Eating Disorders and Depression

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Clinicians have long been intrigued by the idea that eating disorders may be related in some way to depression. Indeed, eating abnormalities, both undereating and overeating, are cardinal symptoms of melancholia and of atypical depression respectively. In addition, clinicians have noted the frequent occurrence of other depressive symptoms in patients with eating disorders. These observations have served as the basis for clinical trials of antidepressant medications in eating disorders, which, in the case of bulimia nervosa, have proven remarkably successful. Yet, if there is a relationship between eating disorders and affective disorders, its exact nature remains obscure.

Some conceptual models for the relationship between eating disorders and depression are depicted in the Figure. One point of view attributes depressive symptoms to the biological or psychological effects of abnormal eating and weight loss. For example, many common depressive features, including depressed mood, irritability, low energy, and low concentration, have been reported in studies of the physical and psychological effects of semistarvation.

Another point of view maintains that in many cases eating disorders are secondary to underlying affective disturbances. The extreme version of this argument contends that eating disorders represent a variant or masked form of depression, a contention that has aroused a great deal of controversy in recent years. Still another view argues that certain individuals may be at risk for both affective and eating disorders—perhaps because of shared psychological

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vulnerabilities, genetic factors, or biological mechanisms. Several recent reviews have summarized in detail the literature pertaining to eating disorders and depression. In this article, we highlight the major findings of:  
- studies on comorbidity of eating and depressive disorders at the time of presentation and at follow-up, 
- family studies of eating disorders and depression, and 
- studies of biological markers in eating disorders. We also discuss the relationship between eating disorders and depression, and the clinical implications of these findings.

**COMORBIDITY OF EATING DISORDERS AND DEPRESSION**

Although the occurrence of depressive symptoms in eating disorders only recently has been subjected to systematic study, clinical observations of depressive symptoms in these patients date at least as far back as the mid-1940s when Binswanger described symptoms of depressed mood, feelings of worthlessness, pervasive anxiety, isolation, and eventual suicide in a patient with features of both anorexia nervosa and bulimia nervosa. More recently, systematic studies have reported that roughly one third to one half of patients with eating disorders suffer from concurrent major depression, and the fraction rises to one half to three quarters when past depression is included. These rates of depression are found in patients with bulimia nervosa as well as in patients with anorexia nervosa.

Our own group has studied this issue in a group of 50 female patients meeting DSM-III criteria for bulimia nervosa who were given a semistructured interview to evaluate their current and lifetime frequencies of other psychiatric diagnoses, using Research Diagnostic Criteria. Our findings for depressive disorders are displayed in the Table. These frequencies, which are comparable to those reported by several other groups, provide striking confirmation of clinical intuition that many patients with eating disorders are depressed as well. In addition, we found that in about one quarter of patients with bulimia nervosa and a history of depression, the first depressive episode clearly preceded the onset of the eating disorder. This suggests that depression, at least in some cases, cannot be attributed entirely to abnormal eating.

Another means of sorting out depressive and eating-related symptoms in these patients is to gather follow-up data on recovered eating disordered patients. Over the short term, underweight patients with anorexia nervosa tend to show improvement in their depressive symptoms with weight gain and nutritional stabilization. However, the small number of long-term follow-up studies that have been performed suggest that a disproportionate number of patients show signs of depression at follow-up, at least in the case of anorexia nervosa.

One limitation of these findings is that patients who present for treatment may represent a sicker, or specifically a more depressed, subgroup of eating disordered patients as a whole. In order to address this issue, Hudson and colleagues simulated...
taneously studied their clinic population and a control group of patients with remitted bulimia nervosa who were not seeking treatment. They found comparable rates of depressive disorders in both groups, suggesting that increased rates of depression in bulimic patients are not restricted to clinic populations.

**FAMILY STUDIES**

Another means of better defining the relationship between the two disorders is to determine the degree to which they tend to appear in the same families. To the extent that biological relatives of patients with eating disorders show an increased frequency of depression compared to the population at large, one may speculate that genetic factors may exist that simultaneously increase the likelihood of developing depression and of developing an eating disorder.

Gershon and colleagues have reported that major affective disorder occurs at about three times the expected frequency in relatives of anorexic subjects. The risk of affective illness was increased even in relatives of probands without concurrent affective disorders. Interestingly, although concordance rates of anorexia nervosa are impressively high (about 45%) in monozygotic twins, rates of anorexia nervosa in dizygotic twins and other first-degree relatives of anorexic patients are only minimally elevated. Such a pattern can occur when multiple genetic factors are required to give rise to an illness—in this case anorexia nervosa—while the presence of only some of these genes is required for a second illness—in this case depression. Thus, the shared genetic risk model of depression and eating disorders finds some support in family data. However, in a study of first degree relatives of hospitalized patients with anorexia nervosa, Strober found increased rates of depression only in the families of those patients with concurrent major depression. They suggest, on the basis of these data, that the evidence for common genetic transmission of anorexia nervosa and affective illness is questionable.

Although less thoroughly studied than anorexia nervosa, bulimia nervosa also seems to be associated with an increased risk of depression in family members. Hudson et al. have reported that the risk for major affective disorder is 32% greater among first-degree relatives of bulimic patients than among relatives of nonpsychiatric control probands. Although this finding was most marked in bulimics who themselves had a history of depression, the relatives of nondepressed bulimic patients also showed an elevated risk for affective disorder compared with controls.

