased, which includes studies that have directly compared BDD and OCD.

BDD, classified as a somatoform disorder, is defined as a preoccupation with an imagined defect in appearance (if a slight physical anomaly is present, the person’s concern is markedly excessive). The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning, and is not better accounted for by another mental disorder (eg, dissatisfaction with body shape and size in anorexia nervosa [AN]).

Characteristics of Body Dysmorphic Disorder and its Relationship to Obsessive-Compulsive Disorder

Phenomenology of Obsessions and Compulsions

Obsessions

BDD preoccupations have many similarities to OCD obsessions.\(^7\) They are intrusive, persistent, repetitive, unwanted thoughts that are recognized as one’s own.\(^13\)\(^,\)\(^16\)\(^-\)\(^18\) Patients usually recognize that the thoughts are excessive, in the sense of realizing that they spend too much time worrying about their appearance. The preoccupations cause significant anxiety and distress, are impairing, and are usually resisted to at least some extent.\(^7\)\(^,\)\(^19\) In one study (n=53 subjects with BDD, 53 subjects with OCD),\(^15\) BDD and OCD did not significantly differ on any individual obsession item on the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS). Sometimes,
BDD and OCD obsessions have similar content—focusing, for example, on symmetry, “just right” concerns, or a desire for perfection.

BDD obsessions differ from OCD obsessions, however, in their focus on physical appearance. Another difference consistently found in BDD-OCD comparison studies is that BDD obsessions are characterized by poorer insight (greater delusionality) than OCD obsessions. Approximately 2% of OCD patients are currently delusional compared with 27% to 39% of BDD patients. Regarding specific components of delusionality, BDD patients are more convinced than those with OCD that their underlying belief (eg, “I am ugly and deformed”) is accurate, more likely to think others agree with their belief, less willing to consider that their belief is inaccurate, and less likely to recognize that their belief has a psychiatric/psychological cause. Thus, OCD criterion C (“the person recognizes that the fear is excessive or unreasonable”) often is not characteristic of BDD, if “unreasonable” is interpreted as having good insight.

Another distinction is that core beliefs underlying the obsessions seem to often differ. While BDD and OCD have not been directly compared in this regard, core beliefs in BDD seem to focus more on unacceptable of the self (eg, feeling worthless [60%], inadequate [71%], or unlovable [41%], or the fear of ending up isolated and alone [69%]). Unlike OCD, BDD beliefs rarely seem to involve moral repugnance.

**Compulsions**

Nearly all patients with BDD perform at least one compulsive behavior, such as comparing with others, camouflaging disliked body areas, mirror checking, reassurance seeking, excessive grooming, skin picking, tanning, excessive exercising, touching disliked body areas, clothes changing, seeking dermatologic treatment or surgery, and compulsive shopping (eg, for beauty products). These behaviors resemble OCD compulsions in that (1) the behaviors are performed intentionally, in response to an obsession; (2) the intent is to reduce anxiety or distress and prevent an unwanted event (eg, being rejected by others or looking “ugly”); (3) most behaviors are repetitive, time-consuming, and excessive; (4) they may be rule bound or done in a rigid manner; and (5) carrying out the act is not pleasurable. Some OCD and BDD compulsions (eg, checking and reassurance seeking) overlap in content/phenomenology, although their specific focus differs, with a focus on perceived appearance flaws in BDD. In one study, BDD and OCD did not significantly differ on any individual Y-BOCS compulsion item.

However, BDD and OCD compulsions differ in their specific content/phenomenology. In addition, some BDD compulsions (eg, mirror checking) do not seem to follow a simple model of anxiety reduction that occurs in the compulsive checking of OCD. Investigation of the functional relationship between anxiety-evoking thoughts (obsessions) and strategies to reduce anxiety (compulsions) is needed to assist in determining the relatedness of BDD and OCD.

**Associated Features**

**Anxiety and Depressive Symptoms**

One BDD-OCD comparison study found higher state anxiety in BDD, whereas McKay and colleagues found higher state anxiety in OCD on one of two measures. In one study, but not the other, BDD subjects had more severe depressive symptoms than OCD subjects. In a third study, depressive symptoms were more severe for comorbid BDD+OCD subjects than for those with OCD only or BDD only, which was accounted for by more severe BDD symptoms in the comorbid group.

