ACUTE TREATMENT OUTCOMES AND FAMILY FUNCTIONING OF

CHILDREN AND ADOLESCENTS DIAGNOSED

WITH ANOREXIA NERVOSA

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Dedicated with love to my husband, John.

ACUTE TREATMENT OUTCOMES AND FAMILY FUNCTIONING OF CHILDREN AND ADOLESCENTS DIAGNOSED WITH ANOREXIA NERVOSA

by

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Recent studies have suggested that there is a relationship between treatment outcomes and baseline factors related to family functioning and specific eating disorder symptoms. However, these relationships have not been studied extensively within a pediatric population hospitalized for treatment. Therefore, it is unknown whether these relationships exist within an acutely ill population and

whether these baseline characteristics improve significantly immediately following acute hospitalization. Given these limitations, the aims of the present study were to identify aspects of family functioning and eating cognitions and attitudes at admission that predict outcome at discharge, and evaluate what aspects of family functioning and eating cognitions and attitudes improve during an acute treatment period. The sample consisted of 41 patients diagnosed with anorexia nervosa or eating disorder not otherwise specified between the ages of 10 and 17 years. At admission, all patients were administered a structured clinical interview to obtain valid psychiatric diagnoses. Additionally, patients completed self-report measures of eating cognitions, eating attitudes, and family functioning; while parents completed a self-report measure of family functioning. Families also participated in a standardized clinician-rated observational measure of family functioning. All measures were re-administered at discharge, and the patient's body mass index (BMI) at admission and discharge were obtained from the medical record. The attrition rate from intake to discharge for this study was 26.8%. Overall, it appeared that parents and patients perceived their families to be healthy at intake, with little improvements noted over the course of treatment. However, standardized observations characterized these families as being affectively avoidant. Additionally, parental perception of adaptive family functioning at intake was predictive of outcome based upon the unit psychiatrist's assessment, and patient perception of healthy familial Expressiveness at intake

was predictive of outcome based upon pathological eating attitudes. BMI and eating attitudes based upon eating behavior during treatment improved significantly over the course of treatment. However, patients continued to endorse unhealthy eating cognitions at discharge. These results suggest that weight restoration and pathological eating behavior are the first symptoms to improve during an aggressive treatment period, and psychological symptoms may require a longer period of treatment to remit.

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LIST OF ABBREVIATIONS

AN: Anorexia Nervosa

BMI: Body Mass Index

CGI-I: Clinical Global Impressions Scale: Improvement

CMC: Children's Medical Center

EAT-26: Eating Attitudes Test—26

ED: Eating disorder (refers to general diagnostic category of eating disorders)

LOT: Length of Treatment

MAC-R: Mizes Anorectic Cognitions Questionnaire—Revised

ns: Non-Significant

SFI-II: Self-Report of Family Functioning—2nd Edition

TCFES: Timberlawn Couple and Family Evaluation Scales

CHAPTER ONE

INTRODUCTION

Statement of the Problem

Although behaviors and symptoms typically associated with disordered eating date back to ancient times (Bemporad, 1997), the occurrence of anorexia nervosa has increased markedly over the past century, with notable peaks occurring in the 1970s and 1980s (Hoek & van Hoeken, 2003; Lewinsohn, Striegel-Moore, & Seeley, 2000). While many of the epidemiological studies related to eating disorders have been conducted within the adult population, it has become evident that eating disorders within the child and adolescent population have increased, necessitating further research of this special population. The incidence of anorexia nervosa has been estimated to be highest among females aged 10 to 19-years (Turnbull, Ward, Treasure, Jick, & Derby, 1996), with females between the ages of 15 and 19-years comprising roughly 40% of all identified cases (Hoek & van Hoeken, 2003). Furthermore, studies have found that the average age of onset appears to be in the mid-to later-teens between the ages of 16 and 18-years (Lewinsohn, Striegel-Moore, & Seeley, 2000). Less conservative estimates of disordered eating, which include sub-threshold levels of eating pathology, suggest that 10% to 50% of high school students and as many as

22% of adolescent and college-aged females engage in disordered eating, such as restricting, binging, and purging (Fisher et al., 1995; Robin, Gilroy, & Dennis, 1998; Vitousek, Watson, & Wilson, 1998). Collectively, these staggering figures give reason to believe that disordered eating is relatively common among children and adolescents, specifically females, when compared to the population at large.