Thus, evidence from family studies provides tentative support for the idea of shared vulnerability to eating and affective disorders. In addition to being of theoretical interest in understanding the relationship between eating and affective disorders, information derived from comorbidity and family studies can have clinical utility in identifying patients who are at increased risk of developing these disorders.

**BIOLOGICAL MARKERS**

Identifying meaningful biological markers in eating disorders so far has yielded results that are, like candidate biological markers in other psychiatric disorders, tantalizing but difficult to interpret. For example, the dexamethasone suppression test (DST), widely studied as a potential biological marker for depression, has been found by several groups to show very high rates of nonsuppression or early escape in underweight patients with anorexia nervosa. However, because starvation alone can bring about similar changes in the hypothalamic-pituitary-adrenal (HPA) axis and because the rate of DST nonsuppression falls dramatically when anorexic patients gain weight, an abnormal DST does not necessarily link anorexia nervosa with depression.

One might expect that biological studies in normal-weight bulimic patients would be more readily interpretable because the confounding factor of weight is eliminated. Indeed, several studies have reported that about one third to one half of normal-weight bulimic patients show DST abnormalities. However, once again the situation is not that simple. A more recent study by our group of the DST in normal-weight bulimic patients has demonstrated that those bulimic patients with DST nonsuppression or early escape tend to have low plasma levels of dexamethasone, suggesting that the abnormality in these patients may lie not in the HPA axis but rather in the absorption or metabolism of dexamethasone.

Another biological abnormality that exists in both eating disorders and depression involves the thyroid stimulating hormone (TSH) response to thyrotropin releasing hormone (TRH). A blunted TSH response has been reported in approximately one quarter of depressed patients and in a variable proportion of anorexic and bulimic patients. However, both acute and chronic starvation can produce abnormalities in the hypothalamic-pituitary-thyroid (HPT) axis. Because abnormal eating patterns are by definition a part of the clinical picture of eating disorders and often a feature of depression as well, it would be premature to conclude that the presence of similar HPT abnormalities in eating disorders and depression implies that they are variant expressions of the same underlying illness. Fichter and associates have performed a valuable series of studies demonstrating the occurrence of HPA, HPT, and other abnormalities in short-term weight loss, and wisely have cautioned that future studies of biological markers in psychiatry must consider nutritional factors.

**TREATMENT OF EATING DISORDERS WITH ANTIDEPRESSANTS**

In the early 1980s, investigators launched preliminary studies of tricyclic antidepressants and monoamine oxidase inhibitors in bulimia nervosa. Several well-designed stud-
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ies have since confirmed that antidepressants of several classes produce a measurable improvement in binge/purge behavior in many patients, including those who are not clinically depressed. Initially, this might seem to suggest a similar underlying pathophysiologic abnormality in bulimia nervosa and depression. However, several diverse psychiatric disorders, including enuresis, panic disorder, posttraumatic stress disorder, obsessive compulsive disorder, and cocaine dependence, have been shown to respond to various antidepressants. The leap from similar treatment response to similar pathophysiology is one that must be taken with appropriate caution.

Interestingly, the small number of systematic studies of antidepressants in anorexia nervosa, unlike studies in bulimia nervosa, have failed for the most part to demonstrate a convincing response. Whether this is because of an underlying etiological dissimilarity between the two eating disorders or because of factors related to the effects of weight loss or of binge eating and purging is unknown as yet.

**DISCUSSION**

How does this information help us choose among models relating depression and eating disorders? The finding that bulimic patients, most of whom are of normal weight, and underweight anorexic patients have comparable rates of depression initially seems to disprove the idea that depression in these patients results from starvation. However, the situation is not quite so simple.

Recent studies have suggested that normal weight women with bulimia nervosa, possibly due to their abnormal eating patterns or frequent dieting, show several of the metabolic features commonly associated with starvation. Perhaps some of the psychological effects of starvation occur along with these physiological effects of starvation in normal weight women with disrupted eating patterns. Yet it seems unlikely that depressive symptoms in patients with eating disorders are entirely the result of nutritional factors.

Similarly, while family and comorbidity studies have suggested a strong relationship between eating and affective disorders, and biological marker studies have yielded some intriguing results, it is premature to conclude that these two types of illness are variant expressions of the same underlying disorder. The shared genetic vulnerability model remains plausible, but much more work remains to be done in affected and unaffected family members to better understand the nature of this vulnerability.

In the meantime, the current state of our knowledge about eating disorders and depression, although incomplete, nonetheless has clinical implications. First, treatment with antidepressants should be considered not only in eating disordered patients who are currently depressed, but also in bulimic patients who are not currently depressed.

Additionally, clinicians should be alert for symptoms of depression in patients with eating disorders, even after the eating disorder has resolved, and in their family members. Just as the initial phase of research into depression and eating disorders was motivated by clinical observation, the further refinement of our understanding of the relationship between the two disorders is enriched by carefully monitoring both depressive and eating-related symptoms as we treat these patients.

**REFERENCES**


