Eating Disorders: Anorexia Nervosa

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Description

SYMPTOMS AND DIAGNOSTIC CRITERIA

Eating disorders are characterized by pathological eating behaviors combined with abnormal thoughts and beliefs about food and weight, including overvaluation of thinness, distorted body image, or obsessively ruminating about food or weight (Fairburn & Harrison, 2003). The DSM-IV has three eating disorder diagnoses: Anorexia Nervosa (AN), Bulimia Nervosa (BN), and Eating Disorder Not Otherwise Specified (EDNOS) (American Psychiatric Association, 1994). Of these, AN is the oldest type of eating disorder described in the medical literature and arguably the most serious of them (Gull, 1874; Lasègue, 1883). The DSM-IV describes four main criteria for diagnosing AN: refusal to maintain body weight in a normal range, an irrational fear of fat or of gaining weight in the context of low weight, body image distortion or denial of the medical seriousness of extreme low weight, and in postmenarcheal females, three consecutive months of amenorrhea. There is a purely restrictive subtype and a binge/purge subtype of AN described in DSM-IV (APA, 1994).

The core features of AN include behavioral, psychological, and physiological factors. There is a clear and unwavering behavioral refusal to obtain or maintain weight acceptable for height and health. The behaviors that illustrate this refusal include a highly restrictive and selective eating pattern, excessive exercise, and in some cases purging behaviors (e.g., vomiting and laxative use). Often these behaviors appear initially innocuous, even consistent with age appropriate developmental concerns about appearance; however, over time, these behaviors take on an urgent and insistent nature while other important social and emotional pursuits are deferred or avoided all together. Hours may
be spent calculating calories and planning meals. Exercise deviates from normal activity levels and it becomes a compulsive activity where attempts to interrupt the behavior are met with extreme resistance. Purging behaviors develop in 15 to 20% of cases as an attempt to limit the potential for weight gain even in the context of extremely limited intake. Over time the experiences of starvation, exercise, and even purging behaviors become self-reinforcing and are experienced as a source of support and comfort for the person with AN.

The psychological basis for these behaviors is usually considered to be an intense fear of fat or of gaining weight. The triggers for the development of this specific type of anxiety and fear about fat and weight gain are extremely variable but commonly include the onset of puberty, development of an extreme health or nutrition focus, being teased about weight or appearance, initiating social or academic transitions (e.g. starting middle school or high school), emergence of a medical illness that initiates weight loss, attempting to improve athletic performance, and experiences of physical and sexual abuse. Whatever the specific trigger may be, the development of this extreme anxiety and fear is the psychological underpinning that putatively supports the behaviors that lead to extreme weight loss. The fear of weight gain and fat is relieved—albeit temporarily—by the various weight loss inducing behaviors. This is accomplished directly because the behaviors lead to weight loss and that reduces the anxiety and fear; and indirectly, by providing a focus for the obsessive worry that accompanies these fears.

Another psychological aspect of AN is the disturbance in the appreciation of current body weight and shape, leading to overestimating body size relative to true body size. This body-image distortion likely results from a consistent and persistent overfocus on the body as a whole or on specific parts of the body (e.g. thighs, buttocks, cheeks) in an attempt to assess the success of efforts to lose weight or fat. Initially, this focus may be a source of limited reassurance, much in the way that constant checking of weight on a scale may be, because changes can be seen and measured. However, over time the hyper focus on the body leads to greater and greater distortions and misperceptions. This can also lead to a severe distortion in evaluating the medical consequences of being severely underweight. This denial of the seriousness of malnutrition is a major source of treatment avoidance and represents a significant psychological hazard for both psychological and medical treatment.

The result of the interaction between the psychological factors of fear, distortion, and denial and the behaviors precipitating extreme weight loss is physiological malnutrition. A commonly used figure for determining malnutrition in adults is a BMI of 17.5 or below, or an Ideal Body Weight (IBW) of 85% or below. However, in children and adolescents these figures can be misleading and growth and pubertal status greatly affect these types of norms. Malnutrition affects most organ systems, as the body responds to a starvation state and conserves energy by cutting back on all but the most essential functions. Blood flow to the periphery is decreased, leading to cold extremities. Skin becomes dry and hair falls out, while the body becomes coated with lanugo, a fine downy hair meant to conserve warmth. Menstruation stops or becomes irregular, and fertility is impaired. Along with these hormonal changes, calcium is lost from the bones leading to osteopenia, or in severe cases to osteoporosis. In children who are still growing, growth can be slowed or stopped. Heart and brain function are preserved for as long as possible but eventually the cardiac muscle weakens, leading to low heart rate, low blood pressure, and possible death. Brain scans have shown shrinkage of the brain during the illness (Katzman, Christensen, Young, & Zipursky, 2001).
Of all these complications, the only one known to definitely persist after weight restoration is osteoporosis—the severity of which depends on the duration of the illness.

The *DSM-IV* attempts to describe discrete eating disorder syndromes, but given the paucity of knowledge about eating disorders it has been less successful with AN than with many other disorders (Fairburn & Bohn, 2005). There is a significantly higher incidence of Eating Disorder Not Otherwise Specified (EDNOS) than of AN or BN—while many patients with EDNOS are just as compromised as patients with AN or BN, but do not meet full criteria (Fairburn & Bohn, 2005). Several of the current criteria for AN are problematic and exclude patients who have the same types of thoughts and behaviors as patients who meet criteria for AN.

Some authors have contested the current suggested weight criterion of 85% or below of ideal body weight (Herzog & Delinsky, 2001). It is extremely difficult to be accurate when calculating IBW to take into account body frame, pubertal status, and ethnicity, and thus difficult to determine when a patient is below 85% of his or her individual ideal body weight. Determination of ideal body weight is even more complicated in younger patients who may be growth retarded due to malnutrition: Should their current ideal body weight be calculated based on current height, or the height they would have been if they had continued their previous growth curve? This criterion excludes patients who start off overweight and lose to a normal weight, who may have just as severe thoughts, behaviors, and medical consequences. Finally, the cut point of 85% or BMI of 17.5 is arbitrary and has varied considerably in various iterations of the *DSM* over time. Even in the current version, these weight cut-points are given as examples, not absolutes, though many clinicians and researchers appear to treat them as such.

Lee (1995) objected to the requirement for weight phobia on cultural grounds. Patients diagnosed with AN in non-Western countries are less likely to report fear of fatness as a motivation for their weight loss, as are younger adolescents and preadolescents (Nicholls & Bryant-Waugh, 2003). Clinically, patients without weight phobia—but who otherwise meet criteria for AN—do not seem to be significantly different from patients who do present with weight phobia, although Strober, Freeman, and Morrell (1997, 1999) in a 10–15 year follow up study—found patients without weight phobia to be less likely to have a chronic course or engage in binge eating, and more likely to be fully weight recovered. Amenorrhea is also a problematic criterion. Amenorrhea may occur before weight loss, or may not occur in patients with severe weight loss. Studies suggest no difference in patients with and without amenorrhea in terms of severity of illness, body image disturbance, depression, or personality disorders (Cachelin & Maher, 1998; Garfinkel et al., 1996). This criterion does not apply to males or to premenarcheal girls, and can be confusing in young adolescent girls who normally have irregular periods or with girls on oral steroids for birth control.

