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## Eating Disorders and their Correlation to Mood Disorders

### Eating Disorder Etiologies

The underlying etiology of eating disorders has been postulated to be of a biological, psychological, and social or cultural nature. Most researchers and clinicians today would agree that the true etiology is multifactorial, thus encompassing all of these perspectives.

Specific biological theories include the possibilities that eating disorders are:

- 1) variants of mood disorders (esp. bulimia): frequent comorbidity of mood disorder, increased mood disorder in first degree relatives, response to antidepressants are supportive of this theory
- 2) based in genetic vulnerability: higher monozygotic than dizygotic twin concordance (50 to 14%) is supportive of this theory
- 3) analogous to substance abuse: dieting and exercising produces release of endogenous opioids, encouraging and allowing an addictive behavior to develop and progress,
- 4) a primary biological effect of starvation: starvation may cause depression, irritability, and food preoccupation so psychological symptoms *result from* rather than *cause* starvation

Psychological theories are mainly rooted in the following aspects of perception, which may contribute to the psychopathology:

maladaptive learned processes that reduce anxiety,  
low self-esteem and self-confidence,  
conflicts over adolescent development, and  
family factors (no proven impact) including:  
parent overinvolvement w/ child,  
overt conflict between parents or parent/child avoided,  
rules about family communication so rigid that do not allow  
expression and tension relief

Social/Cultural theories are some of the most well known theories to both the medical and lay population as media portrayals of the “ideal body” have been frequently cited as contributing to the increasing incidence of eating disorders. The Steiner meta-analysis, as mentioned below, addresses and supports this notion to a certain extent.

## Anorexia nervosa

### *Diagnostic criteria* (according to DSM-IV):

- A. Refusal to maintain body wt at or above a minimally normal wt for age and height (e.g., wt loss leading to maintenance of body wt less than 85% of that expected or failure to make expected weight gain during the period of growth, leading to body wt less than 85% of expected)
- B. Intense fear of gaining wt or becoming fat, even though underweight
- C. Disturbance in the way in which one's body weight or shape is experienced; Undo influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body wt
- D. In postmenstrual females, amenorrhea, i.e., the absence of at least 3 consecutive menstrual cycles

### *Epidemiology*

Prevalence: 0.5 % in young adult women

Gender ratio: 1:10 male:female

Avg age of onset: 17 years but bimodal at 14 years and 18 years

Mortality rate: usually from starvation or suicide, 10% within 10 years, 20% within 20 years

Recovery: full recovery in 30-50% of pts within a few years

### *Associated features*

Obsessive-compulsive disorder or personality traits occur with a lifetime prevalence of 25%

Depressive symptoms occur most frequently with major depression occurring in 50%

### *Treatment*

The goals of treatment when presented with a patient diagnosed with anorexia nervosa are both an immediate improvement in nutritional status and the long-term therapy of the psychiatric disease that has contributed to the condition of the patient. In correcting the physiologic deficiencies of starvation, the majority of patients require hospitalization (those that do not are usually highly motivated with a cooperative family, of a younger age, and having experienced symptoms for a relatively brief duration). For patients who refuse adequate nutritional intake, forced feedings may be a last resort. To this point, the administration of exogenous estrogens, calcium, and/or Vitamin D have not been shown to reverse the osteoporosis or osteopenia suffered during the illness. Once the patient gains greater nutritional stability, psychiatric intervention may be attempted through numerous modalities:

- 1) behavioral treatment for wt gain – positive and negative reinforcements based on absolute weight not eating patterns, more efficient than psychotherapy or pharmacotherapy alone in terms of length of hospital stay
- 2) individual and family therapy for wt gain or psychiatric issues – discusses control by parents, pt's motivation to improve may increase, more useful after initial weight gain causes cognition to improve, more effective to solve psychological issues of disease than to increase wt gain, usually performed once or twice weekly
- 3) pharmacotherapy to increase hunger and/or wt – few studies to date have shown conclusively long-term advantages of medication programs vs. non-medication programs (fluoxetine maintained wt after initial gain in Kaye et al 1991; SSRIs helped for wt gain Gwirtsman et al 1990)
- 4) antidepressants for psychiatric issues – efficacy in underweight pt questionable (possible more cardiotoxicity in tricyclics), despite little evidence SSRIs used for depressive and obsessive-compulsive symptoms when psychotherapy and behavior treatments are failing