Despite the noted surge in the prevalence of pediatric anorexia nervosa, base rates for eating disorders have remained relatively low in the general population and in comparison with that of other psychiatric disorders. As such, there continues to be a paucity of research within this area relative to other psychiatric disorders that affect children and adolescents, specifically regarding treatment outcomes in relation to eating-disordered cognitions and attitudes. However, a recent review of the literature found that body weight and menstrual function, age of onset and duration of illness, intrafamilial disturbances, comorbid psychiatric disorders, psychological and social functioning, and number of hospitalizations were predictive of outcome across several studies (Tozzi, Sullivan, Fear, McKenzie, & Bulik, 2003). Although there appears to be a relationship between treatment outcomes and baseline factors related to family functioning and specific eating disorder symptoms, a clear and consistent relationship has not emerged due to methodological issues, such as inconsistent definitions of outcome, variable follow-up periods, and variable inclusion criteria regarding treatment status (Pike, 1998).

To date, much focus has been on evaluating short-term (3-months to 1-year) and long-term (post 1-year) outcomes of anorexia nervosa, with little attention toward acute or immediate treatment outcomes. A recent study conducted by Treat, Gaskill, McCabe, Ghinassi, Luczak, and Marcus (2005) underscored the importance of studying acute treatment outcomes and anorexia nervosa within an intensive treatment setting. Treat and colleagues contend that gaining a better understanding of treatment outcomes for those requiring hospitalization is vital within the context of increasing pressures to decrease length of hospitalizations, despite the serious physical and psychological ramifications of anorexia nervosa. Expectations of acute and intensive treatment should be based on realistically attainable goals during such an abbreviated period of time, and longer-term treatment planning cannot be effectively executed without a solid understanding of what treatment needs remain following hospitalization. Moreover, treatment and recovery at this specific juncture may prove to be quite critical with regard to the longer term course of illness, recovery, and remission within the pediatric population in light of data which suggests that children and adolescents who are treated earlier in the course of illness are less likely to relapse after recovery (Herzog, Keller, & Lavori, 1988; Pike, 1998; Steinhausen, 2002).

Given the aforementioned limitations of the current literature and the clinical relevance of acute treatment outcomes, further research is warranted

within the area of pediatric anorexia nervosa. The primary aim of the current study is to examine the nature of family functioning and eating disordered cognitions in a sample of children and adolescents hospitalized for anorexia nervosa, and to identify aspects of family functioning and eating disordered cognitions at admission that predict outcome at discharge. Additionally, a secondary aim of this study is to evaluate what aspects of family functioning and eating disordered cognitions improve during an acute and intensive treatment period.

CHAPTER TWO

LITERATURE REVIEW

Significance and Background

Prevalence and Incidence

The pediatric population. As previously noted, the prevalence of eating disorders has increased markedly among children and adolescents, becoming one of the more common disorders in the late teens and early adulthood (Madden, 2004). Specifically, numerous studies have suggested an upward trend in the incidence of anorexia nervosa among females aged 15 to 24-years from 1935 to 1989 and among 10 to 14-year olds starting in the 1950s (Hoek & van Hoeken, 2003). Among adolescent females, the current lifetime prevalence rate for eating disorders is estimated to be 23 in 1,000, with an incidence rate of approximately 2.8% (Lewinsohn, Striegel-Moore, & Seeley, 2000). However, considerably fewer cases have been noted to occur among adolescent males, with a prevalence estimated at 1.4 in 1,000 and an incidence rate of less than 1.0 per 100,000 persons a year (Lewinsohn, Striegel-Moore, & Seeley, 2000). Additionally, when considering anorexia nervosa in isolation from other eating disorders, the prevalence rate is roughly between 0 and .9% with an average of .3% among

adolescent females, while the incidence rate is roughly 8 cases per 100,000 per year (Hoek & van Hoeken, 2003). However, the aforementioned prevalence and incidence rates are likely an under-representation of the true figures given that subthreshold levels of disordered eating are not accounted for and that individuals are generally prone to underreporting symptoms (Hoek & van Hoeken, 2003).

Ethnic and cultural differences. Eating disorders have historically been conceptualized as a psychiatric disorder of affluent, Western, Caucasian females. Although there has been a paucity of empirical research investigating the prevalence and incidence of eating disorders in ethnically and culturally diverse populations such studies have increased in recent years. Hoek and van Hoeken (2003) cite several transcultural studies from the 1970s and 1980s that suggest the prevalence and incidence of anorexia nervosa is significantly lower in non-Western countries. However, subsequent studies have suggested that symptoms related to disordered eating may occur more often in non-Western and developing countries than was thought, although specific prevalence and incidence rates for such countries are difficult to estimate.

