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Eating Disorders: Behind the Scenes

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Etiology, Complications, and Treatment

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ABSTRACT

Eating disorders have become so widespread in our society that it is estimated that seven to ten million women and one million men have an eating disorder. Eating disorders do not discriminate; they can affect people of any race, age, gender, ethnicity, religion, and economic status. Research shows that about 76 percent of eating disorders begin between 11 and 20 years of age. Of all the diagnosed eating disorders, 77 percent will have a duration of one to fifteen years of the individual's life. Six percent of serious eating disorder cases end in death. An eating disorder is defined as a psychological disorder characterized by serious disturbances of eating behavior. There are three main types of eating disorders: anorexia nervosa, bulimia nervosa, and binge eating (1). This paper will discuss the etiology, complications, and treatment of each type of eating disorder.

Anorexia Nervosa

Background

Anorexia nervosa is the first term most often thought of when eating disorders are mentioned and in fact, anorexia nervosa is the eating disorder most often studied. Anorexia nervosa is a psychiatric illness (along with all other eating disorders) defined as a disease characterized by refusal to maintain a minimally normal body weight, intense fear of gaining weight, body image distortion, and amenorrhea in postmenarcheal females (2). An individual diagnosed with anorexia nervosa will often take drastic measures to lose weight such as: self-induced vomiting, starvation, use of laxatives, and excessive exercise. There are two subtypes of anorexia nervosa: restricting (starvation) and binge/purging. For the purpose of this paper, all statements about anorexia nervosa patients are in reference to the restricting type unless otherwise stated.

An individual suffering from anorexia nervosa looks in the mirror and sees fat even when the individual has become extremely thin. Life-time duration of anorexia nervosa is estimated to range from 0.3 percent to 3.7 percent in females and is about one tenth of that for males (2). As demonstrated by the statistics, anorexia nervosa affects women the most, but still approximately 5 percent to 10 percent of all cases involves males (3). Anorexia nervosa also has the highest death rates of all other mental illnesses (4).

An individual must meet certain criteria to be clinically diagnosed with anorexia nervosa. There are four criteria and an individual must meet all four to be diagnosed. Criteria are based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The four criteria are: 1) refusal to maintain a body weight at or above normal weight for age and height, 2) having an intense fear of gaining weight, 3) distorted body image, and 4) amenorrhea (5).

There is not a specific known cause for anorexia nervosa, but it is believed that there are many contributing factors that when combined, increase an individual's risk of developing anorexia nervosa. These include biological factors, psychological factors, family and social issues. Once an individual is diagnosed with anorexia nervosa, physical health, psychological health, and quality of life start to spiral downward quickly. If not treated, this disease may continue throughout the individual's entire life and may lead to death. As previously stated, 6 percent of serious eating disorders cases are fatal (1).

Genetics

Genetics is a topic of concern among researchers in the field of eating disorders. Until the 1990s anorexia nervosa was believed to be caused by psychological and environmental issues. However, this changed when researchers began to find evidence that genetics played a

role in anorexia nervosa. Several studies have produced evidence that there is a linkage between genetics and a predisposition to the development of anorexia nervosa. In the published study, Evidence for a Susceptibility Gene for Anorexia Nervosa on Chromosome 1, in the *American Journal of Human Genetics*, researchers stated that eating disorders such as anorexia nervosa "have a significant genetic component" (6). This study researched 192 families that had at least two family members affected by an eating disorder. One of the two family members with an eating disorder had to be diagnosed specifically with anorexia nervosa. Results of the statistical analysis on the affected participants were significant enough that the researchers decided to narrow down the sample. The researchers then evaluated 37 families that had two members affected by anorexia nervosa rather than just one member affected by anorexia nervosa. Research evidence suggested a genetic linkage on chromosome 1 to anorexia nervosa (6).

Another study published in *Human Molecular Genetics* in 2002 found evidence for genetic linkage on chromosome 1, chromosome 2, and a region on chromosome 13 (7). Analysis on 196 families, all of which had at least one family member diagnosed with anorexia nervosa, was conducted. The focus was on three aspects of genetics: personality, psychiatric traits, and temperament phenotypes. When comparing these traits between affected sibling members (those diagnosed with anorexia nervosa) and non-affected sibling members, two covariates were found in the affected sibling. These two covariates were: a drive for thinness and obsessionality. The researchers analyzed the covariates (together and separately) and found genetic linkage to anorexia nervosa on chromosome 1, chromosome 2, and chromosome 13 (7). This analysis provided evidence that traits found in anorexia nervosa (e.gl drive to be thin, obsessive, perfectionism, etc.) are found on genes in anorexia nervosa patients. Anorexia nervosa and linkage to genetics is still under investigation and a specific gene has not yet been identified.

Genetics is also believed to play a role in the development of depression, which is a biological abnormality common in anorexia nervosa patients. A study published in the *American Journal of Psychiatry* in 2000 concluded that there is a link between anorexia nervosa and genetics, which in turn contributes to the observed comorbidity between anorexia nervosa and major depression. In a study, 2,163 female twins from the Virginia Twin Registry were interviewed (each twin's information was confidential from their co-twin). This study found that 34% of the participants had a shared genetic variance between anorexia nervosa and major depression (8). Genetics is not the only factor that contributes to anorexia nervosa and depression. Other abnormalities in the body have been found to play a role in the development of anorexia nervosa.

Biological Abnormalities

Researchers have begun to study brain chemicals and have found irregularities in neurotransmitters, hormones, amino acids, and neuropeptides. Many of the brain chemicals investigated involve satiety, hunger, and digestion, along with other functions in the body. Some examples of hormones that are low in anorexia nervosa patients are serotonin, cortisol, estrogen, dehydroepiandrosterone (DHEA), and dopamine.

Neurotransmitters and the binding to their neurons have become a major study in all eating disorders. This field of study became a major interest when many cases of anorexia nervosa where also being linked with major depression, stress, obsessive compulsive disorder (OCD), and other psychological disorders. Researchers started to discover that patients suffering with anorexia nervosa (along with other eating disorders) either had deficiencies of neurotransmitters (norepinephrine and epinephrine) or the presence of neurotransmitters that were not properly binding to neurons. Abnormalities of neurotransmitters may cause false

signals of hunger and satiety in the brain. The role of neurotransmitters in the etiology of anorexia nervosa is still being investigated and additional research is needed before conclusive results are established.

Irregularities in hormones also appear to contribute to false signaling which may lead to the development of anorexia nervosa. Serotonin is one of the most commonly studied hormones (and may act as a neurotransmitter) linked to anorexia nervosa and depression. Serotonin is secreted from the gastrointestinal tract and the brain and aids in the regulation of appetite, mood, and muscle contraction (2). Serotonin is often studied in correlation with depression. "Approximately 50 to 75 percent of all individuals with eating disorders have a lifetime history of major depressive disorders," (9). There is much debate as to whether major depression may increase the risk of development of anorexia nervosa or vice versa. Researchers continue to investigate the role of serotonin and its relationship between major depression and anorexia nervosa. Low levels of serotonin have been linked to increased risk of depression because of serotonins' role in regulating mood. Low levels of serotonin are also linked to anorexia nervosa because of serotonins' role in satiety (2). Depression among anorexia nervosa patients is a concern for many reasons; the main reason is the increased risk of suicide. The suicide rate is much higher among anorexia nervosa patients than the general population (4).

