CHAPTER 1

Introduction and Background Information on Anorexia Nervosa

Purpose of This Manual

This manual contains background information essential to the understanding of adolescent anorexia nervosa (AN) and family-based treatment (FBT). It presents a treatment program using FBT, including the details of specific sessions and phases of therapy, based on research that has demonstrated effectiveness. The manual derives from several controlled trials of family treatment for AN, initially done at the Maudsley Hospital in London and subsequently studied at Stanford University's Department of Psychiatry and Behavioral Sciences, Division of Child and Adolescent Psychiatry, and at Lucile Salter Packard Children's Hospital (Eisler et al., 1997; Le Grange, 1993; Le Grange, Eisler, Dare, & Russell, 1992; Russell, Szmukler, Dare, & Eisler, 1987) and at the University of Chicago Eating Disorders Program in the Department of Psychiatry and Behavioral Neuroscience. This manual is intended for use by qualified therapists who have experience in the assessment and treatment of eating disorders in adolescents. It may also be used by therapists in training under the guidance of experienced clinicians. It is not intended as a self-help manual. The treatment described should be conducted with the appropriate consultation and involvement of professionals in pediatric medicine, nutrition, and child psychiatry. This manual is explicitly not to be used for research purposes without further consultation with the authors. Clinical response to these interventions is not guaranteed nor implied by the use of this manual.

The overall perspective of this therapeutic approach is to see the family as a resource in the treatment of adolescent patients with AN. Mobilizing parents and family members as a resource is the most important theoretical position that sets this approach apart from other family and individual therapies for AN. Parents play an important role throughout the three phases of this treatment. Thus the first phase of treatment attempts to reinvigorate parental roles in the family system, particularly as they are related to the patient's eating behaviors. This is considered the key therapeutic maneuver in this phase of family treatment. Therapy is almost entirely focused on the eating disorder and its symptoms and includes a therapeutic family meal. The therapist's goals in this phase are first to develop a strong parental alliance and second to align the patient with a peer or sibling subsystem. Parents are encouraged to work out for themselves the best way to promote weight gain and normalize eating in their child with AN. The second phase begins when the patient accepts parental demands to increase food intake and begins to experience steady weight gain. At this point, family therapy focuses on other family problems and the effect these issues have on the parents' task of supporting steady weight gain in the patient. The third phase begins when the patient achieves a relatively stable weight and self-starvation has abated. The central theme is the establishment of a healthy adolescent relationship with parents in which AN does not constitute the basis of the interaction. This entails working toward increased personal autonomy for the adolescent, age-appropriate family boundaries, and the need for the parents to reorganize their life together as their adolescent children become more independent.

The manual consists of 15 chapters. Chapter 1 provides an introduction and overview of AN in adolescents. Chapter 2 provides a specific introduction to family therapy for AN and provides a more detailed introduction to the therapy that is the subject of the remaining chapters. Chapters 3–13 provide detailed instructions for how to conduct FBT for AN. Particular emphasis is devoted to the initial sessions because these sessions set the tone and therapeutic style that will be employed throughout the treatment. In these chapters, descriptions of what therapeutic maneuvers the therapist is to undertake and why, as well as illustrations of each of these maneuvers are provided. In addition, examples illustrating the series of therapeutic maneuvers as they are integrated in a session are presented. We also present a complete case illustration in Chapter 14. We include in this new edition a concluding chapter that discusses anticipated future directions in research, training, and advocacy related to FBT.

In this introductory chapter, the focus is primarily on general background material related to AN. The pertinent research literature, the way the illness presents in adolescents, and a discussion of treatment options and prognosis are reviewed.