Additionally, studies investigating ethnic differences within the United States have yielded inconsistent results. Although accurate prevalence and incidence figures of adolescent anorexia nervosa in ethnic minority groups are unknown (Nielsen, 2001), there is evidence to suggest that Caucasian adolescents

are more vulnerable to body image concerns and eating disorders than African-American and Latino-American adolescents (Barry & Grilo, 2002). Striegel-Moore, Dohm, Kraemer, Taylor, Daniels, Crawford, et al. (2003) found similar results in a sample of African-American and Caucasian women. In their sample, African-American women were less likely to meet criteria for anorexia nervosa or endorse body image concerns. Ethnic differences detected in studies of anorexia nervosa have supported sociocultural models that predict less risk for an eating disorder among ethnic minorities due to fewer cultural pressures to be thin (Shaw, Ramirez, Trost, Randall, & Stice, 2004). Shaw and colleagues tested the robustness of such sociocultural models in a community sample of adolescent Asians, African-Americans, Latinos, and Caucasians. They found that African-Americans and Latinos were less likely to have internalized a thin ideal when compared to Asians and Caucasians. However, there were no differences across ethnic groups with regard to the following factors: fear of fat, weight and shape concerns, amenorrhea, compensatory behaviors, and body mass index. Therefore, Shaw and colleagues contend that differences across ethnic groups within the United States may be diminishing as increasing sociocultural pressures to be thin promulgate across all ethnic groups. However, the precise nature of such ethnic differences and similarities remain equivocal given inconsistent research findings.

Medical and Psychological Illnesses Concomitant with Anorexia Nervosa

Numerous medical and comorbid psychiatric conditions have been noted to occur in conjunction with anorexia nervosa, complicating diagnostic and treatment implications. Neumark-Sztainer, Story, Dixon, and Murray (1998) investigated medical complications concomitant with eating disorders and found that adolescents diagnosed with anorexia nervosa are vulnerable to various health problems, some of which may be irreversible in severe cases. For instance, adolescents diagnosed with anorexia nervosa are at an increased risk for experiencing dental problems, renal disease, cardiac abnormalities and nutritional deficiencies which may cause growth retardation resulting in short stature, delayed pubertal development, reproductive sterility, incomplete development of secondary sex characteristics, and deficiencies in peak bone mass resulting in osteoporosis in later life. Further, individuals with anorexia nervosa have a mortality rate of 5% to 10% per decade, which is twelve times that of the normal population, and have a one in three chance of relapse (Madden, 2004).

Research has also demonstrated that children and adolescents diagnosed with an eating disorder are more at risk for having comorbid psychiatric disorders and disturbances when compared to non-eating disordered peers. For instance, a study conducted by Lewinsohn, Striegel-Moore, and Seeley (2000) found that 89.5% of adolescent females with eating disorders were also diagnosed with a

comorbid psychiatric disorder, such as depression, anxiety, or substance abuse. Additionally, children and adolescents who engage in severe restriction of food intake are at an increased risk of poor self-esteem, poor concentration, and sleep disturbances, even within cases characterized by subclinical levels of eating pathology (Fonseca, Ireland, & Resnick, 2002). In similar regard, Lewinsohn and colleagues found that adolescents with partial-syndrome eating disorders were more similar to those with full-syndrome disorders than the non-clinical group in terms of comorbidity rates and poorer Global Assessment of Functioning. Given the detrimental physical and psychological correlates related to pediatric anorexia nervosa, further research with this specific population will be vital in mitigating the negative conditions and outcomes related to this disorder.

Pediatric Anorexia Nervosa and Special Considerations

Clinicians and researchers alike have noted the difficulties associated with diagnosis and treatment of anorexia nervosa within the pediatric population. Robin, Gilroy, and Dennis (1998) assert that children and adolescents with anorexia nervosa may present in a somewhat different fashion than adults with anorexia nervosa due to distinct medical and psychological developmental factors. For instance, children and adolescents may not experience the traditional weight loss associated with anorexia nervosa. Rather, they may present with failed

expected weight gain, which is determined by the child's history of height and weight percentiles, anticipated growth trajectory, and average weights and heights of same age/sex peers. Moreover, variable menstrual cycles immediately following the onset of menarche can be difficult to discern from amenorrhea in the pediatric population, and the presence of anorexia nervosa during a prepubertal period can delay the onset of menstruation, preventing an individual from meeting the criterion of amenorrhea. As such, Robin and colleagues contend that many children and adolescents may present below their healthy weight range and experience cognitive symptoms related to anorexia nervosa during a prepubertal period but technically fail to meet DSM-IV diagnostic criteria.