As noted above, the *DSM-IV* gives two subtypes of AN—restricting and binge/purge. The evidence supporting these subtypes as clinically discrete entities is controversial. There is some evidence that patients with the binge-purge subtype have a more complicated course (Herzog & Delinsky, 2001; Herzog et al., 1999; Pryor, Wiedermann, & McGilley, 1996) and other studies have found more depression, substance abuse, and personality disorders in binge-purge patients (Dacosta & Halmi, 1992; Herzog et al., 1996). Other studies have found no difference in recovery rates or psychopathology (Eddy et al., 2002). Many patients appear to cross back and forth between the two subtypes as well as between AN, BN, and EDNOS over the course of their lives. Thus there remains considerable controversy about the validity of the *DSM* diagnostic scheme.
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insofar as it relates to eating disorders and to AN specifically, especially for younger patients with these disorders (Nicholls, Chater, & Lask, 2000). Development of reliable ways to differentiate early stage AN from nonprobable, genetically-based “thinness,” and normative dieting and body discontent remains a challenge, but are needed as intervention early in the disorder is more successful and prevents medical complications.

Case Example

Lisa is a 17-year-old female who reports “according to other people I’m not eating safely.” About six months ago she decided to “lose a few pounds” for her junior prom. She is 5'5”, and initially wanted to decrease her weight from 130lbs to 120lbs. She initiated her dieting by eating what she described as “healthier”: she cut out junk food, desserts, and fast food, and limited her portion sizes. She also started an exercise routine of running three miles and doing 100 sit-ups and 100 push-ups every day. Her weight dropped to 120lbs by prom, but she still felt overweight. She cut more and more foods from her diet until she was eating mostly fruits and vegetables and was skipping lunch at school. She kept careful track of calories and tried not to eat more than 500 kcal/day. Her parents noticed that she was losing too much weight and tried to keep her from exercising, but Lisa felt too guilty about “all those calories just sitting there” and was afraid of gaining weight. She started secretly doing jumping jacks and running in place in her room at night. As her weight decreased further she became more and more irritable, especially when her parents tried to encourage her to eat. Her menses ceased, she felt cold all the time, and her hair started falling out. She stopped spending time with friends because she was worried they would comment on her not eating, and because she just did not have the energy to go out. Her parents became concerned enough to bring her to the pediatrician, who found that her weight was down to 100 lbs and her heart rate was 42 beats per minute. She was admitted to the hospital, which Lisa thought was completely unnecessary. She was furious at being made to gain weight in the hospital as she felt her thighs were still “huge.”

History of Anorexia Nervosa

Although there are reports of disturbed eating patterns and food refusal throughout history, the association with disturbed body image and fear of weight gain did not arise until the late nineteenth century (Saraf, 1998). In 1873 the French physician Charles Lasègue and the British physician Sir William Gull independently described an illness, which Lasègue dubbed “anorexia hystérique” and Gull called “anorexia hysterica,” primarily affecting girls and young women and consisting of severe weight loss, amenorrhea, constipation, and restlessness, without evidence of any organic pathology (Gull, 1874; Lasègue, 1883). Gull later called this illness Anorexia Nervosa in a lecture the next year, but it did not receive much attention. In 1914 a German pathologist named Morris Simmonds found lesions in the pituitaries of some emaciated patients (Simmonds, 1914). AN was theorized to have an endocrine etiology and was called “Simmond’s disease” or “pituitary cachexia.” This notion was refuted after World War II. Psychoanalytic thinking was popular at this time and AN was thought of as a fear of oral impregnation (Thoma, 1967). AN did not receive widespread attention until the work of the psychiatrist Hilde Bruch, who published her pioneering work in the 1970s (Bruch, 1973, 1978). Her formulation emphasized the issues of core low self-esteem, limited self-esteem, familial role, to intrusiveness adolescent, vini Palazzi voped to (Beck, Ru: for AN has: 1987); how a related e: well (Fairl & Bauer, 2 adherents,

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limited self-concept, and distorted body image that she saw in AN patients. Arthur Crisp’s formulation of AN as an effort to avoid the difficulties, physical, emotional, and familial problems related to the onset of adolescence, offers a related psychological formulation to that of Bruch (Crisp, 1997). Theories about family psychopathology related to intrusiveness and over control as contributors to the psychological vulnerability in adolescents with AN were advanced by Minuchin, Rosman, and Baker (1978), and Selvini Palazzoli (1974). At the same time, cognitive behavioral therapies were being de-
volved to address other mental health problems, particularly depression and anxiety (Beck, Rush, Shaw, & Emery, 1979). The application of purely behavioral treatments for AN has been limited and largely confined to inpatient treatment programs (Jenkins, 1987); however, the success of cognitive-behavioral therapy (CBT) for bulimia nervosa, a related eating disorder, has lead to preliminary examination of this approach for AN as well (Fairburn, 1981; Fairburn, Cooper, & Safran, 2002; Pike, Walsh, Vitousek, Wilson, & Bauer, 2004). To date, although each of these proposed psychological theories has its adherents, little substantive research supports any of them.

Epidemiology

Estimates of the incidence and prevalence of AN vary depending on the population studied (community versus clinical samples), but most estimates for point prevalence range from 0.1 to 0.9% of the population, while rates of subthreshold AN (meeting all but one of the DSM-IV criteria) are higher (Hock & Hocken, 2003; Hock et al., 2005). Approximately 5 to 10% of patients with AN are male, although the true incidence in males may be higher as males are less likely to come to clinical attention. The peak age of onset in females may be bimodal at 14 and 18 years of age, but onset of the disorder is seen as young as seven years old and throughout adulthood (Halmi, Brodland, & Loney, 1973). Contrary to previous beliefs, the disorder is prevalent across ethnicities and socioeconomic status. Nevertheless, the prevalence is higher in industrialized Westernized countries, and increases in other countries as they become more industrialized and exposed to Western media. There is controversy over whether the incidence of AN is increasing. Many studies show an increased incidence over the past 50 years, but this may be due to an increase in detection or to a relative increase in the most vulnerable population (i.e., young women). A study by Lucas, Beard, and O’Fallon (1991) that looked at the population of Rochester, Minnesota, over the course of fifty years found that while the incidence in women over 24 years old remained constant, the incidence in the 15 to 24 group went from 16.6/100,000 in 1935–1939, to 7 in 1950–1954, but then rose to 26.3 in 1980–1984 (Lucas, Crowson, O’Fallon, & Melton, 1999). More recent data suggest that this increased incidence in younger age groups continues to the present (van Son et al., 2006).

Dysfunction

NEUROBIOLOGY

The study of the neurobiology of eating disorders is still relatively unexplored. The current avenues of exploration are the investigation of neurotransmitters and appetite related peptides, and the use of neuroimaging techniques to examine the possibility of structural and functional brain contributions to AN.
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Serotonin has been the most popular neurotransmitter targeted for investigation so far, as 5-HT systems are involved in mood and obsessiveness, appetite regulation, and impulse control. Patients with AN have been found to have low levels of 5-HT metabolites in the cerebrospinal fluid and abnormal hormonal response to 5-HT specific challenges. However, specific abnormalities in the serotonin system have not been identified. More recently, interest in the dopaminergic neurotransmitter system has developed because of the role of this neurotransmitter in reward systems and obsessive compulsive thinking and behavior. Some suggest that hypersensitivity of the dopaminergic system may account for the extreme reactions of patients with AN to novel stimuli (Kaye, Strober, & Jimerson, in press).

Recent studies suggest a neural disturbance in eating disorders (Trummer, Eustacchio, Unger, Tillich, & Flaschka, 2002; Uher et al., 2004; Ward, Tiller, Treasure, & Russell, 2000). Indirect evidence in support of a neural basis of eating disorders can be inferred from neuropsychological, electrophysiological, neuropharmacological, structural neuroimaging, and functional neuroimaging investigations (Uher, Treasure, & Campbell, 2002). Although there is a fairly substantial brain imaging literature in AN—including positron emission tomography (PET) and single-photon emission computed tomography (SPECT) studies that show regional differences in metabolism and neurotransmitter (usually receptor) alternations, and fMRI studies demonstrating differences in activation response to stimuli (usually food)—these studies are only on adults and are generally small scale and variable in terms of patient subtypes, state of illness, and regional neuroanatomy examined (Frank et al., 2002; Kaye, Strober, & Jimerson, in press; 1998; Kaye, Grendall, & Strober, 1998; Kaye, Gwirtsman, George, & Ebert, 1991; Uher et al., 2004).