**Suicidality**

In two of three BDD-OCD comparison studies, a higher proportion of BDD subjects reported suicidal ideation. A study that examined lifetime suicide attempts attributed primarily to BDD or OCD found a higher rate among BDD subjects. Another study found a higher lifetime rate of suicide attempts (40%) among subjects with comorbid BDD+OCD than in those with only BDD or only OCD; this appeared accounted for by more severe BDD symptoms in the comorbid group.

**Functioning and Quality of Life**

Functioning and quality of life are similarly very poor in both disorders. One of three BDD-OCD comparison studies found that BDD subjects were more likely to be unemployed and had lower educational attainment. Phillips and colleagues found that those with BDD had missed more days of work or school due to their illness.

**Comorbidity**

Three studies that used the Structured Clinical Interview for DSM-III-R or DSM-IV Axis I disorders found that 32% to 38% of patients ascertained for BDD had lifetime comorbid OCD. Studies that examined comorbidity in patients ascertained for OCD yielded a broad range (3% to 37%) with comorbid BDD. Three BDD-OCD comparison studies found no significant differences in lifetime comorbidity for bipolar disorder, psychotic disorders (excluding delusional BDD or delusional OCD), panic disorder, agoraphobia, specific phobia, posttraumatic stress disorder, somatoform disorders, tic disorder, trichotillomania, or eating disorders. One of these studies examined personality disorders, finding no significant difference for 10 of 11 personality disorders. However, these studies did find that individuals with BDD were more likely to have paranoid personality disorder (in one study) as well as lifetime major depression (in two of three studies), dysthymia (in one of two studies), social phobia (in one of three studies), and substance use disorders (in two of three studies). One study found that OCD patients more often had comorbid generalized anxiety disorder. Taken together, these studies suggest that BDD is more often associated with
comorbidity than is OCD, although these findings are potentially subject to both type I error (because many comparisons were made) and type II error (because some sample sizes were relatively small).

Course of Illness

Studies indicate that mean age of onset of BDD is 16–17 years of age. Two BDD-OCD comparison studies found no significant difference in age at onset, although one study found an earlier age at onset for BDD. The one study that compared onset of subclinical illness found no significant difference between BDD and OCD.

A cross-sectional/retrospective study found that BDD and OCD have a similarly chronic course. In the only prospective study of BDD’s course, over 1 year BDD was chronic in 91% of 161 subjects, consistent with studies suggesting that OCD is often chronic. This prospective naturalistic study also examined longitudinal time-varying associations between BDD and comorbid OCD in 161 subjects. Improvement in comorbid OCD predicted subsequent BDD remission, but improvement in BDD did not predict subsequent remission of comorbid OCD. These mixed findings suggest that BDD and OCD symptoms may be etiologically linked for some subjects. However, full-criteria BDD persisted in about 50% of subjects after their OCD remitted, suggesting that BDD is not simply a symptom of OCD. A stronger longitudinal association was found for BDD and major depression. However, this study was able to detect only larger effects, and relatively few BDD or OCD remissions occurred, which may result in some numerical instability of the results.

Demographic Features

In a nationwide survey in Germany (N=2,552), BDD was somewhat more frequent in women (1.9%) than men (1.4%). An epidemiologic study in Italy (N=673) found that BDD was more common in females (1.4% of women vs 0% of men), whereas a study in the United States (N=373) found that BDD was slightly more common in males (1.2% vs 1.0%). Some non-epidemiologic studies have contained more females than males, others more males than females, and one an approximately equal proportion of females and males. In three BDD-OCD comparison studies, the gender ratio did not significantly differ.

In two of three studies, BDD subjects were significantly younger and less likely to be married than OCD subjects. In one study, BDD subjects had lower educational attainment. BDD patients have also been found more likely than those with OCD to have an occupation or education in art and design (20% vs 3%), raising the possibility that an interest in aesthetics may contribute to BDD’s development.