Serotonin is not the only hormone found to be irregular in patients suffering from anorexia nervosa. Another hormone that has been heavily researched is cortisol. Cortisol's primary role in the body is to increase blood sugar by counter-acting insulin, increase glycogen stores in the liver, help with fat, protein, and carbohydrate metabolism, and aid in the suppression of the immune system in response to stress (2). One study assessed 24 hour multiple blood sampling studies on patients that had been diagnosed with depression and anorexia

nervosa. In the study, a catheter was placed in the forearm of the individual and every 20 to 30 minutes a blood sample was taken from the patient for a 24 hour period. Serum levels of cortisol were collected. "Approximately half of patients with major depressive illness and a similar or greater portion of patients with anorexia nervosa exhibit elevated 24-hour mean levels of plasma cortisol, and this abnormality disappears as the patients recover," (10). Cortisol was found to be broken down slower in the body of patients with anorexia nervosa, which could be a cause for the higher levels of cortisol in the body. Even though cortisol was broken down more slowly in patients with anorexia nervosa, the patients had a higher rate of cortisol production; cortisol is produced proportionally to body size. Anorexia nervosa patients had higher rates of cortisol production for body size. Increased production of cortisol comes from an increased adrenal function in the body, which has been found in anorexia nervosa patients and patients with major depression. The researchers sited the increased adrenal activity as the cause of increased secretions from the hypothalamus (10). The hypothalamus is the portion of the brain that controls body temperature, hunger, thirst, fatigue, and circadian cycles. It also plays a major role in connecting the endocrine system to the nervous system via the pituitary gland (11). Therefore, if anything is irregular with hypothalamic secretions a cascade of problems occur with hormones and body regulation.

Even though there appears to be many similarities in endocrine disturbances in patients with depression and anorexia nervosa, there is not enough data to conclude there is an identical neuroendocrine dysfunction in both major depression and anorexia nervosa. The researchers also pointed out that while a lot has been learned about hormonal disturbances in anorexia nervosa, it is hard to determine the degree to which endocrine dysfunctions contribute to the illness of anorexia nervosa (10).

Estrogen and dehydroepiandrosterone (DHEA) are two sex hormones that are commonly found to be very low in anorexic patients. Estrogen is important for healthy heart and bones. DHEA is also important for bone health and for other functions (12). Estrogen is needed for the proper functioning of the menstrual cycle. Without estrogen a serious complication called amenorrhea may develop. Amenorrhea will be discussed in the complication section of this paper. Decreased levels of estrogen and DHEA in the body increase an individual's risk of developing osteopenia, the precursor to osteoporosis. Bone remodeling, which aids in the prevention of osteoporosis, is highest during adolescence. Calcium, Vitamin D, estrogen, DHEA, and other nutrients are needed during this time of an individual's life to allow for proper bone remodeling and strong bones to be built to prevent osteoporosis. Without estrogen and DHEA, bone remodeling suffers and therefore increases the individual's risk of developing osteoporosis (2).

Psychological Issues

Anorexia nervosa is a psychiatric illness and therefore involves many psychological issues. As previously discussed, depression is a common psychological issue in anorexia nervosa patients and often develops due to low serotonin levels. Perfectionism and low self-esteem are two other psychological issues contributing to anorexia nervosa and seem to go hand-in-hand. Many patients diagnosed with anorexia nervosa are perfectionist, have very low self-esteem, and isolate themselves from family, friends, and other relationships because of this lack of confidence. Anorexia nervosa patients must have the perfect body, the perfect weight, and complete control over the food that enters and leaves the body; this is where the eating disorder begins to develop. Even though anorexia nervosa patients are striving for perfection, low self-esteem persists. Low self-esteem begins to affect relationships with family and friends.

Individuals with anorexia nervosa are ashamed of the eating disorder and ashamed of the feeling of not being good enough for family and friends. The individual slowly starts to withdraw from relationships unable to handle the pressure or the possibility of failure in relationships. A slow, trickling effect caused by low self-esteem, shame, and embarrassment leads to isolation; a common feature of anorexia nervosa patients.

In 1995 a study of 81 female college students measured the links between different dimensions of perfectionism and eating disorder symptoms, body image, and self-esteem. Before beginning the project, researchers categorized perfectionism into three sub-categories. The three levels of perfectionism are: need to appear perfect, avoiding imperfectionism, and avoiding disclosure of imperfection (13).

Female students participating in the study ranged in age from 17 to 45 with the average age being 20.9 years of age. Each participant was assessed based on the 1) Multidimensional Perfectionism Scale, 2) Perfectionistic Self-Presentation Scale, 3) EAT Scale, 4) Bulimia Test, 5) Body Image Avoidance Questionnaire (BIAQ), and 6) Feelings of Inadequacy Scale. The 81 participants were put into small groups ranging from 2 to 8 participants and administered the questionnaires. Results showed a correlation of self-oriented (perfection standards inflicted by the individual) and socially prescribed perfectionism (perfection standards inflicted from society, perceived by the individual) with the EAT Scale. Social perfectionism was correlated with the Bulimia Test and the Body Image Avoidance Questionnaire (related to clothing and restraint). Self-oriented perfectionism was also correlated with Body Image Avoidance Questionnaire in the appearance and global self-esteem categories. Self-presentation perfectionism was correlated with all three (Bulimia Test, EAT Scale, and Body Image Avoidance Questionnaire) scales (13).

"In addition, the need to appear perfect and the need to avoid appearing imperfect were correlated with the bulimia subscale of the EAT. The need to avoid appearing imperfect was positively correlated with BIAQ total scores, and both the need to appear perfect and the need to avoid disclosure were correlated with the BIAQ Restraint Scale. Researchers concluded that while self-oriented perfectionism is related only to anorexia nervosa trends and attitudes; social perfectionism is widely linked to eating disorder behaviors and self-esteem," (13). The study stresses the importance in distinguishing the dimension of perfectionism because certain dimensions are more characteristic of one disease than another. For example, researchers concluded that anorexia nervosa patients tended to have self-imposed perfectionism. "The current findings, and the findings of Davidson, support results of anorexia nervosa and self-imposed tendencies and suggest that self-oriented perfectionism may be specifically linked to dieting and concerns with being thinner, but may not be as involved in other aspects of eating disordered behavior" (13). Social perfectionism was linked to self-esteem and the need to conform to the environmental definition of thin, while other-oriented perfectionisms were found to be linked to body image avoidance.