Neuroimaging generally reveals decreased brain mass and enlarged sulci in patients in the acute malnourished phase of the disorder. This is thought to be an effect either directly of malnutrition or of the effect of increased cortisol on brain tissue, and seems to resolve with weight restoration. There is some question whether the grey matter mass returns completely to normal, as some studies have found continued decreased grey matter after recovery (Katzman, Kapstein, & Kirsh, 1997; Salzer, Bickman, & Lambert, 1999). SPECT studies have shown unilateral hypoperfusion in specific areas of most patients with AN—most commonly in the temporal lobe—that seems to persist after recovery. Rastam, Bjure, and Vestergren (2001) found decreased blood flow to the temporal lobe in 14 of 21 patients seven years after recovery, with no correlation between the blood flow and BMI or IQ. Chowdhury, Gordon, and Lask (2003) found unilateral hypoperfusion correlated with eating disorder psychopathology and not with BMI. Lask et al. (2006) found decreased temporal blood flow to correlate with eating disorder psychopathology and impaired executive functioning, and no correlation between blood flow and BMI, mood, or length of illness. On the three year follow up these investigators found persistent hypoperfusion in 86% associated with persistent eating disorder psychopathology and persistent cognitive impairment (visuospatial processing and memory, and cognitive inhibition) (Lask, 2006). These authors hypothesize that this unilateral hypoperfusion represents a preexisting deficit—likely either genetic or due to perinatal insult—that predisposes patients to develop AN.

Further, there are increasing data supportive of a variety of neurocognitive alterations in AN and BN subjects (Godley, Tchanturia, MacLeod, & Schmidt, 2001; Tchanturia et al., 2004; Tchanturia, Morris, Breccia, Nikolau, & Treasure, in press; Tchanturia, Morris, Breccia, Nikolau, & Treasure, in press).

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Morris, Surguladze, & Treasure, 2002; Tchanturia, Serpell, Troop, & Treasure, 2001). These include problems with attention, executive functioning, divided attention, working memory, response inhibition, and mental inflexibility. These variables, among others, are likely not only to play a role on the etiology and maintenance of AN symptoms, but also to have an impact on treatment response. Attention problems would make it difficult to participate in most psychological treatments, problems in working memory would likely inhibit insight-oriented therapies and cognitive therapies, and mental inflexibility might increase resistance to new ideas and behaviors proposed across a variety of treatments.

Studies also suggest a biological basis in genetics for AN, though the mechanism for this is unclear. Family aggregate studies suggest that AN is familial (Lilenfeld et al., 1998; Strober, Freeman, Lampert, Diamond, & Kaye, 2000) and more recent studies suggest that genetic contributions appear to account for more than 50% of the heritable risk for developing AN (Bulik, 2004; Bulik et al., 2006). The specific genetic mechanism for increasing heritability risk is still unknown but may relate to inherited vulnerabilities related to temperament and anxiety (Wagner et al., 2006).

Behavioral

There are a number of psychosocial factors related to the emergence, maintenance, or recovery from AN that complicate the course. AN often starts with dieting or exercising to lose just a little weight; very few patients start saying “I want to become anorexic.” Anorexia Nervosa may start after an identifiable trigger such as being called fat by a classmate or sibling, or a loss such as death of a grandparent or parental divorce. The patient may want to lose weight for a specific occasion, such as a spring break trip or a Quinceanera. The patient may want to improve athletic performance. Over time the weight loss becomes an end in and of itself and spirals out of the patient’s control. Many patients firmly believe the behavior is within their control until they try to stop, at which point they sometimes realize that their symptoms actually control them.

The stereotype of the person with AN is someone who does not eat at all, but this is usually not the case. Most people with AN restrict their intake in some form: by eating smaller portions, skipping meals, or avoiding foods they believe will make them gain weight or are unhealthy. Converting to vegetarianism in the context of dieting can be an early sign of AN. Only certain foods will be safe to consume and this group typically becomes smaller over time until often a patient eats the exact same thing every day. Some patients drink excessive amounts of water to control hunger, while others will consume very little water because it appears to increase their weight or makes them feel bloated. They may weigh themselves frequently and may feel the day is ruined if their weight has not decreased. Exercise is another common method of weight loss. Exercise may initially be fairly normal in quantity and quality, but often becomes obsessive. Some patients feel so compelled to exercise they will not miss a day despite illness, injury, or other engagements. The patient may feel he or she must exercise in order to be allowed to eat, or may exercise after meals to burn off the calories consumed. In some cases this is called exercise anorexia and the individual exercises at such a high level (training for a marathon or participation in intensive sports) that their caloric intake may be in the normal range. However, such individuals refuse to eat “unhealthy” foods and to increase calories sufficiently to achieve or maintain a healthy weight.
Patients with the binge-purge type of AN usually restrict and/or over-exercise, but they also engage in other, more harmful weight loss behaviors. They may use laxatives, diuretics, or enemas that lead to dehydration rather than true weight loss; they may abuse medications, notably diazepamics on insulin. However, even the feeling of decreased weight provides temporary reassurance about the fear of weight gain. They may also vomit after eating to avoid absorbing calories, although a large portion of the calories are actually absorbed before the patient is able to vomit them up. Some patients also binge eat, but for many these are subjective binge episodes where the patient feels he or she has over eaten and feels out of control after consuming only a small amount of foods, particularly those considered “forbidden.”

Patients with AN are usually extremely preoccupied with food and food preparation, a symptom that is seen with any form of starvation (Franklin, Schiele, Brozek, & Keys, 1948). They may spend hours a day tallying up calories or planning what to eat at the next meal. Some will become very interested in cooking and will cook elaborate meals for others, but will not eat the meal themselves. Others become critical of family members’ “fatty” and “unhealthy” diets. This preoccupation can make it difficult to concentrate on anything else. For example, some patients report they find it difficult to pay attention in school because they are too busy planning the next day’s meals and analyzing the nutritional content.

Cognitive

There are a variety of cognitive problems associated with AN, the most common of which is body image distortion. When body image distortion is present, it can be near-delusional. Some patients may recognize their overall thinness but still believe a particular body part is grossly overweight, while others see themselves as fat all over. Being told by everyone around him or her that they are actually very underweight does little to shake this belief. Patients may have other fixed beliefs such as how food is processed in the body, which also do not yield despite education and reassurance from dieticians or other knowledgeable individuals. Patients with AN typically place a very high value on thinness, making it a large part of how they define their self-worth. Some state “I’d rather be dead than fat.” Hours will be spent in front of the mirror looking for any change in weight, or trying on clothes to see if they still fit. Being overweight—or even of normal weight—is equated with being ugly, disgusting, or out of control.

Denial and deception are very common features of patients with AN. Early in the disorder most patients do not want to recover, and even later on with increased insight they likely will have mixed feelings about recovery (Couturier & Lock, 2006a). Many state, “I want to get better, but I don’t want to gain weight.” They can be very deceptive about how much they eat and exercise and whether they vomit. This is very distressing for parents, spouses, and clinicians trying to help them, as in most instances these individuals have been particularly honest and trustworthy people and remain so in other areas of their lives not related to eating and weight. Patients employ a variety of strategies to hide how little they are eating, claiming they have already eaten with friends or at school, hiding food while at the table, discreetly disposing of food, and spreading food over the plate to make it look like more has been eaten. They may vomit while in the shower, or have the faucet running in the bathroom to cover up the noise of vomiting. Early in the disorder many patients deny trying to lose weight. They may say they are just trying to pre-adolescent for eating I (Couturier their confut (Nicholls & Perfectio 2000). Patiente, intelligen, but result of th Perfection lower sens another, loy of ineffecti strained ter study by W and low se one year o results of cu experent (Emotion. Anxiety or Jeannet, ‘ 1992). Soe because as more obser energy to food, may find that s Thus, sym the onset c there is ev OCD, gen Nussbaum history of (Herzog, N acute sym disorders (sen, 2002) Interact The impac and long-t to phalamic h
are just trying to be healthier or appear mystified by their weight loss. Early teens and preadolescents commonly give nausea, abdominal pain, or just feeling full as reasons for eating less, and the weight phobia is only admitted to the course of treatment (Couturier & Lock, 2006a). These younger patients may not be completely feigning their confusion: they may not be completely conscious of what they are doing and why (Nicholls & Bryant-Waugh, 2003).