Family History

A controlled and blinded family study (80 case probands, 73 control probands) found that BDD occurred significantly more frequently in first-degree relatives of OCD probands than control probands, suggesting that BDD may be a member of the familial OCSDs. In the one BDD-OCD comparison study that examined family history, rates of mood, psychotic, anxiety, and eating disorders were similar in first-degree relatives of BDD and OCD probands. However, first-degree relatives of BDD probands were more likely to have a substance use disorder, which did not appear accounted for by more frequent substance use disorders in BDD probands than in OCD probands.

Genetic Factors and Brain Circuitry

Data on genetic factors and brain circuitry in BDD are limited, with some results but not others supporting a relationship between BDD and OCD. In a preliminary candidate gene study of 57 BDD subjects and 58 healthy controls matched for ethnicity and gender, association was demonstrated for GABAA-γ2 (Sq31.1-q33.2) (P=.032), with the 1(A) allele occurring more frequently in BDD subjects than controls. No association was demonstrated for serotonin (5-HT) receptor, which has been associated with OCD. Neither was an association demonstrated for other tested candidate genes (5-HTTLPR, 5-HT1A receptor, tryptophan hydroxylase, the VNTR polymorphism for the serotonin transporter, dopamine DRD4 or DRD5 receptor genes, or dopamine transporter).

In a small, preliminary morphometric magnetic resonance imaging study in eight women with BDD versus eight healthy controls, the caudate nucleus differed in subjects and controls, consistent with conceptualization of BDD as an OCSD. However, BDD subjects had a relative leftward shift in caudate nucleus asymmetry, whereas magnetic resonance imaging studies of OCD implicating lateralized abnormalities have suggested a rightward shift in striatal asymmetry. Although some previous OCD studies have shown reduced white matter volume, BDD subjects had greater total white matter volume. A small uncontrolled BDD single photon emission computed tomography study (N=6) yielded a broad range of discrepant findings that did not support a close relationship between BDD and OCD. One small, preliminary study found that BDD occurred more frequently in patients with rheumatic fever (two out of 59) than in controls (zero out of 39), raising the possibility that BDD and OCD may share some pathophysiologic mechanisms involving immune function. In a neuropsychological study (N=35), BDD subjects had impaired verbal and nonverbal memory compared with healthy controls. This impairment seemed mediated by deficits in organizational encoding strategies, implicating corticostratal systems. These results are similar to those found for OCD by Deckersbach and colleagues. Another BDD study found impaired executive functioning.

In an information-processing study, BDD patients were more likely than healthy controls to interpret a range of ambiguous situations (appearance-related, social, and general) as threatening, whereas OCD patients exhibited this negative interpretive bias.
only in ambiguous general situations. In another study, BDD patients did not differ from OCD patients or controls in terms of general facial-feature discrimination. BDD patients were less accurate than the control group, but not the OCD group, in identifying facial expressions of emotion; this included misinterpreting faces as angry. Buhlmann and colleagues similarly found that BDD patients, relative to controls, were less accurate in identifying facial emotional expressions in self-referent scenarios, misinterpreting more neutral expressions as contemptuous and angry (this study did not include OCD patients). These findings are interesting, given that a majority of BDD patients have ideas or delusions of reference (e.g., mistakenly believing that other people are mocking them due to their appearance).

Treatment

Pharmacologic Dissection/Pharmacotherapy

The treatment of BDD—both pharmacotherapy and cognitive-behavioral therapy—is described in more detail elsewhere, including in a guideline from the United Kingdom’s National Institute of Clinical Excellence on the treatment of OCD and BDD. In brief, all selective serotonin reuptake inhibitor (SSRI) studies to date (two controlled studies, four open-label trials, and a clinical series) indicate that these medications are often efficacious for BDD. Furthermore, in a controlled and blinded crossover study, the SSRI clomipramine was more efficacious than the non-SSRI antidepressant desipramine, as in OCD. This latter finding is supported by retrospective data suggesting that SSRIs appear more efficacious for BDD than non-SSRIs. While somewhat non-specific, SSRI response in BDD (similar to OCD) is usually slow and gradual, appearing over months, and relatively high SSRI doses are often required (although dose-finding studies have not been done). Unlike OCD, however, in a small double-blind randomized trial, pimozide was not more efficacious than placebo as an SSRI augmentation agent.