Along with perfectionism and low self-esteem, the inability to express emotions (alexithymia) and the need for control are common psychological issues in anorexia nervosa patients. Alexithymia is very common among anorexia nervosa patients. In fact, one source found 65 percent of anorexia nervosa participants scored within the alexithymia range on the Toronto Alexithymia Scale (TAS-20) (14). Another study tested the emotional functioning of young anorexia nervosa patients. This study had one test group and two control groups. The test group consisted of the anorexia nervosa patients and the two control groups consisted of psychiatric patients (with depression or anxiety) and healthy patients. One of the tasks of this

study was to complete an alexithymia questionnaire. The anorexia nervosa patients scored considerably lower on the questionnaire than the healthy patients (15).

Need for control is another psychological issue in individuals with anorexia nervosa. Since humans have the choice of what enters and leaves the body, food is a perfect form of control for anorexia nervosa patients. Individuals control weight, the amount of calories consumed, the amount of fat consumed, and the number of hours spent exercising. Very tight control, to the point of being obsessive, is what anorexia nervosa patients perceive to be a method of staying thin.

Environmental Issues

Even though thousands of studies have been conducted on the etiology of anorexia nervosa patients and many factors contribute to this disease, a strong contributing factor is the individual's environment. Anorexia nervosa is often triggered by a traumatic event (e.g. sexual abuse, divorce, family dysfunction) and/or pressure from society to be as thin as the airbrushed models seen in magazines. The pressure to be thin and beautiful is affecting youth more and more each day and is starting at younger ages. Children as young as eight years of age are being diagnosed with anorexia nervosa and are showing signs and symptoms as early as six years of age (16). The internet, television commercials, billboards, magazines, friends, and family impose perfectionism and pressure to be thin with images, tips, remedies, and advice on how to improve health, lose weight, and look perfect.

Sexual abuse is an example of a traumatic event that may contribute to the development of anorexia nervosa in individuals, especially young girls. "Sexual abuse has been reported to occur in 30 percent to 65 percent of women with eating disorders," (17). Sexual abuse is seen

in three out of every ten anorexia nervosa patients (18). Even though the rate of sexual abuse is very high among individuals with eating disorders, there is controversy about whether sexual abuse actually has an affect on the development of an eating disorder. One reason for the controversy is because the rate of sexual abuse is high among many other psychiatric illnesses, in fact, sexual abuse is found in 13 percent to 40 percent of other psychiatric illnesses. Studies that subcategorized eating disorders from other psychiatric illnesses found that 4 percent to 53 percent of anorexia nervosa patients were sexually abused (17).

One study completed in 1997, categorized participants into four groups: 1) anorexia nervosa, 2) bulimia nervosa with comorbid substance dependence, 3) bulimia nervosa without comorbid substance dependence, and 4) control women. All subjects met the criteria for either anorexia nervosa or bulimia nervosa based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) criteria. Each participant had a face-to-face interview with a master's or doctoral-level psychologist. A modified version of the *Schedule for Affective Disorders and Schizophrenia-Lifetime* questionnaire was used in the interview. Since the questionnaire does not have a section on sexual abuse, the psychologist would ask open-ended questions about sexual abuse during the posttraumatic stress disorder section and the participant was asked to elaborate as much as possible about a history of sexual abuse. A history of sexual abuse was also discussed during the section of the questionnaire that covered difficult times or periods in the individual's life. The psychologist would ask the participant to outline all difficult periods.

In the same study, three-fourths of the first-degree relatives of participants were interviewed either face-to-face or by telephone. The relatives were asked questions about whether or not the participant had been sexually abused. "There were significantly different overall rates of sexual abuse across the four subject groups," (17). The bulimia nervosa with

substance abuse group had significantly higher rates of sexual abuse than the other three groups. Both the bulimia without substance abuse group and the anorexia nervosa group had significantly higher rates of sexual abuse than the control women group. The type of sexual abuse among each group was also studied and results showed that fondling by a nonfamily member was the most common form of sexual abuse for the anorexia nervosa group.

The study also compared age of onset of the eating disorder and the age sexual abuse occurred. There were no significant differences for the ages of eating disorder onset compared to the age of the participant's first encounter of sexual abuse. However, in a majority of all subjects that reported sexual abuse, the sexual abuse preceded the eating disorder. Twenty-three percent of the anorexia nervosa group had a history of sexual abuse (17).

Development of eating disorders as related to sexual abuse is still inconclusive and continues to be investigated. One reason is that there are a variety of outcomes from current literature on sexual abuse in relation to the development of eating disorders. Another reason this study concluded the link to be inconclusive was because sexual abuse occurred in only 40 percent of all eating disorder participants, which caused doubt that sexual abuse is a factor that contributes to the development of eating disorders (17).

Bulimia Nervosa

Background

Bulimia nervosa is another type of disordered eating. Bulimia nervosa is defined as episodes of binge eating (eating very large quantities of food) followed by compensatory behavior. Compensatory behaviors are usually one or more of the following: self-induced vomiting, abuse of laxatives and enemas, diuretics, pills that help weight loss, and excessive

exercising. There are two subtypes of bulimia nervosa patients: purging and non-purging. A binge episode is described as the consumption of large amounts of food, often 1,000 to 2,000 calories, in two hours or less (2).

As with anorexia nervosa, there are criteria that an individual must meet before being clinically diagnosed with bulimia nervosa. Criteria for diagnostic purposes are stated by the DSM-IV and are as follows: 1) recurrent episodes of binge eating, 2) recurrent inappropriate compensatory behaviors to avoid weight gain, 3) binge eating and compensatory behaviors occur at least twice a week for three months, 4) self-evaluation is influenced by body shape and weight, and 5) symptoms do not occur exclusively during episodes of anorexia nervosa (9). It is estimated that about 1.5 percent of females and 0.5 percent of males have suffered from bulimia nervosa. Between 1 percent and 2 percent of late adolescent and adult women meet the criteria for bulimia nervosa. Only about 50 percent of people with bulimia nervosa fully recover, about 30 percent show some improvement, and about 20 percent remain bulimic throughout lifetime (18). Most often the picture of an individual with an eating disorder is of a young girl who resembles a skeleton. An individual suffering from bulimia nervosa often is of normal weight or may be slightly underweight or overweight.

The majority of studies on bulimia nervosa have been more focused on the prevalence of the illnesses and not as much on the etiology. Etiology of bulimia nervosa is very similar to that of anorexia nervosa. Biological abnormalities, psychological disorders, and environment all seem to play a role in the contribution to bulimia nervosa. Research is on-going in the area of etiology of bulimia nervosa.