Overview of Anorexia Nervosa in Adolescents

AN is a serious psychiatric illness with a prevalence estimated at 0.48% among girls ages 15–19; it is estimated to be 9 to 10 times more common in girls than in boys (Lucas, Beard, O'Fallon, & Kurland, 1991; Hoek & van Hoeken, 2003; Hoek et al., 2005; Keski-Rahkonen et al., 2007; van Son et al., 2006). In AN, pathological thoughts and behaviors concerning food and weight, as well as emotions about appearance, eating, and food, co-occur. These thoughts, feelings, and behaviors lead to changes in body composition and functioning that are the direct result of starvation. Treatment of AN is complex and requires attention to broad psychiatric, medical, and nutritional aspects of the disease (American Psychiatric Association, 2000; Steiner & Lock, 1998). In adolescents, the illness severely affects physical, emotional, and social development (Fisher et al., 1995; Le Grange, Eisler, Dare, & Russell, 1992; Lucas et al., 1991; Yates, 1990). Unfortunately, AN becomes a chronic illness for many patients. Multiple hospitalizations and prolonged treatment are often the rule for many patients with AN (Kreipe & Uphoff, 1992; Kreipe et al., 1995; Steiner, Mazer, & Litt, 1990; Yager et al., 1993).

Epidemiology and Comorbidity

Lucas et al. (1991) performed a population-based incidence study of AN in Rochester, Minnesota, over a 50-year span (1935–1984). The incidence rate for females decreased from 16.6 per 100,000 person-years between 1935 and 1939 to 7 between 1950 and 1954 and increased to 26.3 between 1980 and 1984. The incidence rates for women over 20 years remained constant, but there was a significant increase for females ages 15–24. The overall age-adjusted incidence rate was 14.6 for females and 1.8 for males. Recent epidemiological studies continue to support these earlier findings (Hoek & van Hoeken, 2003; Keski-Rahkonen et al., 2007; van Son et al., 2006).

Considerable evidence suggests that AN often co-occurs with other psychiatric disorders. Depression is a common comorbid diagnosis, with

a lifetime prevalence rate of up to 63% in some studies (Herzog, Keller, Sacks, Yeh, & Lavori, 1992). In addition, Smith, Nasserbakht, Feldman, and Steiner (1993) found that persistent AN coexisted with a high incidence of anxiety disorders. In particular, Rastam (1992) found that 35% of patients with AN also suffer from comorbid obsessive–compulsive disorder. More recent studies continue to highlight these high rates of comorbidity among adolescents with AN (Bulik, Sullivan, Fear, & Joyce, 1997; Godart, Flament, Perdereau, & Jeammet, 2002; W. Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Keel, Klump, Miller, McGue, & Iacono, 2005). A moderate degree of overlap between avoidant personality disorder and AN has been shown in adult patients (Herzog, Keller, & Lavori, 1992), but it is unclear whether this overlap will be true for adolescents or children.

Etiology and Risks for Development

The causes of AN are unknown. Most clinicians and researchers agree, however, that AN has multiple determinants (Garfinkel & Garner, 1982; Garner, 1993; Hsu, 1990; Lask & Bryant-Waugh, 1992) that emerge in a developmental sequence (Steiner & Lock, 1998; Steiner, Sanders, & Ryst, 1995). Onset usually occurs during adolescence, the mean age being 17 (Hsu, 1990). Most accounts of eating disorders emphasize the individual's difficulty negotiating the developmental demands of adolescence. Recent research focuses on antecedent risks for these disorders (Steiner & Lock, 1998; Sharpe, Ryst, Hinshaw, & Steiner, 1998; Stice, Agras, & Hammer 1999). These studies recognize that premorbid histories identify probable antecedent risks for the disorders. For example, in a short-term prospective study, Attie and Brooks-Gunn (1989) tested the hypothesis that the development of eating problems represents an accommodation to puberty. They followed a group of girls from 7th through 10th grades for 2 years. They found that eating problems emerged in response to pubertal change, especially fat accumulation. Girls who felt most negatively about their bodies at puberty were at highest risk of developing eating difficulties after initial eating-problem scores were taken into account.

There also appear to be some context-dependent risk factors associated with the development of AN. Among these are teasing by peers (Fabian & Thompson, 1989), discomfort in discussing problems with parents (Larson, 1991), maternal preoccupation with restricting dietary intake (Hill, Weaver, & Blundell, 1990), and acculturation to Western values in immigrants (Pumariega, 1986; Steinhausen, 1995). In addition,

a variety of associated risks have been identified; being female and having a pear-shaped body and a body mass index high in fat are identified as constituting risk (Radke-Sharpe, Whitney-Saltiel, & Rodin, 1990). In some studies, a high incidence of sexual abuse has been reported by women diagnosed with an eating disorder (Palmer, Oppenheimer, Dignon, Chalonor, & Howells, 1990; Rorty, Yager, & Rossotto, 1994). Although some studies of families with eating disorders describe distinct profiles by both self-report and observational methods (Steiger, Leung, & Houle, 1992), these observations have not been confirmed in systematic studies of risk. Based on these reports, families of patients with AN appear more controlled and organized, whereas families of bulimic patients are more chaotic, conflicted, and critical.