## Bulimia nervosa

*Diagnostic criteria* (according to DSM-IV):

- A. Recurrent episodes of binge eating
- B. Recurrent inappropriate compensatory behavior in order to prevent wt gain
- C. Binge eating and compensatory behaviors both occur, on average, at least twice a week for 3 months
- D. Self-evaluation is unduly influenced by body shape and wt
- E. The disturbance does not occur exclusively during episodes of Anorexia Nervosa

### *Epidemiology*

Prevalence: 2 % in young adult women

Gender ratio: 1:9 male:female

Avg age of onset: late adolescence/early adulthood

### *Associated features*

Borderline personality traits are seen in 50% of pts

Major depression or anxiety are found in 75% of pts

Substance abuse can eventually be seen in 33% of pts

### *Treatment*

Unlike pts suffering from anorexia nervosa, pts diagnosed with bulimia nervosa usually do not require hospitalization unless there is a complete failure to respond to treatment as an outpatient or unless a mood disorder with suicidal ideations is prominent. In the absence of these conditions, treatment usually is comprised of the following:

- 1) cognitive and behavioral treatment – includes several weeks of group sessions with multiple stages, 1<sup>st</sup> stage: establish control over eating by self-monitoring (with daily logs or diaries) and response prevention including avoiding environments that encourage behavior, 2<sup>nd</sup> stage: restructure unrealistic expectations, 3<sup>rd</sup> stage: prevent relapse for 6 months to a year (most likely time to relapse), 50-90% improve by reducing bulimic episodes, 1/3 stop entirely with this form of therapy
- 2) psychodynamic therapy as for borderline personality disorder – few studies have compared to cognitive and behavioral treatment, focuses on issues of grief, losses, and interpersonal difficulties such as personality disorders, most programs integrate psychodynamic with cognitive and behavioral treatment
- 3) antidepressants – studies support theory that SSRIs, tricyclics, MAOIs reduce bulimic episodes (Agras et al 1992, Levine et al 1992, Walsh et al 1991), fluoxetine and imipramine and despiramine most often used, higher doses of fluoxetine than needed for depression, comparable doses of imipramine and despiramine as needed for depression, MAOIs have side effects and need tyramine free diet (not often used), similar effects as those of cognitive and behavioral treatment: 50-90% reduce episodes, 1/3 stop entirely

Further information provided by the articles cited in the reference section but not otherwise mentioned include:

Lewinsohn study: comorbidities of eating disorders with other psychopathology: 89.5% prevalence over lifetime for female adolescents 23 per 1000  
for male adolescents 1.4 per 1000  
comorbidities: depression: 84.2% vs. 31.0 % (control)  
subthreshold bipolar disorder: 26.3% vs. 3.8% (control)  
anxiety: 21.1% vs. 10.9% (control)  
substance abuse: 31.6% vs. 9.5% (control)

Steiner meta-analysis: Lucas et al, 1991 found that the prevalence of eating disorders has increased over the last 50 years with a presence in a wider range of socioeconomic groups both of which are seen to correlate to an increase in the favorable media portrayal of “thinner” individuals

*comorbidities:*

Herzog et al, 1992 found 62% lifetime comorbid affective disorder especially in pts with mixed anorectic and bulimic features

Rastam, 1992 found 35 % of anorectic have obsessive compulsive disorder

Herzog et al, 1992 found overlap between avoidant personality and anorexia nervosa and borderline personality and bulimia nervosa

*risk factors:*

Marchi and Cohen, 1990 found maladaptive early eating patterns increased the likelihood of later problems (picky eating and digestive maladies predict anorexia, while pica and fighting with meals predict bulimia)

female gender, pear-shaped body, and body mass index high in fat are considered risk factors for both eating disorder types while sexual abuse seems to be a greater risk factor for bulimia

References:

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