Genetics

Studies have linked bulimia nervosa to chromosome 10, especially bulimia with self-induced vomiting (19). A first study published on the linkage of chromosome 10 and bulimia nervosa was completed in 2003 and involved 308 families that had been diagnosed with bulimia nervosa. This study did a linkage analysis to try and find regions of the genome that were holding genetic abnormalities predisposing an individual to bulimia nervosa. First, the researchers tested all 308 families using the logarithm of odds score (LOD score) (20). The LOD score is a statistical estimate of whether two loci are near to each other and might be inherited as a pair. The LOD score of the 308 families was a 2.92 on the chromosome 10. The next test was narrowed down to 133 families, all of which had at least two relatives diagnosed with bulimia nervosa. The LOD score of the second cohort group was 3.39 on chromosome 10. "These results provide evidence of the presence of a susceptibility locus for bulimia nervosa on chromosome 10p," (20).

Recently, a hereditary gene for an addictive behavior has been linked to bulimia nervosa. Many suffering from bulimia nervosa have some type of addictive behavior, such as substance abuse of alcohol, drugs, or addictive behaviors (e.g. exercise, purging, over-eating, etc.). Scientific research recently has shown that people are born with an inherited predisposition towards developing bulimia, particularly when susceptibility to addiction is in the genes (21). There has not been a specific addiction gene found, just a general gene. The addiction gene is not a concrete link to bulimia nervosa because not all individuals who inherit the addiction gene suffer from bulimia or any other types of addictions. The role of genetics in bulimia nervosa is still being investigated.

Psychological Issues

Along with biological disturbances, psychological issues are present in bulimia nervosa patients. Depression, anxiety, and obsessive-compulsive disorder (OCD) are commonly found in individual's suffering from bulimia nervosa. Substance abuse also occurs in bulimia nervosa patients and may lead to more altered biological functions. One study found a significant comorbidity between bulimia nervosa and anorexia nervosa, major depression, alcoholism, panic attacks, anxiety, and phobia (21).

Depression appears to be caused by low levels of serotonin, epinephrine, norepinephrine, and dopamine. Serotonin controls depression, anxiety, impulsiveness, and influences appetite as stated in the psychological issues section of anorexia nervosa. When serotonin levels are low mood and appetite are thought to be affected. An individual suffering from bulimia nervosa that has been diagnosed with major depression is found to have low serotonin levels; just as anorexia nervosa patients with depression have low serotonin levels. Low serotonin levels are often studied in correlation to depression and anorexia nervosa, but studies are now beginning to look at low serotonin levels and affects on bulimia nervosa as well as anorexia nervosa.

Perfectionism, low self-esteem, and impulsiveness are other psychological issues that are common among people with bulimia. Like anorexia nervosa sufferers, many bulimia nervosa patients strive for perfection. Never being good enough, smart enough, thin enough, or perfect enough causes individuals to have low self-esteem. Even though perfectionism is found among bulimia nervosa patients, perfectionism is more highly associated with anorexia nervosa. People with bulimia nervosa tend to be more impulsive. Binges consist of consuming thousands of calories in one sitting followed by compensatory behaviors to rid the body of the calories. One study compared 58 normal weight bulimia nervosa patients to 27 normal controls in the areas of

bulimia nervosa, depression, impulsiveness, impulse control (dyscontrol), and obsessive traits. Normal weight bulimia patients scored higher in each category of the questionnaire compared to the normal control group. About half of the normal weight bulimia group met Lacey's multi-impulsive bulimia criteria (22).

Environmental Issues

As in anorexia nervosa, environmental factors such as family dysfunction (e.g. divorce), pressures to be thin, and sexual abuse all contribute to the development of bulimia nervosa. Of these environmental factors, sexual abuse has been shown to play a larger role in the development of bulimia nervosa. In the early 1990s, a study conducted by Pope and Hudson concluded that there was no evidence that childhood sexual abuse contributed to the development of bulimia nervosa. This particular study started many controversial debates that lasted for several years. "Well-designed national studies by Dansky, Brewerton, Wonderlich, and others have supported the idea that childhood sexual abuse is indeed a risk factor for the development of bulimic pathology among women," (4). Studies are currently investigating the degree to which environmental factors, especially sexual abuse, contribute to the development of bulimia nervosa.

Complications of Anorexia Nervosa and Bulimia Nervosa

The health complications that may arise from anorexia nervosa and bulimia nervosa are destructive to the body and may even be fatal if left untreated. Both illnesses affect many systems of the body such as: circulatory system, gastrointestinal tract, central nervous system, endocrine system, skeletal system, and the oral cavity (more common among bulimics).

Circulatory System

Electrolyte abnormalities are common in anorexia nervosa and bulimia nervosa where self-induced vomiting, abuse of laxatives, diuretics, and enemas are present. Restricting anorexia nervosa and non-purging bulimia nervosa often have normal electrolyte balance. Self-induced vomiting and abuse of laxatives, diuretics, and enemas cause the loss of bicarbonate, chloride, potassium, sodium, and fluids often putting individuals below normal serum range. The body has a narrow pH range of 7.35 to 7.45, when the body loses bicarbonate the blood becomes alkaline which is known as metabolic alkalosis (2). If left untreated, metabolic alkalosis is fatal. Potassium is needed in the body for a healthy heart beat and sodium and chloride are needed by the body to maintain blood pressure.

Hypomagnesemia is another common electrolyte imbalance, but this imbalance is mostly found in bulimics that abuse diuretics. Low levels of magnesium and calcium can cause muscle cramps. Of all the electrolyte imbalances, metabolic alkalosis is the most common among bulimics who frequently engage in self-induced vomiting or abuse of diuretics (23).

With the loss of fluids and sodium the patient quickly becomes dehydrated, this in turn increases blood urea nitrogen and creatinine levels in the urine. While self-induced vomiting does more damage to the body, diuretics will cause loss of sodium and calcium via action on the kidneys. The body secretes aldosterone (a hormone) in response to loss of sodium and low intravascular volume. Aldosterone signals the body's rennin-angiotensin system to counter act this dehydration and low intravascular volume by signaling the body to reabsorb sodium and bicarbonate from the kidneys. Laxative abuse is also common among anorexia nervosa and bulimia nervosa. Laxatives cause losses of chloride and potassium in the stool (23). As stated

previously, chloride is needed for maintenance of blood pressure and potassium aids in a healthy heart rhythm.

Electrolyte imbalances may cause muscle weakness, fatigue, dizziness, and even depression. Potassium, calcium, and magnesium help maintain heart rhythm; if levels become too low (severe hypokalemia, hypocalcemia, and/or hypomagnesemia respectively) the individual may develop cardiac arrhythmias and sudden death (23).

Gastrointestinal

Another set of medical complications that are seen in anorexia nervosa and bulimia nervosa patients are problems with the gastrointestinal (GI) tract. One of the first and most common problems in eating disorder patients is delayed gastric emptying, gastroparesis, which usually occurs after about the first 10 pounds of weight loss and may cause bloating, constipation, early satiety, nausea, and reflux/vomiting (not self-induced). Delayed gastric emptying is often caused by decreased motility of the gastrointestinal tract. Delayed gastric emptying causes early satiety, this occurs because the food is not passed on from the stomach to the small intestine as in healthy individuals. Instead, the food remains in the stomach which leads to early satiety. Delayed gastric emptying and decreased GI motility is usually solved with slow weight restoration; however, because of bloating and feeling uncomfortable, the patient will not eat a meal. Feeding the patient liquids is the recommendation. Also, medications are often given to help with the motility of the stomach, allowing the stomach to empty quicker, reducing early satiety (23).