Perfectionism is another common characteristic of patients with AN (Halmi et al., 2000). Patients with AN tend to do very well in school, not because they are more intelligent, but because they work harder than others (Bryant-Waugh & Lask, 1995). As a result of their drive and perfectionism, they often see things in all-or-nothing terms. Perfection is an impossible goal and the pursuit of it leads to lower self-esteem and a lower sense of self-efficacy (Forsberg & Lock, 2006). In the context of AN, as the patient reaches each goal weight they feel they could still lose more so they set yet another, lower, goal weight. Other common personality traits are obsessiveness, a sense of ineffectiveness, rigidity, and harm-avoidance (Klump et al., 2000). A fearful, constrained temperament may be an inherited risk factor for Anorexia Nervosa. A recent study by Wagner et al. (2006) found persistent high harm avoidance, high persistence, and low self-directedness in patients with a history of AN or BN that was still evident one year or greater after recovery, indicating that these characteristics are not just results of current malnutrition, and may be premorbid risk factors associated with temperament (Wagner et al., 2006).

EMOTIONAL

Anxiety and depression are common features in AN (Godart, Flament, Perdereau, & Jeannot, 2002; Herzog, Nussbaum, & Marmor, 1996; Klump et al., 2000; Rastam, 1992). Some symptoms of anxiety and depression are a direct effect of malnutrition because as people lose weight they have less energy, are more irritable, and become more obsessive. Social withdrawal and isolation are common as the patient has less energy to go out with friends. Patients may also want to avoid social situations with food, may worry about friends commenting on their weight and eating habits, or may find that socializing interferes too much with their rigid eating and exercise routines. Thus, symptoms of depression and obsessive-compulsive disorder (OCD) that start after the onset of an eating disorder sometimes resolve with improved nutrition. However, there is evidence of increased incidence of a history of anxiety disorders—especially OCD, generalized anxiety, and social phobia in patients who develop AN (Herzog, Nussbaum, et al., 1996). Depression is less likely to predate the AN, but patients with a history of AN have a higher incidence of depression than in the general population (Herzog, Nussbaum, et al., 1996). In follow-up studies, even after recovery from the acute symptoms of AN, a significant minority continue to have anxiety and depression disorders (Herzog, Nussbaum, et al., 1996; Lock, Couturier, & Agras, 2006; Steinhausen, 2002).

INTERACTION WITH EXTERNAL AND ENVIRONMENTAL FACTORS

The impact of severe malnutrition on overall physical health is considerable. The short and long-term medical complications of AN include changes in growth hormone, hypothalamic hypogonadism, bone marrow hypoplasia, structural abnormalities of the brain,
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cardiac dysfunction, and gastrointestinal difficulties (Fisher et al., 1995; Rome & Ammerman, 2003). The more important medical problems for adolescents are the potential for growth retardation, pubertal delay or interruption, and peak bone mass reduction (Fisher et al., 1995). Vomiting, laxatives, diuretics, and enemas can all lead to electrolyte imbalances in the blood as well as dehydration; vomiting can also cause bleeding in the stomach or esophagus, and over time causes erosions in the tooth enamel. The mortality rates associated with AN are higher than for any other psychiatric disorder (Herzog et al., 2000). Follow-up studies of varying lengths suggest that the aggregate mortality rate is approximately 5.6% per decade (Sullivan, 1995). These findings were confirmed in a more recent 11-year follow-up study with a crude mortality rate of 5.1% (Herzog et al., 2000). The standardized mortality ratios for death and suicide in this study were substantially higher than those expected in the general population. Overall, about half the deaths were due to suicide and the remainder due to the physical complications of AN.

Sociocultural factors likely play an important factor in triggering behaviors leading to AN. Social pressures to be thin, especially in females, are considerable (Anderson-Pye & Becker, 2004; Levine & Harrison, 2004). Various media presentations of ideal beauty continue to glamorize an overly thin ideal. Silverstein, Peterson, and Perdue 1986 found that rates for AN are highest immediately after periods when the beauty ideal for women is the thinnest. Cultures that value plumpness tend to have lower rates of AN, and non-Western countries tend to have increased rates of AN after being exposed to Western media. For example, a study of ethnic Fijian adolescent girls found an increased incidence of eating disorders following novel exposure to the Western aesthetic ideal via television after it was first introduced in the mid-1990s (Becker, Burnwell, Herzog, Hamberg, & Gilman, 2002). In general, males with AN have very much the same presentation as females, although they may come to clinical attention later in the disorder because of a lower clinical index of suspicion for these problems in males. Some males are more likely to be concerned with having a low percent body fat or being very muscular, rather than low weight per se (Carlat, Camargo, & Herzog, 1997; Pope, Gruber, Choi, Olivardia, & Phillips, 1997). Although there is still less cultural pressure on males to diet in order to accomplish the thin ideal, over the eighties and nineties the male ideal has become more and more muscular and aesthetic (Leit, Pope, & Gray, 2001; Pope, Olivardia, Gruber, & Borowiecki, 1999). Male action figures have become extremely muscular and are now just as unrealistic for a male body type as the Barbie doll is for a female body type (Pope, Olivardia, Gruber, & Borowiecki, 1999). Despite the similarities between boys and girls with AN at presentation, some data suggest that males with AN have a generally better prognosis than their female counterparts (Strober et al., 2006).

The overvaluation of this thin ideal leads to destructive weight loss behaviors in many people, but only in a relatively few instances do these concerns lead to the extreme weight loss associated with AN. Thus, it is likely an interaction between the overvaluation of the ideal of thinness with personality characteristics such as perfectionism, obsessiveness, and suppression of emotions that increases the risk that sociocultural factors will lead to AN. Parents often describe their children who develop AN as having been perfect before the onset of the disorder (i.e., compliant, high-achieving, and never having had any problems). Difficulty expressing negative feelings is thought to be a factor in the disorder; controlling one's own body is felt to be more manageable than expressing negative emotions and coping with interpersonal conflicts. Low self-esteem is another on weight enough the AN eme cognitive, the develop separation related to the extrem- forestalls ti

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While t minated c lished res whether fical probl Given the will be ut more on tiability (Dare & l presumed developm
is another aspect of these difficulties: negative feelings about oneself become focused on weight and appearance, giving the false hope that if only one’s weight were low enough the patient would feel better about him or herself.

AN emerges in some children as a reaction to puberty, with its dramatic physical, cognitive, and social changes. Crisp (1997) formulates AN specifically as a flight from the developmental challenges of adolescence related to changes in body composition, separation from parents, taking up social and sexualized gender roles, and other issues related to autonomy. The behaviors of AN effectively stifle many of these efforts and the extreme weight loss returns the body to a preadolescent state, or in younger patients forestalls that physical development altogether.

As is the case with many psychiatric disorders, major psychosocial stressors can trigger AN. The stressor can be a loss, such as the death of a much-loved grandparent or parental divorce, or a major change such as a move or starting high school or college. A history of sexual or physical abuse can be a risk factor (Neumark-Sztainer, Story, Hannan, Beuhring, & Resnick, 2000). However, triggers that initiate AN can also be more minor events, such as being teased and called fat by a sibling or peer, or being told by a well-intentioned and obesity-conscious pediatrician to lose weight. Although much emphasis is often placed on uncovering these triggers in psychotherapy for AN, their specific relationship to AN and its resolution remains unclear.