In addition to the aforementioned physical developmental concerns, Robin et al. (1998) delineate psychological developmental considerations as well. For instance, the fear of gaining weight, body image concerns, and cognitive distortions related to anorexia nervosa are highly related to one's ability to engage in abstract thought, which typically does not develop until adolescence. As such, younger children may experience difficulty verbalizing such concerns and may not fully understand the psychological underpinnings of their illness. Given their developmental level, children and younger adolescents may therefore express eating-related concerns in more concrete terms or may manifest such concerns primarily in their behavior, such a food rituals, eating slow, and hiding food (Jaffe & Singer, 1989). Given the aforementioned factors related to pediatric anorexia

nervosa, Robin et al. contend that DSM-IV diagnostic criteria may not be sensitive in detecting some cases of pediatric anorexia nervosa, resulting in a diagnosis of eating disorder not otherwise specified.

Etiology

The etiology of anorexia nervosa appears to be quite complicated and multifactorial in nature. Although a myriad of theories have been proposed regarding the development of anorexia nervosa, the most compelling theory to researchers and clinicians alike appears to be a biopsychosocial diathesis-stress model. Although a comprehensive description of the biopsychosocial etiology of anorexia nervosa cannot be covered within the scope of this literature review, a brief overview of sociocultural, familial, and psychological factors will be provided. Over the past two decades, much focus has been on identifying sociocultural factors related to the etiology of eating disorders. However, in recent years more attention has been dedicated to exploring the etiology of anorexia nervosa in terms of family functioning and individual psychological characteristics. Although all etiological components of anorexia nervosa cannot be definitively identified, numerous studies have successfully identified important characteristics of this population in relation to the multifaceted etiology.

Sociocultural Influences

The larger societal backdrop of individual development plays an important role in the development of eating disorders. According to Polivy and Herman (2002), the incidence of eating disorders is higher in societies characterized by flourishing economies in which a paucity of food is of little concern. However, of all the sociocultural influences studied to date, it appears that the media has been implicated as one of the larger influences in the widespread idealization of unrealistically thin body images among females (Polivy & Herman, 2002; Tozzi, Sullivan, Fear, McKenzie, & Bulik, 2003). Additionally, several studies have underscored the powerful effect of peer influence with regard to disordered eating patterns and attitudes, especially among children and adolescents. However, it is difficult, if not impossible, to discern whether peer groups influence the individual, or whether a group of individuals who are independently vulnerable to eating disorders are drawn to each other (Polivy & Herman, 2002).

The Role of the Family

The role of the family has been studied in relation to numerous psychiatric disorders given the genetic and environmental influence it exerts. As such, several research projects have investigated the nature of family functioning within the

context of eating disorders. Minuchin, Rosman, and Baker (1978) provided one of the earliest descriptions of anorexia nervosa and family functioning, describing such families as more enmeshed, rigid, and conflict avoidant. Since this hallmark publication, a myriad of studies have examined the nature of family functioning and anorexia nervosa, with some results failing to support the relationship between pathological family functioning and anorexia nervosa (Rastam & Gillberg, 1991). For instance, Waller and colleagues (1989, 1990) found that families of anorectics report more global family dysfunction than control families, while other studies have found the opposite to be true (North, Gowers, & Byram, 1995).

Communication. Studies of communication have implicated poor conflict resolution as a hallmark characteristic of families who have a child with anorexia nervosa. According to Botta and Dumlao (2002) communication and conflict resolution are inextricably related because "communication behavior often creates conflict, reflects conflict, and is the vehicle for managing conflict" (p. 200). Families of anorexic patients tend to avoid conflict and encourage conformity and obedience, perhaps in effort to increase a sense of family cohesion and stability (Kog & Vandereycken, 1989). As such, anorexic patients have been noted to be more submissive (Botta & Dumlao, 2002) and to struggle with communicating feelings as a whole, especially overt self-expressions such as conflict (Humphrey,

1989; Latzer, Hochdorf, Bachar, & Canetti, 2002). The avoidance of open communication coupled with lack of long-term and satisfying conflict resolution may engender feelings of powerlessness, ineffectiveness, and negative self-esteem, making one more vulnerable to restrictive eating habits in attempts to rectify such feelings (Botta & Dumlao, 2002).