As stated earlier, reflux is also a common GI tract problem. Some anorexia nervosa patients will experience a mild version of reflux due to weight loss-induced gastroparesis.

Severe reflux is seen in bulimia nervosa patients that purge. The strong acidic contents of the stomach that reflux into the esophagus will cause a burning sensation, or "heartburn." This may result in sore throat, coughing, dysphagia (difficulty swallowing), or painful swallowing. Anorexia nervosa patients are often treated with antacids for mild reflux, but bulimia nervosa patients are treated with stronger medications, such as H_2 blockers. Frequent reflux in bulimics put this group at risk of developing esophagitis that could be a precursor to Barrett's esophagus, which is a precancerous condition (23).

Another common GI tract problem is constipation and diarrhea. Constipation is commonly seen in anorexia nervosa patients and may be caused by one of two things: decreased caloric intake or medications. Decreased calorie intake is often the primary cause of constipation. With the decreased consumption of calories, the muscles of the intestinal tract decrease motility. Decreased motility of the GI tract causes constipation. Diarrhea occurs in bulimia nervosa patients due to abuse of laxatives. Frequent diarrhea is harmful to the body because it causes large losses of electrolytes and fluids. Getting a patient to stop the use of laxatives is the larger problem than the diarrhea itself. Patients continue the use of laxatives for two reasons: 1) the patient's body has become dependent on the laxative for bowel movements and 2) the patient believes it is a good way to lose weight even though laxatives act on the colon and calories are absorbed in the small intestine (23). The treatment team must encourage the patient to stop the abuse of laxatives in order for the GI tract to regain normal function.

Abdominal pain is also a common complaint of anorexia nervosa patients but is usually linked to gastroparesis, reflux, bloating, constipation, or symptoms common in irritable bowel

syndrome. If abdominal pain continues after treatment of other complications then further evaluation should be done to check for possible complications (23).

Cardiovascular

Even though gastrointestinal tract problems can be severe and even fatal, cardiovascular complications from eating disorders are much more serious and life-threatening. "Patients with anorexia nervosa have the highest premature mortality rate of any diagnostic group of psychiatric patients," (23). Anorexia nervosa patients are at risk of heart failure during early weight restoration, not just their lowest weight. The heart of an anorexic patient shrinks significantly, has decreased cardiac output (leads to bradycardia), and loses muscle mass over time. When the patient enters into recovery and begins to gain weight the heart heals at a slower rate than the rest of the body. In severe anorexia nervosa patients, it is common for the heart to fail during recovery. The heart has become so small and weak it is unable to handle the "work" of rehabilitation. Rehabilitation of severe eating disorder patients must be a slow process and monitored closely to decrease the risk of heart failure (23).

There are several factors that may increase an eating disorder patient's risk of cardiovascular disease such as: type, duration, and severity of purging methods; duration and rapidity of weight loss; and severity of abnormal eating habits (23). Anorexia nervosa and bulimia nervosa patients abusing diuretics have the greatest risk for cardiac complications because of potassium losses.

Death due to cardiovascular complications among bulimics is much less common but still occurs. Most often, deaths of bulimia nervosa patients from cardiac complications is due to hypokalemia from purging (23). However, it is becoming common for bulimia nervosa patients

to use a medication called ipecac to induce vomiting. This medication contains emetine which can cause irreversible cardiomyopathy. The abuse of ipecac by patients with bulimia nervosa is increasing primarily for the purpose of self-induced vomiting (5).

Common signs and symptoms of cardiac complications in eating disorder patients are: fatigue, dizziness, blue nail beds, palpitations, chest pains, leg pains, and dyspnea. Physical examinations, X-rays, and electrocardiogram (EKG) are all tests that may be done to evaluate the presences and/or severity of cardiac complications (23).

Endocrine and Skeletal

Anorexia nervosa and bulimia nervosa patients have complications with endocrine and skeletal systems. Eating disorders are stressful on the body, which release certain hormones during stressful times to help the body adapt. The hypothalamus is the part of the body, found in the brain, that responds to stress. It reacts by releasing adrenocorticotropic hormone (ACTH), cortisol, growth hormone, prolactin, epinephrine, and norepinephrine. Inflammatory mediators (e.g. cytokines, interleukin-1, interleukin-6, etc.) are also released. These inflammatory mediators are thought to play a role in the break down of bone, which leads to osteoporosis (23).

While some hormones are being released during eating disorders, other hormones are being diminished. Most anorexia nervosa patients will experience amenorrhea, which is the loss of the menstrual period for three consecutive months. "In patients with anorexia nervosa, 20 percent may actually experience amenorrhea before the onset of weight loss, and 50 percent may experience amenorrhea during the course of dieting," (23). Many more anorexia nervosa patients will experience amenorrhea after severe weight loss. The reason for the cessation of the menstrual period is because of the diminishment of the hormone gonadotropin-releasing

hormone (GnRH); this hormone is made and secreted from the hypothalamus. GnRH signals for the secretion of luteninizing hormone (LH) and follicle-stimulating hormone (FSH), which determine when a female's menstrual cycle begins and ends. Therefore, without GnRH a woman's menstrual cycle does not function properly. The diminishment of GnRH also decreases sex hormones (e.g. estrogen, progesterone, and testosterone) in the blood (23).

Many other hypothalamic abnormalities occur in eating disorder patients, especially anorexia nervosa patients. There are different axes of the hypothalamus and one of the axes, the hypothalamic-pituitary-adrenal axis, increases its secretion of cortisol during a starvation state. Increase in cortisol production inversely decreases the production of male sex hormones. "These reduced androgen levels foster a diminished anabolic state and may adversely affect bone remodeling and diminish sexual drive," (23). Another hypothalamic abnormality found among anorexia nervosa patients involves vasopressin and growth hormone. Basal growth hormone is increased and insulin-like growth factor is decreased because of abnormalities in vasopressin and growth hormone. A reduction of insulin-like growth factor prevents bone formation and low vasopressin levels have shown an increase risk of diabetes in some patients. Also, the body struggles to regulate its own temperature when the hypothalamus is not functioning properly (23). Cold intolerance is a common sign among anorexia nervosa patients (2).

Additional endocrine complications that must be monitored in anorexia nervosa patients are: the thyroid (decreased T_3 and T_4) hypoglycemia, decreased serum leptin levels, and increased low-density lipoprotein (LDL), high-density lipoprotein (HDL), and total cholesterol. Thyroid abnormalities usually correct themselves with weight and diet restoration. Hypoglycemia is caused by the depletion of glycogen stores from lack of food and excessive

exercise. Once all the glycogen stores are used, glucose levels in the blood fall to low levels (23). Hypoglycemia is corrected with adequate caloric intake.