Familial attitudes about food, dieting, and appearance may affect the development of AN (Woodside et al., 2002). Families who place a high emphasis on thinness and appearance, or who view dieting as normal, can instill these values into their children perhaps putting them at higher risk for AN. Early work by Minuchin, Rosman, and Baker 1978 hypothesized that “psychosomatic families” contribute to the maintenance of the behaviors related to AN. They described these families as having rigid organization and diffuse boundaries, as being emotionally enmeshed with one another, and as avoiding expressing conflict whenever possible. The authors proposed that this constellation of behaviors made supporting individuation during the adolescent period extremely challenging and provided the family milieu for developing AN. Others suggested that AN may develop and be maintained as a way to divert attention from other, deeper difficulties within the family, such as marital tension (Selvini Palazzoli & Viaro, 1988). Bruch also believed that family dysfunction played an important role in the etiology of AN (Bruch, 1973). She described an “anorexigenic” mother who is poorly attuned to her infant’s needs and feeds the child on her own schedule, not according to the infant’s hunger. The infant learns not to trust her own ability to distinguish her inner states and develops a sense of ineffectiveness.

While these ideas about family dysfunction may be credible theories and clearly dominated clinical work for many years, there is only limited support for them in published research at this point (Strober & Humphrey, 1987). It is difficult to determine whether family problems are a result of coping with a severe behavioral and psychological problem such as AN, or whether these problems antedated the onset of the disorder. Given the low base rates of AN, it is unlikely that large prospective studies of families will be undertaken to clarify this question, but recent treatments that have focused more on utilizing the strengths of families—rather than pointing out their presumed liabilities—have resulted in good response rates, at least among adolescents with AN (Dare & Eisler, 1997). These results lend credence to the notion that in many cases presumed to be caused by family psychopathology that factor may not be central to the development or maintenance of AN.
Assessment

There are a variety of structured interviews and assessments available for eating disorders and AN (Alison, 1995). The Eating Disorders Examination (EDE) is a semi-structured interview lasting about one hour and is the most commonly used measure in eating disorder treatment studies (Cooper, Cooper, & Fairburn, 1989; Cooper & Fairburn, 1987). The investigator is required to ask certain key questions about eating disorder behaviors and cognitions, but is also allowed to ask additional questions to clarify a response. It is considered the gold standard in assessment of psychopathology in eating disorders; however, it takes about an hour to administer and the interviewer must have gone through thorough training to achieve adequate reliability. The EDE gives categorical data for diagnosis based on the DSM-IV, and continuous data for four subscales (restraint, eating concern, shape concern, and weight concern). It has two behavioral indexes (overeating and methods of extreme weight control). The EDE focuses mostly on the previous four weeks, although some questions go back three or six months as this is required to determine clinical diagnoses. It has been shown to have good reliability and good discriminant and concurrent validity. A child version (CheDEE) is available and has been found to be both reliable and valid for children ages 8–14 (Bryson-Waugh, Cooper, Taylor, & Lask, 1996). In addition, the EDE-Q is a self-report from of the EDE that is also useful because it reduces subject and assessor burden and because it can be completed in less than 15 minutes. Reports suggest that it is a reliable measure for symptom change in adolescents with eating disorders (Passi, Bryson, & Lock, 2003; Binford, Le Grange, & Jellinek, 2005). Although the Schedule for Affective Disorders and Schizophrenia for School-Aged Children (6-18 years; K-SADS) is a widely used interview for detecting psychiatric disorders in children and adolescents and includes diagnosis specific impairment ratings and generates DSM-IV diagnoses, it does not provide sufficiently detailed evaluation of eating disorder related thoughts and behaviors needed to assess treatment response, hence the EDE is the preferred treatment outcome measure (Kaufman et al., 1997). Multiple self-report measures to assess eating disorders are available. Two of the most popular are the Eating Attitudes Test (EAT) and the Eating Disorders Inventory (EDI). The EAT consists of 40 items that employ a 6-point Likert rating scale (Garner & Garfinkel, 1979). The EAT-26 takes 26 of the 40 items which tend to account for most of the variance in total score, and is very highly correlated with the EAT (Garner, Olmsted, Bohr, & Garfinkel, 1982). It is easy to administer and takes less than 10 minutes. The EAT has good internal consistency and test-retest reliability (Carter & Moss, 1984), and has good concurrent and discriminant validity with other eating disorders measures (Garner, 1997). The EAT consists of seven factors: food preoccupation, body image for thinness, vomiting and laxative abuse, dieting, slow eating, clandestine eating, and perceived social pressure to gain weight. The EDI is a 64-item measure designed to assess symptoms of AN and BN (Garner, Olmsted, & Polivy, 1983). It consists of eight subscales: drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturational fears. It was revised in 1991 to include 27 more items and three more subscales (i.e., asceticism, impulse regulation, and social insecurity; Garner, 1991). The EDI is used as a screening measure, and to measure symptom severity and treatment outcome. It has good test-retest reliability and good concurrent validity with the EAT (Garner, Olmsted, & Polivy, 1983). The EDI provides measure of AN, BN, and above, the EDI provides measure of EDNOS.

The Morgan et al., 1998) report that weight and nutrient intake are maintained within intermediate levels of weight, while the EDE-Q is a self-report measure of the EDE that is also useful because it reduces subject and assessor burden and because it can be completed in less than 15 minutes. Reports suggest that it is a reliable measure for symptom change in adolescents with eating disorders (Passi, Bryson, & Lock, 2003; Binford, Le Grange, & Jellinek, 2005). Although the Schedule for Affective Disorders and Schizophrenia for School-Aged Children (6-18 years; K-SADS) is a widely used interview for detecting psychiatric disorders in children and adolescents and includes diagnosis specific impairment ratings and generates DSM-IV diagnoses, it does not provide sufficiently detailed evaluation of eating disorder related thoughts and behaviors needed to assess treatment response, hence the EDE is the preferred treatment outcome measure (Kaufman et al., 1997). Multiple self-report measures to assess eating disorders are available. Two of the most popular are the Eating Attitudes Test (EAT) and the Eating Disorders Inventory (EDI). The EAT consists of 40 items that employ a 6-point Likert rating scale (Garner & Garfinkel, 1979). The EAT-26 takes 26 of the 40 items which tend to account for most of the variance in total score, and is very highly correlated with the EAT (Garner, Olmsted, Bohr, & Garfinkel, 1982). It is easy to administer and takes less than 10 minutes. The EAT has good internal consistency and test-retest reliability (Carter & Moss, 1984), and has good concurrent and discriminant validity with other eating disorders measures (Garner, 1997). The EAT consists of seven factors: food preoccupation, body image for thinness, vomiting and laxative abuse, dieting, slow eating, clandestine eating, and perceived social pressure to gain weight. The EDI is a 64-item measure designed to assess symptoms of AN and BN (Garner, Olmsted, & Polivy, 1983). It consists of eight subscales: drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturational fears. It was revised in 1991 to include 27 more items and three more subscales (i.e., asceticism, impulse regulation, and social insecurity; Garner, 1991). The EDI is used as a screening measure, and to measure symptom severity and treatment outcome. It has good test-retest reliability and good concurrent validity with

Interventions

A range of interventions is available for eating disorders. The treatment modalities include cognitive behavioral therapy, family-based therapy, and medication. Cognitive behavioral therapy is the most commonly used treatment for eating disorders and involves teaching patients to recognize and challenge negative thoughts and behaviors. Family-based therapy is typically used for adolescents and involves involving the family in treatment. Medications, such as SSRIs, are often used in conjunction with therapy. It is important to note that treatment for eating disorders is often challenging and may require a multidisciplinary approach.