Cognitive-Behavioral Therapy

Like OCD, exposure and response prevention appears efficacious for BDD. However, reports of this technique—without concomitant use of cognitive approaches—are limited to a retrospective study and small case series with up to 10 subjects. Most studies (two waiting-list controlled studies and case series) have used both cognitive and behavioral techniques. Cognitive-behavioral therapy strategies for BDD are focused specifically on appearance-related thoughts and behaviors (e.g., appearance concerns and body image, muscle dysmorphia symptoms, pursuit of surgery and dermatologic treatment). Clinical experience suggests that, unlike OCD, habit reversal is frequently needed when treating patients with BDD, especially for common, core BDD symptoms, such as skin picking. Clinical experience also suggests that BDD patients’ greater delusionality may make them more difficult to engage in treatment and effectively treat. Unanswered questions requiring study are whether the greater delusionality in BDD requires inclusion of a cognitive component to explicitly target poor insight and greater focus on motivation and engagement in therapy.

Summary

More research is needed on BDD and OCD, especially direct comparison studies of the two disorders in all of the above domains. Studies are also needed which compare BDD with other disorders. Research on these disorders' etiology and pathophysiology may be particularly informative. Nonetheless, available data from a variety of domains indicate that BDD and OCD have many similarities but also some differences, suggesting that they are not identical disorders but are probably related. Data on phenomenology, comorbidity, the apparent selective response of BDD to SSRIs, and the aforementioned family study offer particularly strong support for the conceptualization of BDD as an OCSD.

Body Dysmorphic Disorder’s Diagnostic Criteria: Some Issues for the DSM-V

Several aspects of BDD’s diagnostic criteria that need to be considered are:

- “Preoccupation” needs to be better operationalized;
- The word “imagined” is problematic and may lead to underdiagnosis of BDD, given that most patients have poor or absent insight and do not realize that their view of their appearance is imagined or distorted;
- A requirement for clinical significance is needed to differentiate BDD from normal appearance concerns. However, it is unclear how to best operationalize this construct. This issue also pertains to other DSM-IV disorders;
- Careful consideration must be given to insight (delusionality), including its implications for BDD diagnostic criteria and the classification of delusional and non-delusional variants of BDD. This issue, too, pertains to other disorders in the DSM.

Delusionality: A Dimensional Construct?

Whether delusionality is a dimensional construct or both a dimensional and categorical construct is an important classification issue with clinical implications. A related issue is the relationship between delusional and non-delusional variants of disorders. The DSM-IV classifies delusional BDD as a psychotic disorder (delusional disorder, somatic type). Delusional patients may be diagnosed with both delusional disorder and BDD (i.e., double coding is allowed). OCD and hypochondriasis also have delusional variants that the DSM-IV classifies as psychotic disorders. In addition, OCD has a poor-insight specifier. OCD patients with and without insight seem generally similar in terms of demographic and clinical characteristics, and studies comparing delusional and non-delusional variants of BDD suggest that they have many more similarities than differences. Findings such as these raise the question of whether delusional and non-delusional variants of some disorders constitute the same disorder and whether delusionality is a
dimensional construct. If so, how should delusionality be incorporated into DSM-V, and how should delusional and nondelusional variants of disorders optimally be classified? Research is needed on the construct of delusionality and the delusional and nondelusional variants of disorders such as BDD, OCD, hypochondriasis, major depression, and eating disorders.75

Anorexia Nervosa and Bulimia Nervosa

For at least 50 years, AN has been linked to OCD.79 Earlier reviews of major comorbid symptoms80 showed that OCD symptoms in AN were the second most frequently reported after depression. Solyom and colleagues81 found that, even after excluding food- and body-related obsessions, individuals with AN had high trait scores on inventories assessing OCD symptoms that were comparable with scores of patients diagnosed with OCD.