Infertility

Infertility is another common medical complication seen among women with eating disorders. "Approximately 5-10 percent of the women seen in fertility clinics are found to have anorexia nervosa or bulimia nervosa. Within this same clinic population of infertile women, among those with amenorrhea or oligomenorrhea, approximately 60 percent have eating disorders," (5).

Osteoporosis

As stated earlier, many of the hormones that are increased or decreased during hypothalamic abnormalities have an effect on bone formation. Bone formation is greatest during adolescence; adolescence is also the age with the highest rate of onset for eating disorders. Since eating disorders greatly affect bone remodeling, the risk of osteopenia (the precursor to osteoporosis) is high. "Significant bone loss occurs among young patients with anorexia nervosa," (5). Bone loss can lead to the development of osteoporosis which is often irreversible in eating disorder patients and results in fragile and brittle bones. As the patient ages or loses more bone mass, the risk of bone fractures increases greatly.

Oral and Dental

Cardiovascular, endocrine, and skeletal complications are more associated with anorexia nervosa and gastrointestinal complications with bulimia nervosa. Another complication that is mostly associated with bulimia nervosa patients is oral and dental complications. Oral and dental complications are often the first signs discovered in bulimics from the frequent regurgitation of acidic contents of the stomach. Dentists are often the first to make the

discovery. These complications include: cheilosis, enamel erosion, gingivitis, salivary gland enlargement (seen in about 50 percent of bulimics), and hyperamylasemia (23). Cheilosis are painful sores on the corners of the mouth. Enamel erosion makes the patient very sensitive to hot and cold foods, making it more difficult for them to eat. Susceptibility to dental cavities increases. Enamel erosion has been shown to occur in up to 38 percent of bulimia nervosa patients. Gingivitis, characterized by painful gums, is another complication that makes eating difficult (23).

Binge Eating Disorder

Background

Binge eating is often confused with bulimia nervosa. Binge eating is similar to bulimia in that the individual goes through periods of over-eating, consuming thousands of calories in one sitting. However, this disease differs from bulimia in that it is not followed by compensatory behaviors as bulimia nervosa. The term "binge eating disorder" is the newest category of eating disorders. In fact, binge eating disorder was not even recognized until 1992 when "binge eating disorder" was termed at an International Eating Disorders Conference (4). It is estimated that 1 out of every 142 people, or 19 million people in the United States, has binge eating disorder. "Onset of binge eating disorder generally occurs in late adolescence or in the early twenties, with women being 1.5 times more likely to develop this disorder than men (2). This disorder is more commonly seen in obese individuals and is estimated to affect 2 percent of the general population (1). It has also been estimated that binge eating disorder is prevalent among 15 percent to 50 percent of participants in weight control programs (2).

The criteria for diagnosis of binge eating disorder stated by the DSM-IV is as follows: eating faster than normal, eating until uncomfortably full, eating large amounts even if not

hungry, eating alone often because of embarrassment, and feelings of depression, guilt, or disgust when eating. Behaviors must happen over a period of six months, occurring two times per week with no compensatory behaviors following the binges (4).

As in anorexia nervosa and bulimia nervosa, binge eating disorder is caused by multiple factors. Binge eating disorder is affected by biological, psychological, and environmental factors. Biological factors that have been found to be associated with binge eating are abnormalities in the hypothalamus, genetic mutations, night eating syndrome, and low serotonin levels. Psychological factors that have been found to be linked to binge eating disorder are depression, low self-esteem, body dissatisfaction, loneliness, trouble with expressing feelings, and trouble with impulse control. Environmental factors that appear to be linked to binge eating are society pressures to be thin, parental influences (parents using food as rewards or comforts), criticism of their bodies, and sexual abuse (4).

Biological Complications

Of the altered biological factors found to be associated with binge eating, abnormalities in the hypothalamus appear to be the strongest contributing factor. "It has been proposed that hypothalamic-pituitary-adrenal (HPA) is one of the primary biological origins of alterations in eating behaviors observed in anorexia, bulimia, and binge eating disorder," (9). The hypothalamus controls hunger and satiety in an individual. In patients suffering from binge eating disorder, the hypothalamus sends false signals of hunger and satiety. Levels of 24-hour plasma cortisol, cortisol secretion, urinary free cortisol, luteinizing hormone, follicle stimulating hormone, T4, thyroid-stimulating hormone (TSH), and thyroid-stimulating hormone (TSH) response have all been found to be elevated in the hypothalamus in individuals with binge eating disorder. Hypothalamic levels that are decreased in binge eating patients are: T3 levels, growth

hormone releasing hormone response, and growth hormone secretion in response to chemical stimulation (9). All of the increased and decreased functioning of the hypothalamus alters its signaling of hunger and satiety.

Low serotonin levels are also found among binge eaters. Serotonin plays many roles in the body such as influencing mood and satiety. Low levels of serotonin are thought to contribute to binge eating disorders in two ways: 1) binge eaters will continue to eat and not feel full because signals of satiety are not being sent to the brain via serotonin, and 2) low serotonin levels are also associated with over-all bad feelings and depression, therefore, individuals with binge eating disorder use food to control their feelings to make up for the chemical imbalance (24).

Night eating syndrome is yet another biological issue that has been found in binge eaters. This syndrome occurs when the individual consumes more than half of the daily energy intake during and/or after dinner but before breakfast. One study compared five females with night eating syndrome to five-gender, weight, and age matched controls. All ten participants performed a 120 minutes corticotrophin-releasing hormone test. Blood samples were drawn to look at adrenocorticotropic hormone (ACTH) and cortisol levels. The five females with night eating syndrome had significantly decreased corticortropic releasing hormone (CRH)-induced ACTH and cortisol levels than did the control groups. "In conclusion, disturbances in the hypothalamic-pituitary-adrenal axis with an attenuated ACTH and cortisol response to CRH were found in subjects with night eating syndrome," (25).

Genetics

In the early 1990s researchers began to find other biological connections to the etiology of binge eating disorder. Studies began to find a possible connection between obesity

(individuals unable to control food intake as in binge eating) and genetics. Researchers had already concluded that obesity was linked to genetics, but had not been able to find a specific gene. After several studies, including a study lead by Dr. Kaplan an associate professor at Albert Einstein College of Medicine of Yeshiva University, found individuals that had inherited NRXN3 had a 10 to 15 percent increased risk of being obese (26). Gene NRXN3 had previously been found in alcoholics, cocaine addicts, and other substance abusers. Gene NRXN3 is not the only gene linked to obesity, two other genes have been linked to obesity and all three genes are active in encoding brain proteins. The significance of the discovery of genetics linked to obesity is prevention. If doctors know the specific genes that are associated with increased risks of obesity, then genetic testing may be used to warn individuals at risk. Individuals at risk of obesity will then know to monitor diet and increase physical activity to reduce risk of becoming obese (26).