An understanding of the biological basis of AN is still in its infancy. Progress is being made in identifying how genes are involved in psychiatric disorders (Berrettini, 2000; Hudziak & Faraone, 2010). Understanding of genetic contributions to the development of eating disorders is limited (Gorwood, Kipman, & Foulon, 2003; Kaye et al., 2008; Klump & Gobrogge, 2005), but family studies, twin studies, molecular genetics (genome-wide linkage and candidate gene association studies), and genome-wide association studies (GWAS) have all been conducted in relation to AN. Family studies show that AN clusters in families to a substantial degree because of genetic factors (Lilenfeld et al., 1998; Strober, Freeman, Lampert, Diamond, & Kaye, 2000). Studies suggest that rates of these eating disorders are about five times the expected rates in affected compared with unaffected families (Tozzi et al., 2005). Genetic factors likely account for more than 50% of the heritable risk for developing an eating disorder (Bulik, 2004; Lilenfeld et al., 1998). Twin studies find heritability estimates ranging from approximately 30 to 75% in AN (Bulik, Slof-Op't Landt, van Furth, & Sullivan, 2007; Bulik et al., 2006; Bulik, Sullivan, Wade, & Kendler, 2000; Bulik, Sullivan, & Kendler, 1998). In addition, Klump, McGue, and Iacono (2000) found that genetic and environmental influences on eating-disordered thoughts and behavior differed by age, with heritability being a factor only in the adolescent-onset group (Klump et al., 2000; Klump, Burt, McGue, & Iacono, 2007).

Turning now to molecular genetic data, linkage studies in AN are few. Grice et al. (2002), using a sample of pure restricting-type AN, found a susceptibility locus on chromosome 1, while Devlin et al. (2002) found additional loci on chromosomes 2 and 13. Still other linkage reports have found statistically significant findings on chromosome 1 related to eating behavior and satiety, but none of these studies have been

replicated. A promising area of research is examining genes related to the serotonin pathway; some studies suggest that dysregulation of serotonin (5-hydroxytryptamine, or 5-HT) may contribute to the development of eating disorders (Ferguson, La Via, Crossan, & Kaye, 1999; Frank et al., 2002; Kaye, Gwirtsman, George, & Ebert, 1991).

Another strategy for understanding the biological basis of AN that has been rapidly developing is the use of functional magnetic resonance imaging (fMRI) to explore functional brain changes in AN. Using food-relevant paradigms, Gordon, Dougherty, and Fischman (2001) found elevated temporal lobe activation in low-weight patients with AN, and Kurosaki, Shirao, Yamashita, Okamoto, and Yamawaki (2006) identified elevated medial prefrontal cortex (PFC) and anterior cingulate cortex (ACC) activation in both underweight and recovered patients with AN. Studies have found evidence of increased reward sensitivity in AN (Jappe et al., 2010). Kurosaki et al. (2006) used brain scans on participants undergoing a taste test with sucrose and found that patients with AN who had regained weight showed comparatively decreased activation in the insula and striatum during the sucrose tasting compared with controls. In addition, Wagner and colleagues (2007) explored reward processing using a gambling game. They found that subjects with AN had increased dorsolateral PFC activation compared with controls. Recent neuroimaging data have examined how cognitive control may play a role in AN through the involvement of frontostriatal brain circuitry (Marsh et al., 2009; Zastrow et al., 2009). Zastrow et al. (2009) found decreased activation in the ACC and striatum associated with impaired cognitive-behavioral flexibility in patients with AN. Further, Oberndorfer, Kaye, Simmons, Strigo, and Matthews (2011) using a stop-signal (go/no-go) task demonstrated that patients with AN showed comparatively lower activation levels in these regions, suggesting that they required fewer cognitive resources than healthy controls to inhibit response.