Despite this association between eating disorders and OCD, relatively few studies have directly compared the phenomenology or neurobiology of these disorders. One factor confounding direct comparisons is that AN and bulimia nervosa (BN), a related disorder, are often associated with malnutrition and extremes of eating behaviors, which have substantial effects on behavior and physiology. Thus, the question is often raised as to whether behavioral symptoms are merely state-related. In response to this concern, studies82-85 show that obsessive and anxious symptoms occur premorbidly and persist after recovery but are exaggerated by malnutrition. In addition, recent studies86 showed that the majority of individuals with AN and BN exhibit childhood perfectionism and obsessive-compulsive personality patterns, and these symptoms predate the onset of AN and BN. Therefore, it can be argued that these are traits that create a vulnerability for developing an eating disorder.

Characteristics of Anorexia Nervosa and Bulimia Nervosa and Their Relationship to Obsessive-Compulsive Disorder

AN and BN tend to present in adolescence and occur more frequently in females.87 They affect an estimated 0.5% and 1.5% to 3%, respectively, of females in the general population. These disorders are characterized by restricted and/or binge eating, a relentless pursuit of thinness, and obsessive fears of becoming fat. Although these disorders are often thought to be caused by psychosocial factors, recent studies88-90 show substantial genetic and neurobiological etiological influences. These are often chronic, disabling conditions.

AN is divided into two subtypes: restricting and bulimic. In the restricting subtype, subnormal body weight and an ongoing malnourished state are maintained by unremitting food avoidance. In the bulimic subtype of AN, there is comparable weight loss and malnutrition, yet the course of illness is marked by supervening episodes of binge eating and/or some type of compensatory action, such as self-induced vomiting or laxative abuse. Individuals with BN remain at normal body weight, although many aspire to ideal weights far below the range of normalcy for their age, gender, and height. The core features of BN include repeated episodes of binge eating followed by compensatory self-induced vomiting, laxative abuse, or pathologically extreme exercise or fasting, and excessive concern with weight and shape. Although abnormally low body weight is an exclusion for the diagnosis of BN, some 25% to 30% of bulimia patients have a prior history of AN. In addition, some individuals with AN convert to BN. Despite differences in the topography of their feeding behavior, these disorders share equivalent concerns with weight and shape as well as low self-esteem, depression, anxiety, obsessionality, and perfectionism.

Prevalence and Phenomenology of Obsessions and Compulsions in Anorexia Nervosa and Bulimia Nervosa

Recently, Godart and colleagues84 reported that the lifetime prevalence of OCD ranged from 9.5% to 62% in restricting-type AN (RAN), from 10% to 66% in bulimic-type AN (BAN), and from 0% to 42.9% in BN individuals. These studies have tended to use relatively small samples and methods have varied in terms of rigor. A recent study from the Price Foundation Genetic collaboration85 used trained raters and a combination of the Structured Clinical Interview for DSM-IV Axis I Disorders and the Y-BOCS to establish OCD diagnoses in 672 individuals with an eating disorder. The overall rate of OCD was 41%, with no difference in frequency between RAN, BAN, and BN.

Several studies have assessed the frequency of specific types of obsessions and compulsions in AN and OCD patients using checklist categories from the Y-BOCS. Two studies found symmetry obsessions to be the most common obsessions in AN patients, occurring in 68.8% of AN patients in one study91 and in 72% of AN patients in the other study.92 These studies found similar rates of ordering and arranging compulsions. The frequency of other types of obsessions and compulsions among the AN patients differed in prevalence between these two studies. A third study93 found that AN subgroups were similar to OCD patients in terms of the frequency of obsessions in the symmetry and somatic categories or in the compulsion categories of ordering and hoarding. In all other categories, the AN subgroups had a significantly lower frequency compared with the OCD patients.