Psychological Issues

As with other eating disorders, biological abnormalities are not the only contributing factor. Psychological disturbances such as depression, dissatisfaction with body image, inability to express emotions, and low self-esteem may contribute to the development of binge eating disorder. A study among 159 clinic patients tested for a correlation among most of these disturbances and binge eating and was published in 1999. The purpose of the research was to find the correlations between binge eating, body image, depression, and self-efficacy in an obese clinical population. All of the participants completed an Eating Habits Questionnaire. Results indicate increased negative body image, higher rates of depression, and lower rates of self-efficacy related to binge eating. One analysis found that body image characterized by shame and concern with public appearance had the highest correlations to binge eating. The researchers

concluded that while this study extended information to the understanding of causes of binge eating, more research needed to be done to find conclusive correlations between binge eating and these contributing factors (27).

Environmental Issues

Of environmental factors that are associated with the risk of developing binge eating disorder, the two most prevalent issues are sexual abuse and inability to express emotions. Research is still being conducted to find the extent to which environmental factors play a role in the risk of developing binge eating disorder. It has been found that increased dieting, body dissatisfaction, pressure to be thin, depressive symptoms, eating disturbances, and low self-esteem and social support predicted the development of binge eating disorder by 92 percent (28). The role that sexual abuse plays in the development of binge eating is still a controversy. Many studies have concluded it plays a large role and that many individuals binge eat and gain weight as a way to make them unattractive to the opposite sex in hopes of not being sexually abused again. One study, however, concluded that depression and weight dissatisfaction was more influential in the development of binge eating then sexual abuse (22).

Complications of Binge Eating Disorder

Binge eating disorder complications are different from those of anorexia nervosa and bulimia nervosa because binge eating is associated with obesity. Medical complications that follow binge eating are: obesity, joint pain, muscle pain, type 2 diabetes mellitus, hypertension, hypercholestremia, and cardiovascular disease (4).

Obesity, Joint, and Muscle

Binge eating is often associated with obesity and therefore health complications that occur with obesity are a concern for binge eating disorder patients. As the individual consumes excessive calories and does not compensate for them, the person quickly gains weight, reaching obese levels. Obesity often causes joint and muscle pain from the excess weight and strain that is put on the frame of the body.

Type 2 Diabetes Mellitus

Type 2 diabetes is a common complication of binge eating disorder. As weight increases, insulin resistance occurs. Insulin resistance is when the body's receptor sites for insulin do not properly bind to the insulin or the insulin binds to the receptor site but it is not functioning properly. Insulin is responsible for the uptake of glucose from the bloodstream into the cells. Cells need glucose for energy; glucose is the primary energy fuel for the body (2). As glucose accumulates in the blood, a condition called hyperglycemia develops. Excess glucose in the blood increases the viscosity of blood; this in turn causes the heart to work harder to pump the blood through the veins. Increased work for the heart also increases the risk of heart disease (common in type 2 diabetes). If excessive glucose accumulates in the blood and not in the cells, the body does not receive fuel. Signs of hyperglycemia are excess thirst, excess urination, and fatigue (2).

Cardiovascular Disease

An individual suffering from binge eating disorder stores excess calories consumed as fat. Excess fat accumulation begins to build up in the blood causing hypercholestremia and atherosclerosis (blockage that can lead to a myocardial infarction). Blockage, hardening, and narrowing of the veins and arteries causes the heart to have to work harder to pump blood

throughout the body, this in turn leads to hypertension. As stated previously, type 2 diabetes also increases the risk for cardiovascular disease.

Treatment

Types of Treatment

Eating disorder treatment ranges in a spectrum from outpatient to day treatment, to residential treatment, to inpatient treatment. Treatment of eating disorders is individualized because each eating disordered patient is very unique. The two primary types of treatment are inpatient and outpatient. Treatment for both anorexia nervosa and bulimia nervosa are very expensive and the treatment may last for several years. It is estimated that inpatient treatment may cost $30,000 or more per month. Outpatient treatment is even more expensive. It is estimated to cost around $100,000 or more, including therapy and medical monitoring (1).

Outpatient treatment is usually the first step used for eating disorder patients. This type of treatment is most effective when a multidisciplinary team work together to help the patient. A multidisciplinary team consists of: a physician, a mental health therapist, a psychiatrist, and a registered dietitian. In outpatient treatment the individual suffering from either anorexia nervosa or bulimia nervosa will meet with the multidisciplinary team weekly (as recovery progresses meetings may be biweekly or even monthly) while still living at home. With a strong multidisciplinary team and family support, it has been shown that for approximately 70 percent of eating disorder patients, outpatient treatment is effective (3).

The second type of treatment is inpatient treatment. Inpatient treatment is preferred for severe eating disorders or for those whom received outpatient treatment without success. The patient suffering from anorexia nervosa or bulimia nervosa lives in a residential center during

inpatient treatment. Duration of stay at an inpatient facility typically lasts 30 to 60 days. At an inpatient treatment facility, the eating disorder patient receives intensive care from a full multidisciplinary team.

Treatment Team

As stated previously, a treatment team consists of a physician, a mental health therapist, a psychiatrist, and a registered dietitian. Each team member has an important role in the team, but must also communicate with each team member to ensure the most effective treatment for the patient. The physician's role is to handle the medical implications and complications that occur with eating disorders, such as distention of the gastrointestinal tract, pancreatitis, liver failure, kidney failure, and many other problems that occur in the body. Often, patients with severe eating disorders will be hospitalized several times from health complications, so the medical doctor is very important in the recovery of the patient. The physician will regulate electrolytes and fluids, increase GI motility, stabilize the heart, and any other medical complications that occur with the patient. A registered dietitian also plays a role in the physical well-being of the patient. The role of the registered dietitian is to educate the patient on the effects the diet has on the body and what starvation will do to the body. The patient and registered dietitian work together to form a meal plan that is easy for the patient to follow and will allow for gradual weight gain or restoration to normal weight. Both the physician and the registered dietitian must continuously educate the patient about the short-term and long-term consequences of the eating disorder.

Since eating disorders are categorized as a mental illness, the mental health therapist plays one of the most important roles in the treatment of eating disorders. The therapist must first build a strong and trusting relationship with the patient. One of the mental health therapist's

roles is to listen to the patient. The therapist helps the patient deal with the real issues behind the eating disorder, because often food is just masking the real issues. The last member of the treatment team is the psychiatrist. The role of the psychiatrist is to prescribe and handle all the medications the patient is taking. An eating disorder patient is often on several medications to treat things such as depression, obsessive compulsive disorder, anxiety, and other medical complications. Each team member and patient must work closely together and be open and honest in order for treatment to be successful.

Conclusion

Eating disorders are very serious psychiatric illnesses. Illnesses have progressed to epidemic levels within the past three decades. Anorexia nervosa has the highest mortality rate of all mental illness, which indicates the severity of taking "dieting" too lightly. The etiology is complex and involves multiple factors such as biological, psychological, and environmental factors. Complications effect many systems in the body and can even be fatal. Treatment must be multi-disciplinary and individualized for each patient. As research is currently being conducted and education is provided for those at risk, hopefully the development of these devastating disorders will decline.