In addition, clinicians and researchers also see an important role for the patient's motivational state in the genesis of AN. The psychobiological perspective of Crisp and colleagues (Crisp, 1997; Crisp et al., 1980) suggests that the symptoms of starvation and emaciation represent attempts to cope with the demands of adolescence by regression to an earlier developmental level. Bruch's (1973) psychodynamic formulation conceives of the patient as becoming overwhelmed by feelings of ineffectiveness and emptiness and the concomitant inability to access her own thoughts, feelings, and beliefs. Experiencing the self as lacking "a core personality" (Bruch, 1995, p. 10), she experiences the demands of puberty as overwhelming

and retreats to a rigid preoccupation with food and eating. These theories have not been empirically tested, but they are used to support interventions for AN aimed at promoting adolescent autonomy.

Efforts are also being made to reconcile biological deficits and developmental factors with a psychodynamic understanding of eating disorders. Children at risk for the development of eating disorders may be vulnerable because of appetite and satiety dysregulation (Steiner, Smith, Rosenkrantz, & Litt, 1991; Stice et al., 1999). In addition, temperamental traits are important. For example, Strober (1991) emphasizes that patients with AN commonly express traits of harm avoidance, low novelty seeking, and high reward dependence—traits heavily influenced by genetic factors (Cloninger, 1986, 1987, 1988). These traits are at odds with the developmental tasks associated with puberty that require risk taking and independence. Adolescents without these abilities may retreat from demands for which they feel ill prepared. In addition to these temperamental factors, personality differences that may serve as precipitants to an eating disorder have been noted in persons with AN. Patients with AN are typically anxious, inhibited, and overcontrolled (e.g., Casper, Hedeker, & McClough, 1992; Leon, Fulkerson, Perry, & Cudeck, 1992).

Treatment

One of the major complications of AN is that severe medical problems commonly co-occur with the illness. The short- and long-term medical complications of AN in adolescents are well known (Fisher et al., 1995). Changes in growth hormone, hypothalamic hypogonadism, bone marrow hypoplasia, structural abnormalities of the brain, cardiac dysfunction, and gastrointestinal difficulties are all common. Recent studies continue to document that the most significant medical problems for adolescents that differ from those of adults are the potential for significant growth retardation, pubertal delay or interruption, and peak bone mass reduction. Risks of death as a result of complications of AN are estimated at 6–15% (Steinhausen, Rauss-Mason, & Seidel, 1991, 1993), with half the deaths resulting from suicide (Arcelus, Mitchell, Wales, & Nielsen, 2011; Crow et al., 2009; Keel et al., 2003).

Usual treatment of AN requires a multidisciplinary approach. Guidelines for the psychiatric and medical treatment of AN are published (American Psychiatric Association, 2000; Kreipe et al., 1995; National Collaborating Centre for Mental Health, 2004). Overall, results of all treatment types are modest to moderate (Kreipe & Uphoff, 1992).

Inpatient Treatments

The role of hospitalization for AN has changed dramatically over the past 20 years, at least in the United States. Currently, hospital treatment in the United States is limited to brief acute weight restoration and refeeding. A variety of older reports appeared to demonstrate the effectiveness of inpatient hospitalization for weight gain during acute treatment of AN. For example, Bossert, Schmeolz, Wieland, Junker, & Krieg, (1988) found overall clinical improvement in 16 female patients after an average 3-month inpatient treatment using a behavioral approach, whereas Jenkins (1987), using a strict behavioral refeeding program for a 6-month treatment period, found that 70% showed continued improvement at a 3-year follow-up. However, hospital-based treatments have unclear longer term benefits, as McKenzie (1992) reported that about 40% of hospitalized patients with AN are readmitted at least once. Further, these patients spent more time in the hospital with each admission than did any other patient group with nonorganic disorders. Two randomized clinical trials (RCTs) suggest that outpatient treatment is overall as effective as inpatient treatment for AN. The first study, published in 1991, found no differences in the outcomes of patients randomized to outpatient treatment compared with inpatient treatment, though all treated patients fared better than those who were not treated (Crisp et al., 1991). A more recent study specifically examined adolescents with AN. A total of 167 adolescents with AN were randomly allocated to standard outpatient care, specialized cognitive-behavioral therapy (CBT), or a 16-week inpatient specialized program (Gowers et al., 2007). There were no differences in outcome at the end of treatment or at follow-up among the groups, though the specialized CBT treatment was the most cost-effective (Byford et al., 2007).