Outpatient

Variants of outpatient treatment may include individual therapy, group therapy, and family therapy. In addition, medication may be prescribed in some cases. The choice of treatment depends on the individual's needs and the severity of their disorder.
Interventions

A range of psychological, behavioral, and environmental treatments are used for AN. However, there are few systematic studies of treatments for AN despite the seriousness of the condition. The low base rate and medical complications/risks contribute to the difficulty of conducting randomized trials. Outpatient treatments include psychodynamic, cognitive-behavioral, and family therapy. Behavioral and nutritional treatments characterize most hospital or residential treatment regimes.

Outpatient

Variants of three psychological approaches for AN have been studied in controlled outpatiendrials: individual psychodynamic psychotherapy, cognitive-behavioral therapy (CBT), and family-based therapy (FBT). The results of these studies are summarized in Table 13.1 using the Morgan-Russell Outcome Scales as an outcome wherein a good or intermediate categorical outcome was defined as having achieved normal weight or menstruation or both (Morgan & Russell, 1988). In general, the studies suggest for adolescents with AN, some type of family therapy is superior to other treatments, though most of the studies are small in scale (Le Grange & Lock, 2005; Russell, Szmukler, Dare, & Eisler, 1987). For adults with AN, the treatment studies are characterized by high dropout rates and small numbers of subjects (Halmin et al., 2005). For AN, client reluctance to even engage in treatment creates multiple issues, especially for those over 18 years old. Thus, there is little evidence to support any specific treatment approach for this population, though both CBT for relapse prevention (Pike et al., 2004) and specialist individual treatment have shown some promise (Mcintosh et al., 2005).
<table>
<thead>
<tr>
<th>Study</th>
<th>Type of therapy</th>
<th>N</th>
<th>Age</th>
<th>Treatment Duration (months)</th>
<th># sessions</th>
<th>Drop-out rate</th>
<th>End of Tx outcome Morgan-Russell</th>
</tr>
</thead>
<tbody>
<tr>
<td>Studies of adolescent patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Russell, Szumkler, Dare, Eisler (1987)*</td>
<td>Whole family vs. individual therapy</td>
<td>21</td>
<td>16.6</td>
<td>6–12</td>
<td>13</td>
<td>19%</td>
<td>Family therapy = 50%*</td>
</tr>
<tr>
<td>Le Grange, Eisler, Dare, &amp; Russell (1992)</td>
<td>Whole family vs. separated family therapy</td>
<td>18</td>
<td>15.3</td>
<td>6</td>
<td>9</td>
<td>12%</td>
<td>Individual therapy = 18%</td>
</tr>
<tr>
<td>Robin et al. (1999)**</td>
<td>Family therapy vs. individual therapy</td>
<td>37</td>
<td>13.9</td>
<td>12–18</td>
<td>47</td>
<td>11%</td>
<td>68% overall; no differences between groups</td>
</tr>
<tr>
<td>Eisler et al. (2000)</td>
<td>Whole family vs. separated family therapy</td>
<td>40</td>
<td>15.5</td>
<td>12</td>
<td>16</td>
<td>10%</td>
<td>Family therapy = 81%*</td>
</tr>
<tr>
<td>Lock, Agras, Bryson, &amp; Kraemer (2005)</td>
<td>Family treatment, 6 vs. 12 months</td>
<td>86</td>
<td>15.1</td>
<td>6 or 12</td>
<td>10 or 20 sessions</td>
<td>12%</td>
<td>63% overall; No differences between groups</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No differences in outcome between groups</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>96% overall</td>
</tr>
<tr>
<td>Studies of adult patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Russell, Szumkler, Dare, &amp; Eisler (1987)*</td>
<td>Family therapy vs. individual therapy</td>
<td>36</td>
<td>24.1</td>
<td>12</td>
<td>13</td>
<td>29%</td>
<td>Family = 29%</td>
</tr>
<tr>
<td>Hall &amp; Crisp (1987)</td>
<td>Dietary advice vs. family/individual therapy combined</td>
<td>30</td>
<td>19.6</td>
<td>6–12</td>
<td>12</td>
<td>17%</td>
<td>Individual = 60%</td>
</tr>
<tr>
<td>Channon, de Silva, Hemsley, &amp; Perkins (1989)</td>
<td>CAT, behavior therapy and no treatment</td>
<td>24</td>
<td>22.4</td>
<td>6–12</td>
<td>24</td>
<td>13%</td>
<td>Psychotherapy = 46%</td>
</tr>
<tr>
<td>Crisp et al. (1991)</td>
<td>Assessment, inpatient vs. outpatient psychotherapy</td>
<td>90</td>
<td>22</td>
<td>10</td>
<td>11</td>
<td>24%</td>
<td>Dietary advice = 33%</td>
</tr>
<tr>
<td>Treasure et al. (1995)</td>
<td>Cognitive-analytic therapy (CAT) vs. education</td>
<td>30</td>
<td>25</td>
<td>5</td>
<td>20</td>
<td>33%</td>
<td>No differences between groups</td>
</tr>
<tr>
<td>Dare, Eisler, Russell, Treasure, &amp; Dodge (2001)</td>
<td>Focal, family, CAT or treatment as usual (TAU)</td>
<td>84</td>
<td>26.3</td>
<td>7–12</td>
<td>16</td>
<td>46%</td>
<td>Focal = 33%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Family = 36%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CAT = 27%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>TAU = 5%**</td>
</tr>
<tr>
<td>Pike, Walsh, Vricosek, Wilson, &amp; Bauer (2004)</td>
<td>CBT vs. nutritional counseling (NC) (for relapse prevention)</td>
<td>33</td>
<td>25</td>
<td>12</td>
<td>44 (CBT) 27 (NC)</td>
<td>45%</td>
<td>CBT = 44%</td>
</tr>
<tr>
<td>McIntosh et al. (2005)</td>
<td>CBT vs. IPT vs. Specialist Supportive Individual Treatment (SSIT)</td>
<td>56</td>
<td>17–40</td>
<td>5 or more</td>
<td>More than 20</td>
<td>38%</td>
<td>NC = 7%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Specialist = 56%*, CBT = 32%, IPT = 10%</td>
</tr>
<tr>
<td></td>
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<td></td>
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</tr>
</tbody>
</table>

* Denotes significant difference in outcome between groups.
<table>
<thead>
<tr>
<th>Study</th>
<th>Patients</th>
<th>CBT vs. Treatment as usual (TAU)</th>
<th>CBT vs. Baseline (CBT)</th>
<th>CBT vs. Education</th>
<th>CBT vs. Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAU</td>
<td>69% 46%</td>
<td>84 26</td>
<td>7-12</td>
<td>26</td>
<td>20</td>
</tr>
<tr>
<td>Baseline</td>
<td>63%</td>
<td>33</td>
<td>16</td>
<td>26</td>
<td>25</td>
</tr>
<tr>
<td>Education</td>
<td>65%</td>
<td>49</td>
<td>15</td>
<td>25</td>
<td>22</td>
</tr>
<tr>
<td>Exercise</td>
<td>66%</td>
<td>55</td>
<td>14</td>
<td>24</td>
<td>21</td>
</tr>
</tbody>
</table>
Derived from the psychodynamic tradition, individually based psychotherapy is one of the main outpatient treatment approaches for adolescent AN. As formulated by Capp (1978), individual therapy aims to address the underlying patterns of thought and behavior that contribute to disordered eating. For adults with AN, Monti et al. (2005) developed a modified version of Capp's approach specifically for adolescents, focusing on the development of self-esteem, self-efficacy, and the restoration of body image. More recently, Robin et al. (2019) developed a treatment program for adolescents that integrates cognitive-behavioral techniques with individual therapy to address the unique challenges faced by this population.