Despite the finding that the general climate of family communication may be one of conflict avoidance, other studies investigating dyadic communication have found that communication styles within the family can vary a great deal depending on who is involved in the communication (Botta & Dumlao, 2002). For instance, one parent may encourage open communication and healthy conflict resolution more so than the other. Lattimore, Wagner, and Gowers (2000) observed that mother-daughter dyads of anorexic teens tended to engage in more destructive communication when compared to a group of psychiatric controls during a series of problem-solving tasks. For instance, mothers and anorexic daughters engaged in more frequent blame, criticism, disagreement, mind-reading, and negative affect. Conversely, their interaction was characterized by a lack of agreement and positive affect. Although these results support previous findings that communication is generally unhealthy in families of children and adolescents diagnosed with anorexia nervosa, they do not confirm previous findings that these families are “conflict avoidant”.

Similarly, patterns of interaction between eating disordered patients and their fathers revealed destructive communication marked by a high degree of negative affect, belittling, and blaming by fathers (Humphrey, 1989). Other investigations of dyadic communication have suggested that relationships with fathers have a stronger relationship with daughters' self-esteem than relationships with mothers (Gecas & Schwalbe, 1986). In 2002, Botta and Dumlao surveyed 210 undergraduate female students with the aim of elucidating the relationship between eating disordered behaviors and father-daughter communication. They found that open communication and effective conflict resolution between fathers and daughters are related to less eating pathology, underscoring yet again the importance of paternal relationships with children. As such, healthy family communication and effective conflict resolution, especially where fathers are involved, may serve as protective factors and offset the degree of eating disordered symptoms and severity (Fonseca, Ireland, & Resnick, 2002).

Boundaries and enmeshment. Several studies of family functioning have cited interpersonal boundary dissolution and enmeshment, characterized by lack of differentiation and individuation among family members, as important variables in the development and maintenance of anorexia nervosa (Minuchin, 1978; Rowa, Kerig, & Geller, 2001). While some researchers contend that boundary problems and enmeshment are not unique to anorexia nervosa, and that

these are familial characteristics associated with other psychiatric disorders and adjustment problems of childhood (Fisher et al., 1995; Fullinwider-Bush & Jacobvitz, 1993; Rowa, Kerig, & Geller, 2001), other studies have suggested that boundary dissolution is a defining characteristic of anorexia nervosa (Humphrey, 1989; Kog & Vandereycken, 1989). For instance, Rowa and colleagues (2001) investigated the nature of intergenerational boundaries in a sample of 30 women with anorexia nervosa and compared them to 65 control women from an undergraduate psychology course. They administered the Parent-Child Boundaries Scale and found that women with anorexia nervosa reported more boundary problems than non-eating disordered women.

Additionally, the role of parental bonding has been studied in relation to family functioning and difficulties with boundary maintenance and enmeshment. Although definitive results have not emerged with respect to parental bonding, some studies have found that anorectic patients describe their parents as more rejecting and neglecting than controls (Humphrey, 1989), which is contradictory to the portrait of enmeshment reported by Minuchin and colleagues. However, other studies have found that parents of anorectic patients tend to be more overprotective (Walters & Kendler, 1995). Calam, Waller, Slade, and Newton (1990) conducted one of the earlier studies evaluating the relationship between eating pathology and parental bonding with a group of British women. They compared 31 anorexics, 34 bulimics with a history of anorexia nervosa, 33

bulimics without a history of anorexia nervosa, and 242 community volunteers. The Parental Bonding Instrument was utilized to generate separate maternal and paternal scores on "care", which is a measure of warmth, empathy, and emotional support; as well as "protection", which measures intrusiveness, control, and overprotection. Calam and colleagues found that females with eating disorders reported their mothers and fathers as less caring and warm, and their fathers as overprotective, which is commensurate with other studies (Palmer, Oppengeimer, & Marshall, 1988; Steiger, J., Goldstein, & Leichner, 1989).

In 2000, Shoebridge and Gowers engaged in further investigation of the nature between maternal overprotection and childhood anorexia nervosa. Through retrospective interviewing and medical record reviews, Shoebridge and Gowers found that mothers of anorexic daughters tended to be more protective and concerned during their child's early life when compared to a normal control group. As such, maternal and paternal overprotection may engender enmeshed and highly dependent relationships within the family, which inhibits a child's ability to develop a healthy sense of self-efficacy, autonomy, and independence (G. Waller, Slade, & Calam, 1990).