There are several aspects of OCD symptoms in eating disorder patients that should be highlighted. The obsessions and compulsions of AN patients are largely ego-syntonic despite interfering significantly in their lives. AN subjects feel compelled to perform these rituals and behaviors, even though these behaviors may lead to anxiety. However, they are not regarded as unwanted. In addition,94 phobic thoughts of food and weight repeatedly enter the mind of AN patients, but not necessarily against their will. Although these thoughts or preoccupations may be distressing, they are not regarded as senseless. Similarly, such ego-syntonic acceptance of symmetry and exactness symptoms, unrelated to eating, also occur in AN patients.91
There are other reasons why AN and BN are not often considered to be part of the OCSDs. AN and BN are relatively female-gender specific, and tend to have a narrow range of onset near in time to puberty. AN and BN involve severe and intense body-image distortion, in which emaciated individuals perceive themselves as fat, as well as stereotypic hyperactive motor behavior. Finally, there is often denial of emaciation or illness, lack of insight, and resistance to treatment.

**Obsessive-Compulsive Personality Disorder Symptoms in Anorexia Nervosa and Bulimia Nervosa**

It has long been recognized that AN and BN individuals commonly have personality disorders. A review\(^8\) of assessments done by raters found that cluster C disorders are common in RAN and cluster B and C disorders are common in BN. Rates of obsessive-compulsive personality disorder (OCPD) were between 2% and 30% for RAN and 3% and 19% for BN. A family study\(^1\) that used blind raters and a control sample found that 46% of RAN had OCPD compared with 4% of BN and 5% of control women. Halmi and colleagues\(^9\) found that the frequencies of OCD, OCPD, and OCD/OCPD in a large eating disorder sample were 20%, 13%, and 16%, respectively, with no difference among eating disorder subtypes. Thus, the frequency of OCD and OCPD diagnosis tends to be similar within eating disorder subgroups.

**Onset of Obsessional Symptoms in Anorexia Nervosa and Bulimia Nervosa**

Series of studies\(^1\) have consistently found that anxiety disorders in AN and BN most commonly appear in childhood, before the onset of AN or BN. Of the 41% of AN and BN individuals who had a lifetime diagnosis of OCD,\(^5\) 61% experience the onset of OCD in childhood, prior to the onset of an eating disorder. The mean age of onset of OCD was 14 years and the mean age of onset of AN or BN was 17 years. When the entire sample of AN and BN subjects were considered, 23% reported the onset of OCD before the onset of AN or BN. In addition, individuals also commonly reported\(^5\) the onset of social phobia, specific phobia, and generalized anxiety disorder in childhood before they developed an eating disorder. People with a history of an eating disorder who were not currently ill and had never been diagnosed with OCD or anxiety disorder tended to be anxious, perfectionistic, and harm avoidant. The presence of a lifetime anxiety disorder or current AN or BN tended to exacerbate anxious, perfectionistic, and harm avoidant symptoms.

**Anorexia Nervosa and Bulimia Nervosa in Individuals with Obsessive-Compulsive Disorder**

Studies, using a variety of methods, have found increased rates of eating disorder in individuals with OCD. Tamburrino and colleagues\(^1\) found that 42% of 31 women with OCD had a past or current history of an eating disorder (RAN: 26%, BN: 3%, BAN: 13%). Fahy and colleagues\(^8\) found that 11% of 105 women with OCD previously had AN. Rubenstein and colleagues\(^9\) found that of 62 OCD patients, 13% met full criteria for AN or BN and another 18% met subthreshold criteria for an eating disorder. Another study,\(^10\) which administered the Eating Disorder Inventory, found that scores of female patients with OCD were midway between those of individuals with an eating disorder and healthy comparison women.

**Family History Studies**

Several studies have found that relatives of individuals with an eating disorder have increased rates of OCD. Lilenfeld and colleagues\(^1\) found a 10% rate of OCD in first-degree relatives of RAN, which was significantly higher than the 3% rate of OCD in relatives of controls. Relatives of BN had a 7% rate of OCD, which was not significantly different than relatives of controls. Bellodi and colleagues\(^1\) found OCD in 16% of relatives of RAN, 15% of relatives of BAN, and 10% of relatives of BN. Together these were significantly higher than the 1.4% rate of OCD in relatives of controls. In comparison, Bienvenu and colleagues\(^1\) found no difference in the rates of AN and BN in first-degree relatives of OCD patients compared to first-degree relatives of controls.