In addition to individual therapy, cognitive-behavioral therapy (CBT) has been shown to be effective in the treatment of AN. CBT focuses on identifying and changing negative thinking patterns and behaviors that contribute to disordered eating. The first phase of CBT for AN involves teaching patients to recognize and challenge thoughts that are contributing to their disordered eating. The second phase involves helping patients develop healthier strategies for coping with their emotions and stressors. The third phase focuses on maintaining healthy eating habits and weight management.

Family therapy has also been shown to be effective in the treatment of AN. In this approach, family members are involved in the treatment process, working together to address issues related to disordered eating. Family therapy can help family members understand the underlying factors that contribute to disordered eating and develop strategies for supporting their loved ones in their treatment.

In summary, treatment for adolescent AN involves a combination of individual and family therapy, as well as other approaches such as education and lifestyle changes. Treatment is tailored to meet the unique needs of each individual, with a focus on addressing the underlying patterns of thought and behavior that contribute to disordered eating.
trial reported, SSIT turned out to be superior to both CBT and interpersonal psychotherapy (IPT), raising the question of what may be the mechanism of change (McIntosh et al., 2005). A careful reading of the manual suggests that SSIT maintains a consistent focus on making behavioral changes in eating patterns within a supportive, motivation-enhancing therapeutic stance that appears to minimize patient resistance, perhaps by reducing fears of losing control over the change process. In the trial, IPT was less effective than CBT, suggesting that this less directive, client-focused approach is not a viable alternative treatment for adult AN even though IPT had been more promising in treatment of BN, and especially Binge Eating Disorder (BED). The common element of SSIT and CBT seems to be the explicit focus on setting behavioral goals. It has been suggested that CBTs direct challenging of distorted cognitions may elicit greater client resistance in AN than does in BN since the symptoms of AN are more ego-syntonic. If this hypothesis is supported by subsequent research, it would provide guidance to clinicians regarding differential foci of treatment of disordered eating when low weight is the primary target.

Cognitive-Behavioral Therapy for Relapse Prevention

Cognitive-Behavioral Therapy (CBT) for AN is based on the approach originally developed by Beck for treating depression and anxiety (Beck, Rush, Shaw, & Emery, 1979). CBT was first applied to BN and was quickly established as the first line of treatment (Fairburn, 36,37). Refinements of the approach—to take into account the challenges of AN—were made by Vitousek and Pike, including addressing the ego-syntonic nature of the disorder, the interaction of physical and psychological contributions to symptoms, distorted beliefs about food and weight, and pervasive deficits in self-esteem (Garner & Bemis, 1982; Pike, Walsh, Vitousek, Wilson, & Bauer, 2000). CBT focuses on cognitive and behavioral features associated with the maintenance of eating-related pathology. It aims to reduce concerns related to eating and weight, modify beliefs about weight and food, and over time to shift the focus from the focal symptoms of AN to more general aspects of the self that may predispose the individual to AN. Initial reports are promising, but further evaluation is needed. Furthermore, CBT for AN has been studied exclusively in adults with the disorder (Pike et al., 2004; Serfaty, 1999). However, many of these are young adults, suggesting it may be applicable to older adolescents. At the current time, it appears CBT may be more useful to offer after weight restoration rather than during acute treatment, when weight regain is the primary target (Pike et al., 2004).

Inpatient, Day-Hospital, and Residential Treatments

In addition to outpatient treatment approaches, more intensive treatment in hospital, day hospital, or residential programs is frequently utilized for more severe and/or chronic AN. The approaches used are usually based on behavioral principles as weight restoration is the initial goal (Jenkins, 1987). Although these treatment approaches have not been systematically investigated, they are often used for medical urgencies that develop in the context of AN due to malnutrition and as respite care for families and providers when outpatient approaches are not perceived as making adequate progress. The single review summarizing the effectiveness of inpatient and outpatient care for AN concluded that outpatient treatment in a specialist eating disorder service was as effective as
inpatient treatment among those who did not warrant emergency admission (Meads, Gold, & Burls, 2001). As outpatient care is significantly less expensive than inpatient care, reliance on such care is likely to increase and, hopefully, more rigorous evaluation of these programs will be undertaken in the future (Lock, 2003; Streigel-Moore, Leslie, Petrill, Garvin, & Rosenheck, 2000).

Inpatient psychiatric treatment programs vary greatly but most involve a combination of nutritional rehabilitation, education, medical intervention, psychotherapeutic treatment, psychosocial rehabilitation, and family therapies. Longer-term residential care programs provide intensive services (24 hour programming) and stays are generally between one and two months. Objective and systematic data on the outcomes of patients treated in residential centers is not available. Reports from these proprietary programs themselves suggest that treatment promotes recovery, prevents relapse, and reduces the development of a chronic course of AN. Because of the high cost and uncertain outcomes of these programs it is important that evaluation of residential treatment for AN be undertaken (Frisch, Franko, & Herzog, 2006). Day hospital programs for AN are also available for adults and older adolescent patients (Birchell, Palmer, Waite, Gadsby, & Gatward, 2002; Gerlinghoff, Backmund, & Franzen, 1998; Robinson, 2003; Zipfel, Lowe, Deter, & Herzog, 2000). These programs usually provide services 4 to 7 days per week and include supervised meals, therapeutic groups, and individual therapy (Olmsedt, 2002). Intensive outpatient programs (IOPs) are somewhat less intensive programs, such as three hours twice a week, and often serve a transitional role between hospital and outpatient care.

**Psychopharmacologic**

A variety of medications have been tried in treating AN (Attia, Mayer, & Killory, 2001). To date, none of them appears to be systematically useful. The use of fluoxetine has been suggested as being useful in preventing relapse after weight restoration, but more recent studies suggest that this may not be as helpful as initially believed (Kaye et al., 2001; Walsh et al., 2006). Newer antipsychotic medications may also be of use, but systematic studies are not yet available (Malina et al., 2003).

**Treatment Recommendations**

What is the clinician to make of the current data about treatment? It is sobering to see the paucity of research currently available to guide treatment. Fewer than 600 patients have been studied in systematic treatment trials (Le Grange & Lock, 2005). Further, there is no theoretical agreement about the best approach. For adolescents, all systematic studies have included a family treatment component. In all cases, family therapy has been as good as or better than individual treatment for this age group. Clinicians might reasonably take from this that family involvement in treatment of adolescent AN is likely to be beneficial. However, only one type of family treatment, FBT, has been systematically studied to date. This approach would appear to be the current first line approach for adolescent AN and has received tentative recommendation from several groups in this regard (Agency for Healthcare Resource and Quality, 2006; National Institute for Health and Clinical Excellence, 2004). The data supporting individual approaches for adolescent AN is quite limited (Robin et al., 1999). What data is available suggests that individual therapy could be a valuable treatment as well for adolescents, but even if...
but even these individually focused therapies involve parents to support adolescent development and autonomy.

It is interesting to postulate why FBT appears to be an effective treatment for AN given the psychopathology of the disorder. First, FBT takes seriously the notion that the degree of cognitive distortion is severe in AN, enough so that trusting the patient to make judgments about appropriate care in relation to food, weight, and exercise is dubious at best. The treatment approach is responsive to the extraordinarily ego-syntonic nature of AN. Instead of expecting the patient to be motivated for recovery, FBT encourages parents to recognize that this is highly unlikely given the patient’s attachment to these disordered beliefs and, therefore, initially gives the parents the responsibility to make the necessary decisions. FBT also stresses the importance of behavioral disruption of weight loss and has an explicit behavioral focus. AN is maintained, to a large degree, by these behaviors and disrupting them is seen as a key intervention. In addition, by reducing the role of professionals as intermediaries between the adolescent and AN by helping parents to make these behavioral changes at home, a major opportunity for splitting, deception, and confusion is eliminated—any of which may increase the likelihood that the patient will be able to maintain their unhealthy behaviors. It should be noted that FBT targets relatively short duration AN and recognizes the likely negative effects of long-term behavioral maintenance on the developing sense of identity increasingly aligned with AN thinking. In contrast to FBT, individual approaches rely more directly on the therapeutic relationship to develop trust so that, ultimately, the patient may decide to follow the therapist’s advice to eat and gain weight as well as challenge her distorted cognitions. Unfortunately, developing a trusting relationship takes time and may not be successful, especially because of the ego-syntonic nature of AN. Both of these eventualities may limit the usefulness of individual therapy for AN. Furthermore, quick decisive intervention may prevent unhealthy behaviors from becoming more firmly entrenched, ultimately shortening the course of the disorder and reducing medical complications.