The combination of the increasing pressure to reduce both the use of high-cost inpatient treatment and the disruption to the adolescent's usual life and the evidence that outpatient treatments can be as effective warrants a focus on outpatient therapies for AN. One of the goals of FBT is to prevent or reduce the need for inpatient treatment by assisting the family in the outpatient setting to promote weight gain in their child. If these efforts fail, hospitalization is still an important treatment option.

Outpatient Psychosocial Treatments

Outpatient treatment approaches for AN are also being explored, including individual, family, and group therapy. A number of preliminary

controlled and uncontrolled treatment trials of AN took place in the 1970s and 1980s. Minuchin et al. (1978) reported a good outcome in 80% of cases using structural family therapy. In addition, Stierlin and Weber (1989) published results of a study using family treatment with a group of 42 families without a control group. At follow-up they found that about two-thirds were improved. These results came from treatments that were quite brief—most lasting about 6 months. Only 25% of patients received treatment that lasted more than 1 year. The average was only six sessions of treatment per family (Stierlin & Weber, 1989). Thus these early uncontrolled studies suggested that effective treatment of AN could be accomplished in a relatively short time using a low-intensity family approach .

It is striking how few RCTs have been conducted for AN considering how long the illness has been known in the medical literature and how severe are the medical and psychological problems associated with it (Le Grange & Lock, 2005). A recent review of studies for adult AN finds no systematic evidence that any treatment is effective (Bulik, Berkman, et al., 2007). Treatments studied included CBT (Pike, Walsh, Vitousek, Wilson, & Bauer, 2003; Treasure et al., 1995). For the most part, the main finding from these studies of adults with AN is that patients will not stay in treatment with no evidence of benefit from any specific approach (see Table 1.1). In contrast, there are now six RCTs of treatments for adolescent AN that demonstrate the effectiveness of FBT (Eisler et al., 2000; Le Grange,

TABLE 1.1. Attrition Rates in Published RCTs of Adult AN

Study	Number of subjects	Attrition rate
Russell, Szmukler, Dare, & Eisler (1987)	36	31% (11/36)
Channon, de Silva, Hemsley, & Perkins (1989)	24	13% (3/24)
Treasure et al. (1995)	30	33% (10/30)
Serfaty, Turkington, Ledsham, & Jolley (1999)	35	34% (12/35)
Dare, Eisler, Russell, Treasure, & Dodge (2001)	84	64% (54/84)
Pike, Walsh, Vitousek, Wilson, & Bauer (2003)	33	46% (15/33)
McIntosh et al. (2005)	56	38% (21/56)
Halmi et al. (2005)	122	63% (77/122)
Walsh et al. (2006)	93	57% (53/93)
Total nine studies	513	50% (256/513)

Eisler, Dare, & Russell, 1992; Lock, Agras, Bryson, & Kraemer, 2005; Lock et al., 2010; Robin et al., 1999; Russell et al., 1987). There are a number of reasons that adolescents with AN might be more responsive than adults to treatment, including the shorter duration of their AN, parental insistence on treatment, and parental involvement in treatment. We review these adolescent studies in detail in Chapter 2.

Medication Treatments

There are a few studies of medication treatment for AN, although most exclusively examine adult samples (Garfinkel & Garner, 1987; Couturier & Lock, 2007). During periods of acute medical compromise, psychopharmacological agents are of limited use. Medications most frequently used include antidepressants and low-dose neuroleptics. Low-dose neuroleptics are purportedly used to address severe obsessional thinking, anxiety, and psychotic-like thinking. However, there is little evidence that they help with those symptoms or behaviors associated with body shape and weight. Many older small studies have demonstrated few significant improvements in patients as a result of psychopharmacological intervention (Agras & Kraemer, 1983). Early studies explored the role of serotonin reuptake inhibitors in the treatment of AN in terms of relapse prevention, but more recent systematic studies suggest that they do not prevent relapse (Gwirtzman, Guze, & Yager, 1990; Kaye et al., 2001; Walsh et al., 2006). Although case reports and case studies suggest that newer antipsychotics (i.e., atypical antipsychotics) are helpful for weight gain and agitation, a recently published pilot RCT did not find support for the usefulness of risperidone in adolescents with AN (Hagman et al., 2011). Thus, although it appears that medications may be useful in some cases, it is unclear when and for whom they will be beneficial. Nonetheless, the use of psychopharmacological treatment for comorbid disorders, such as depression and anxiety, seems to be indicated (Couturier & Lock, 2007). The treatment proposed in this manual allows the use of these agents for comorbid conditions.