Lilenfeld and colleagues\(^1\) reported that the first-degree relatives of RAN had a 19% frequency of OCPD, which was significantly higher than the 7% rate in BN relatives and 6% rate in relatives of control women. Rates of OCPD among relatives of AN probands with and without OCPD were virtually identical, suggesting shared familial transmission of AN and OCPD. These findings raise the possibility that OCPD and AN represent a continuum of phenotypic expressions of a similar genotype. Alternatively, RAN may occur only in the presence of risk factors for both an eating disorder and OCPD.

While studies are too few to draw definitive conclusions, these findings raise the possibility of some shared transmission between eating disorder and OCD and/or OCPD. OCD is thought\(^1\) to be a heterogeneous condition. Perhaps AN and BN share transmission with the cluster of OCD patients with symmetry and exactness symptoms, or with some dimension of OCPD.

**Neurobiology and Genetics**

Considerable physiological studies have shown that individuals with AN and BN have disturbances of serotonin activity.\(^5\) These disturbances occur when individuals are ill, and persist after recovery. A disturbance of 5-HT function has also been implicated in OCD.\(^1\) Whether people with an eating disorder and OCD share identical disturbances of 5-HT function is less clear. As noted above, malnutrition and pathological eating influence 5-HT function, making direct comparisons problematic. One potential means of answering such questions is to assess 5-HT genes. In fact, there are indications that individuals with eating disorder and OCD may have alterations in the 5-HT\(_{2A}\) receptor and 5-HT transporter genes.\(^1\) However, these studies tend to be small, with many inconsistent findings. Confirmation of such findings will need to rely on much larger samples and contemporary assessment of haplotypes as well as an understanding of the many genes that contribute to 5-HT and related neuronal circuit function.
Brain Imaging

Considerable studies have used brain imaging to study OCD. A review by Saxena\textsuperscript{104} reports that studies of OCD patients have consistently found altered activity in the orbitofrontal cortex, with less consistent abnormalities in the caudate nuclei, thalamus, and anterior cingulate, which also show neurochemical alterations suggestive of neuronal dysfunction. Many fewer brain imaging studies have been done in AN and BN,\textsuperscript{106} and no studies to our knowledge have directly compared eating disorders with OCD. It is well known that malnourishment in both AN and BN is associated with shrinkage of brain tissue as well as metabolic alterations. Thus, comparing studies in ill, symptomatic eating disorder subjects to those of OCD subjects may be problematic, because it is not certain how brain imaging findings in eating disorders are influenced by malnutrition. Another approach is to study AN and BN subjects after recovery, when there is normalization of eating, nutrition, and weight. Many temperament characteristics, including obsessionality, persist, in the recovered state, but there is normalization of brain mass.\textsuperscript{108} Thus, any persisting alterations are likely to be traits, but they may also reflect a “scar” caused by chronic malnutrition. Still, brain imaging studies in AN and BN subjects\textsuperscript{107} have tended to find alterations in limbic regions. These findings are consistent with dysfunction of affect regulation, executive function, and impulse control. However, the question of whether eating disorder and OCD involve similar regions/circuits remains uncertain and will likely require direct comparisons of matched eating disorder and OCD subjects. It should also be noted that several studies have found relationships between body image distortion and changes in the left parietal lobe in AN.\textsuperscript{107} It has long been recognized that the parietal cortex mediates perceptions of the body. In summary, neuroimaging data suggest some shared brain circuitry between AN/BN and OCD, but state and methodological differences confound comparisons.

Treatment

Several consensus publications have recently reviewed treatment options for AN and BN. These include the American Psychiatric Association guidelines\textsuperscript{109} and the National Institute of Clinical Excellence guidelines.\textsuperscript{110} These publications should be consulted for more specific details about therapy. In addition, websites such as the National Eating Disorders Association (www.nationaleatingdisorders.org) can be accessed for more information and treatment referrals.