For adults with AN, the available data provides little in the way of guidance. In addition to the problem of few studies and small numbers of participants in these studies, the retention rate in adult treatment studies is less than 50% on average (Halmi et al., 2005). This compares with retention rates of 80 to 85% in adolescent AN treatment studies. Nonetheless, the two largest studies provide conflicting conclusions about what type of treatment to use for adults with AN. Dare, Eisler, Russell, Treasure, and Dodge (2001) reported that specific therapies outperformed nonspecific supportive care, while McIntosh et al. 2005 found that nonspecific treatment outperformed specific treatments. In Dare’s study, nonspecific care was provided by nonspecialists and was perhaps a truer control for nonspecific treatment effects. McIntosh and colleague’s SSIT introduced the specialist’s experience and clinical guidance, including specific encouragement of behavior change. This formulation of therapy brings into question the purely supportive, that is, nonspecific, nature of the approach. In addition, individual CBT—another specific treatment—appears to be useful, at least for relapse after weight restoration for adults with AN (Pike et al., 2004). Thus, it might be reasonably concluded that for adults with AN specialist care and specific treatments are the best current strategies, though none of the specific approaches has truly demonstrated efficacy for low weight AN in this age group.

The failure to identify any psychopharmacologic interventions for AN may seem surprising, especially as antidepressants have been useful for adults with BN (Walsh et al.,
Eating Disorders: Anorexia Nervosa

As is the case with psychotherapy research for AN, few studies, low numbers of participants, and poor retention make definitive conclusions about the usefulness of medications for AN impossible. Even in the study by Walsh and colleagues, where the authors concluded that fluoxetine was not of benefit in relapse prevention after weight restoration for adults with AN, the dropout rate was over 50% calling into question the study’s conclusions because of the lack of sufficient follow-up data (Walsh et al., 2006). Instead, it may be that medications are helpful for a subset of patients with AN, while for the majority medication use is unacceptable rather than ineffective. Treatment of any type is often rejected because of the ego-syntonic nature of AN, but medications in particular tend to be refused (Halmi et al., 2005). Current enthusiasm for the possible benefit of atypical antipsychotics for AN is dampened by reports of patient refusal to take these medications for fear of weight gain—a known side effect of these medications. Although there is not systematic evidence yet available to support the use of any medications for AN, there is reason—based on case reports—to believe that both antidepressants and atypical antipsychotics can be useful in some cases. Use of medications for the primary symptoms of AN should be considered as a second or third line approach, but should not be discounted as not useful in all cases. The use of medications to assist with extreme anxiety, severe obsessive thinking and compulsive behavior, and depression remains an important adjunctive therapy in many instances.

Discouraging as the current state is about data from treatment studies for AN, there are some clear directions for future research. For adolescent AN the priority is to establish the relative importance of family therapy, particularly FBT in relation to age appropriate individual treatments and other family therapies. Studies are currently underway to examine just these issues. In addition, studies of medication use in adolescent AN, where compliance may be less of an issue, are needed. Again, a study to examine the role of antidepressant use as an adjunct to therapy is in process.

For adults with AN, the need to develop strategies to engage and keep patients in therapy (both psychological and psychopharmacological) is the next key advance needed (Halmi et al., 2005). Until this problem is resolved, it will be difficult to mount studies to provide efficacy data because of poor retention. At the same time, smaller studies to develop innovative approaches are needed as none of our current therapies are robust even among those who stay in treatment (Agras et al., 2004). It may be necessary, particularly with adults with AN and highly resistant adolescents with AN, to address key cognitive processes prior to addressing weight gain in order to make progress. A new therapy, cognitive-remediation therapy (CRT) aimed at cognitive abnormalities in AN including overall inflexibility of thinking and extensive attention to detail (difficulties with set-shifting, perseveration, and focusing on details compromising context) is a possible candidate (Tchanturia, Morris, Surguladze, & Treasure, 2002; Holliday, Tchanturia, Landau, Collier, & Treasure, 2005; Tchanturia, Whitney, & Treasure, in press; Whitney, Easter, & Tchanturia, in press; Davies & Tchanturia, 2005). Because many potentially effective psychological treatments (e.g., CBT) depend on fundamentally intact executive cognitive functions, cognitive impairments likely have a significant negative impact on both therapeutic engagement and the usefulness of such treatments. This, in turn, may explain in part why such treatments are rejected by patients, are not as effective as they might be, or both. Preliminary data suggests that CRT is both acceptable and feasible for adults with AN while also improving general cognitive skills needed to make use of more specific psychotherapies (Davies & Tchanturia, 2005). Future treatment studies that include CRT as a pretreatment for other more
Summary and Future Directions

Anorexia Nervosa is a serious psychiatric illness that typically begins during adolescence. The disorder also appears to be on the rise in this age group. The disorder is characterized by extreme fear of fat and/or weight gain that leads to behaviors such as extreme dieting, exercising, and purging. These behaviors in turn lead to extreme malnutrition and serious medical deterioration. Genetic, social, personality, and developmental factors likely contribute to the etiology of the disorder. There are few effective treatments for the disorder, especially in adults with more chronic illness. Mortality rates related to AN are extraordinarily high for a psychiatric disorder and long-term, comorbid psychiatric disorders are a common outcome.

There are important areas for future research in all aspects of AN. Little is known about the brain functioning of these individuals and studies using newer technologies—such as functional neuroimaging—could lead to innovations in our understanding of both the etiology and treatment of the disorder. Currently, there is a critical need for more studies of treatments and the development of new treatments for AN. Such treatments need to span the range from prevention of the development of AN—through developmentally tailored approaches for adolescents—to rehabilitation, medication and cognitive interventions for adults with chronic illness.
References


Crisp, A. H., Norton, J., Yellman, study of the effect of obesity and family psycho

British Journal of Psychiatry.


307–324). New Yo

Dare, C., Eisler, I., & Dodge, E. (2001). F with anorexia nerv

outpatient treatment, 178, 216–221.


EATING DISORDERS: ANOREXIA NERVOSA


Klumpp, K., Bulik, M., Benbent, M., and character *Journal of Nervous and Mental Disease, 185*, 185-191.


Leit, R., Pope, H., expectations of the *Playboy, cc Eating Disorders*.


EATING DISORDERS: ANOREXIA NERVOSA


Strober, M., Free. Long-term course in anorexia nervosa: Surviving the outcome of patients’ study. Inte. 22, 339–343


Strober, M., Free. Long-term course in anorexia nervosa: Surviving the outcome of patients’ study. Inte. 22, 339–343


Strober, M., Free. Long-term course in anorexia nervosa: Surviving the outcome of patients’ study. Inte. 22, 339–343


Strober, M., Free. Long-term course in anorexia nervosa: Surviving the outcome of patients’ study. Inte. 22, 339–343


Strober, M., Free. Long-term course in anorexia nervosa: Surviving the outcome of patients’ study. Inte. 22, 339–343


Strober, M., Free. Long-term course in anorexia nervosa: Surviving the outcome of patients’ study. Inte. 22, 339–343