Bibliography

1. ANAD resources page. Anorexia Nervosa and Associated Eating Disorders Web site. http://www.anad.org/getInformation/abouteatingdisorders/. Accessed March 27, 2010.
2. Mahan LK, and Escott-Stump S. *Krause's Food & Nutrition Therapy*. 12th ed. St. Louis, MO: Saunders Elsevier; 2008.
3. Mitchell, JE. *The Outpatient Treatment of Eating Disorders*. Minneapolis, MN: University of Minnesota Press; 1997.
4. Costin C. *The Eating Disorder Sourcebook: A Comprehensive Guide to the Causes, Treatments, and Prevention of Eating Disorders.* Illinois: Lowell House; 1996.
5. Mehler PS and Anderson, AE. *Eating Disorders: A Guide to Medical Care and Complications.* 2nd ed. Maryland: John Hopkins University Press; 2010.
6. Grice DE, Halmi, KA, Fichter MM, et al. Evidence for a Susceptibility Gene for Anorexia Nervosa on Chromosome 1. *AJH*. 2002; 70.3. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC384957/. Accessed March 27, 2010.
7. Devlin B, Bacanu SA, Klump KL, et al. Linkage Analysis of Anorexia Nervosa Incorporation Behavioral Covariates. *HMG*. 2002; 11.6. http://eatingdisorders.ucsd.edu/research/genetics/PDF/devlin2002linkage.pdf. Accessed March 27, 2010.
8. Wade TD, Bulik CM, Neale M, et al. Anorexia Nervosa and Major Depression: Shared Genetic and Environmental Risk Factors. *AJP*. 2000; 157. http://ajp.psychiatryonline.org/cgi/reprint/157/3/469. Accessed March 29, 2010.
9. Woosley MM. *Eating Disorders: A Clinical Guide to Counseling and Treatment*. Illinois: American Dietetic Association; 2002.
10. Walsh TB. Endocrine Disturbances in Anorexia Nervosa and Depression. *PM*. 1982; 44.1. www.psychosomaticmedicine.org/cgi/repreint/44/1/85.pdf. Accessed April 1, 2010.
11. Williams MH. *Nutrition for Health, Fitness, and Sport*. 5th ed. New York: WBC/McGraw-Hill; 1999.
12. Katzman DK. Medical Complications in Adolescents with Anorexia Nervosa: A Review of the Literatre. *Int JED*. 2005; 37. http://onlineacademics.org/CopyrightedAitken/Adolescence/EatingDisorderArticleKatzman/pdf. Accessed April 3, 2010.
13. Hewit PL, Flett GL, Ediger E. Perfectionism Traits and Perfectionism Self-Presentation in Eating Disorder Attitudes, Characteristics, and Symptoms. *Int JED*. 1995; 18.4. http://hewitlab.psych.ubc.ca/pdfs/1995hfe.pdf. March 28, 2010.
14. Beales D and Dolton R. Eating Disordered Patients: Personality, Alexithymia, and Implications for Primary Care. *Brit JGP*. 200; 50. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC13/3605/pdf/10695062.pdf. March 26, 2010.
15. Zonnevyl-Bender MJS, VanGoozen SHM, Cohen-Ketens PT, et al. Emotional Functioning in Adolescent Anorexia Nervosa Patients. *Euro Ch & Adol Psy*. 2004; 13.1. http://www.springerlink.com/content/akvgbwq3klbmp957/. March 26, 2010.
16. Rose D. Anorexia Symptoms Found in Under-13s. *TimesOnline*. Times Newspaper LTD. 2007. http://www.timesonline.co.uk/tol/news/uk/health/article1572684.ece. March 20, 2010.

17. Deep AL, Lilenfeld LR, Plotnicov KH, et al. Sexual Abuse in Eating Disorder Subtypes and Control Women: The Role of Comorbid Substance Dependence in Bulimia Nervosa. *Int JED*. 1999; 25. http://eatingdisorders.ucsd.edu/research/imaging/PDFs/1999/deep1999role.pdf. March 22, 2010.
18. Disordered Eating: Information about Eating Disorders resource page. http://www.disrodered-eating.co.uk/eating-disorders-statistics/anorexia-nervosa-statistics-uk.html. March 23, 2010.
19. Collier DA and Treasure JL. The Etiology of Eating Disorder. *Brit Jn Psy.* 2004; 185. http://bjp.rcpysch.org/cgi/content/full/185/5/363. March 23, 2010.
20. Bulik CM, Devlin B, Bacanu SA, et al. Significant Linkage on Chromosome 10p in Families with Bulimia Nervosa. *AJHG.* 2003; 72.1. http://www.ncbi.nlm.nig.gov/pubmed/12476400. March 23, 2010.
21. Kendler KS, MacLean C, Neale M, et al. The Genetic Epidemiology of Bulimia Nervosa. *AJP.* 1991; 148. http://ajp.psychaitryonline.org/cgi/content/abstract/148/12/1627. March 24, 2010.
22. Newton JR, Freeman CP, Munro J. Impulsivity and Dyscontrol in Bulimia Nervsoa: Is Impulsivity an Independent Phenomenon or a Marker of Severity? *Acta Psy Scan.* 1993; 87.6. http://www.ncbi.nlm.nih.gov/pubmed/8356889. March 25, 2010.
23. Mehler PS and Anderson AE. *Eating Disorders: A Guide to Medical Care and Complications*. 1st ed. Maryland: John Hopkins University Press, 1999.
24. Rose T. Linking Addiction to Food to Low Serotonin. *Bra Chem & Fo Add.* 2006. http://weightloss.suite101.com/article/cfm/brain_chemistry_and_food_addiction. March 25, 2010.
25. Birketvedt GS, Sundsfjord J, Florholmon JR. Hypothalamic-Pituitary-Adrenal Axis in the Night Eating Syndrome. *AJP.* 2002; 282.2. http://ajpendo.physiology.org/cgi/content/full/282/2/E366. March 23, 2010.
26. Eating Disorder Resources. Obesity Linked to Addiction Gene in Brain. http://www.eating-disorder-resources.com/eating-disorder-articles/food-addiction/obesity-linked-to-addiction-gene-in-brain/. March 29, 2010.
27. Cargill BR, Clark MM, Pera V, et al. Binge Eating, Body Image, Depression, and Self-Efficacy in an Obese Clinical Population. *Obe Res.* 1999; 7.4. http://www.ncbi.nlm.nih.gov/sites/entrez/10440594?dopt=Abstract&holding=f1000,f1000m,isrctn. March 25, 2010.
28. Stice E, Presnell K, Spangler D. Risk Factors for Binge Eating Onset in Adolescent Girls: A 2-Year Prospective Investigation. *Hea Psy.* 2002; 21.2. http://psychnet.apa.org/journals/hea/21/2/131. March 25, 2010.