In brief, controlled trials have provided convincing evidence for the efficacy of both psychological and antidepressant treatments for BN. Still, while most subjects show a decrease in the frequency of binge eating and purging episodes, relatively few develop complete abstinence. In contrast, relatively few controlled trials have been done in AN. Individuals with AN are often resistant to treatment, as they tend to have ego-syntonic symptoms or may deny being underweight or in need of treatment. Few controlled trials of any therapy have been performed, in part because it has been difficult to enlist cooperation of individuals with AN, and in part because psychological and pharmacologic strategies that have been successful in other disorders appear to be less effective in this illness. For severely emaciated patients, hospitalization for supportive medical care and weight restoration may be useful or necessary. Still, relapse is common after discharge. More recent controlled trials\textsuperscript{111} have suggested that the Maudsley Family therapy techniques seem to be particularly helpful in younger patients with AN. Follow-up studies\textsuperscript{111} suggest that these interventions may also help prevent relapse in patients who achieve weight restoration. Recent studies\textsuperscript{112} have begun more systematic evaluation of the potential benefits of manualized cognitive-behavioral and family therapies for this disorder. A range of medications, including SSRIs and neuroleptics, have been tried in AN, but there have been few controlled trials in ill, malnourished patients. Some data\textsuperscript{113,114} raised the possibility that fluoxetine may be useful in reducing relapse after weight restoration, particularly in those with RAN compared with BAN.

Summary

There is compelling evidence that AN/BN and OCD are related disorders. Individuals with AN and BN often have comorbid OCD, and eating disorders occur in some subjects who present with OCD. Moreover, OCD symptoms first present in childhood in about a quarter of individuals who later develop AN or BN. Finally, a diagnosis of OCD occurs in family members of people with AN and BN. There are clear differences between the disorders, however. AN and BN are gender specific and accompanied by eating and body-image symptoms. AN in particular is associated with OCPD, perfectionism, and inflexible, ascetic personalities, and these symptoms are common in family members. And AN individuals, particularly in the ill state, have ego-syntonic symptoms, which are resistant to treatment, and lack insight. It has been proposed\textsuperscript{103} that OCD is a heterogeneous condition. Perhaps AN and BN have more in common with OCD patients with symmetry and exactness dimensions or with OCD patients with OCPD and/or overvalued ideation. These questions are unlikely to be answered until direct comparisons of phenomenology, genetics, and brain imaging are conducted in eating disorder and OCD patients.

Some Issues for the DSM-V

The DSM-V should consider including as part of the diagnostic criteria or as an associated descriptive feature a description of common premorbid vulnerabilities, such as obsessionality, anxiety, harm avoidance, and perfectionism. These traits also often persist after people recover from AN and BN. More recently it has become clear that certain neurocognitive patterns occur in AN which persist after recovery, such as delayed set-shifting. Together these appear to be traits that contribute toward a vulnerability of developing AN and BN.

While many malnourished individuals with AN have normal laboratory exams, there is considerable evidence that they have abnormal brain neurotransmitter function, which contributes to symptoms and perhaps resistance to treatment. While the optimal way to reflect this in the DSM-V is unclear, information on neurobiology as well as genetic contributions to the development of
eating disorders should be included.

AN is associated with a cluster of common symptoms, such as overexercise, denial, and resistance to treatment. These clinical features should be included in DSM-V, as they are key elements that make it difficult to treat AN.

Conclusion

Views of the relationship between OCD, BDD, and ED have tended to be based largely on observations of phenomenology. While such observations are of critical importance, it is necessary to also increase understanding of these disorders' etiology and pathophysiology, including how behavior is coded in the brain. It has been difficult to correlate brain function and behavior in living human beings because our technology has been indirect and relatively imprecise. Now, a revolution is taking place in understanding the brain and behavior. Considerable basic science studies have produced a wealth of information about the function of brain pathways and neurotransmitters. Moreover, substantial advances have occurred in technologies, such as brain imaging and genetics, that make it possible to apply this new knowledge to studies in living people. Increased understanding of environmental determinants of illness, and their effects on the brain, is also occurring. The shared symptoms of BDD, ED, and OCD suggest that they may involve the same brain pathways. However, each may be a syndrome consisting of one, or likely many, different patterns of molecular disturbances. A definitive answer to questions about the relationship between these syndromes may not be possible until we have the tools necessary to understand the genetic and environmental bases of these disorders and to better define brain pathway function and behavior.

References