Outcomes for Patients with Anorexia Nervosa

A variety of studies have looked at the short-term, intermediate, and long-term outcomes of patients with AN after treatment. Most of these studies are of adult populations, although many of the patients in these studies

presumably had AN as teenagers. Studies have generally demonstrated that approximately half have good outcomes, one-fourth have intermediate outcomes, and about one-fourth do poorly (Herzog et al., 1999; Ratnasuriya, Eisler, & Szmukler, 1991; Smith et al., 1993; Steinhausen et al., 1991, 1993; van der Ham, Van Strien, & van Engeland, 1994; Walford & McCune, 1991; Steinhausen & Weber, 2009). Less than 5% of the patients died (Yager et al., 1993), but death rates as high as 20% have been reported in chronically ill adults with AN (Ratnasuriya et al., 1991). Assessment of recovery in these studies has been generally confined to measures of weight and nutritional rehabilitation, but some studies indicate that other psychiatric and social aspects of the illness persist. Herzog et al. (1996) reported that the bulimic subtype of patients with AN had a higher short-term recovery rate than patients with restricting-type AN. Treatment compliance and personality variables may be important mediators of improved treatment outcome (Steiner et al., 1990). Higher levels of general psychopathology increase the risk of poorer treatment outcomes, though depression itself was of no predictive value in adolescent samples (Herpertz-Dalmann, Wewetzer, Schulz, & Remschmidt, 1996). It should be noted that the definitions of recovery are disputed in AN (Couturier & Lock, 2006a, 2006b). In fact, outcomes vary considerably depending on the outcome measure utilized. One study found recovery rates in the same population that ranged from 3 to 95% based on changing definitions (Couturier & Lock, 2006a). Because there is evidence that adolescents who are not ill for a long duration can make a full psychological and weight recovery, the bar for recovery should likely be set high for this age group (Lock et al., 2010).

Summary

This chapter reviewed the etiology, clinical presentation, treatment, and prognosis of AN to provide general background information. It stressed several important aspects of AN and its treatment that bear specifically on the approach used in this manual. The first of these is that AN is a disorder that primarily begins in adolescence and seems to bear some relation to difficulties associated with adolescent development. As such, approaches that take into account the developmental issues associated with adolescence are most likely to succeed. Recovery appears to be best for patients who are treated early in the course of the illness, supporting the idea that adolescent interventions should be paramount in preventing

the development of a more chronic and unremitting form of the illness. In addition, although treatment approaches have not been particularly well studied, we note that for adolescents FBT appears to be superior to individual approaches. This may seem counterintuitive to some clinicians who emphasize the adolescent's need for autonomy and self-control, which is indeed an expected part of adolescent development. Instead, FBT emphasizes that the adolescent with AN who is not able to manage eating and weight gain without the help of parents must "get back on track" so that the usual work of adolescent individuation can be taken up again without the symptoms of AN. Thus families appear to be an important resource for adolescents in recovering from AN. The manualized version of FBT described in the following chapters takes these observations into account. That is, it is designed to specifically address the need for weight restoration by parents of adolescents whose eating behavior is out of their control; it also aims to support the developing adolescent in the context of the family.

In sum, FBT, described in the following pages, is an empirically supported treatment that allows a rapid and relatively short outpatient treatment schedule that fits well within the frame of accepted treatments for AN. This treatment differs from many others in several important ways as well. Among these are the use of the parents to help in promoting weight gain at home, the initial focus on normalizing eating until weight is recovered, and the deferring of general adolescent and family issues until the eating-disordered behavior is well under control. A more thorough discussion of the advantages of this approach is undertaken in Chapter 2.