Perception of family functioning. In exploring the relationship between family functioning and anorexia nervosa, researchers and clinicians have found that perception of family functioning may differ from member to member

(Guttman & Laporte, 2002). For instance, several studies have found that patients tend to report more family dysfunction than their parents (S. Gowers & North, 1999; North, Gowers, & Byram, 1997; G. Waller, Slade, & Calam, 1990). For instance, North and colleagues (1997) found that female adolescents rated their families as less healthy than their mothers. Additionally, they found that adolescents' perceptions and clinician rated perceptions of overall family functioning at the beginning of inpatient treatment were commensurate with each other and predictive of 2-year outcome. Other studies have yielded similar results, supporting the notion that perception of family functioning is not always commensurate between patients and parents. For instance, a study conducted by Gowers and North (1999) found that adolescent patients and clinicians rated family functioning more stringently than parents, suggesting that patients are more insightful regarding family functioning.

Despite studies supporting the notion patients and parents disagree regarding the quality of their family functioning, other studies have found that parent and patient perceptions are positively correlated (Guttman & Laporte, 2002; McDermott, Batik, Roberts, & Gibbon, 2002). In 1995, North, Gowers, and Byram found that patients and parents reported healthy family functioning while clinicians rated them unhealthy. Guttman and Laporte have hypothesized that similarities in patient and parent self-report of family functioning may be

indicative of enmeshed family relations, specifically when such perceptions are incongruent with clinician rated family functioning.

Psychological Characteristics

Individual psychological factors, such as personality, temperament, attitudes, and core beliefs have been studied in relation to anorexia nervosa for several decades. Anorectic individuals have been noted to be more pessimistic, insecure, anxious, resistant to change, harm avoidant, and rigid when compared to others (Fassino et al., 2002; Latzer, Hochdorf, Bachar, & Canetti, 2002); making them more susceptible to "internalizing" comorbid disorders, such as depression, anxiety, and substance abuse (Latzer, Hochdorf, Bachar, & Canetti, 2002). Many researchers as well as clinicians have hypothesized that such psychological characteristics are engendered by lack of encouragement toward personal growth within the family and surmounting social pressure regarding appearance and body type (Fassino et al., 2002; Latzer, Hochdorf, Bachar, & Canetti, 2002). Although many of these characteristics have been found to be common among individuals diagnosed with other psychiatric disorders, studies have found evidence supporting the strong presence of these characteristics in individuals diagnosed with anorexia nervosa.

For instance, several studies have found that anorexic individuals tend to have lower self-esteem and negative self-concept when compared to normal controls (Cooper & Turner, 2000). A study conducted by Jacobi, Paul, de Zwaan, Nutzinger, and Dahme (2004) investigated self-concept disturbances among a group of eating disordered, anxiety disordered, depressive disordered, and healthy individuals. Individuals diagnosed with eating disorders reported lower self-esteem and more prominent feelings of ineffectiveness when compared to healthy controls, even after controlling for depression. Additionally, low self-esteem and feelings of ineffectiveness were more prominent in eating disordered individuals, specifically those diagnosed with anorexia nervosa, when compared to psychiatric controls. However, differences between psychiatric controls and eating disordered individuals were not as marked as those found between eating disordered individuals and healthy controls (Jacobi, Paul, de Zwaan, Nutzinger, & Dahme, 2004).

Additionally, disturbances in body image and concerns about weight and shape have been identified as a core component to the psychopathology of anorexia nervosa. In 2000, Cooper and Turner administered the Eating Disorder Belief Questionnaire and the Eating Attitudes Test to a group of anorexics, dieters, and female controls to investigate differences in assumptions and beliefs among the groups. Results of this study support that anorexic individuals endorse more concerns about weight, shape, and eating when compared to healthy

controls and a group of dieters. Other studies have yielded commensurate results regarding the cognitive content and beliefs of individuals diagnosed with anorexia nervosa (De Panfilis, Rabbaglio, Rossi, Zita, & Maggini, 2003; Marshall, Palmer, & Stretch, 1993). For instance, anorexic individuals have been found to endorse a high degree of body dissatisfaction, drive for thinness, and eating disordered attitudes as measured by the Eating Disorder Inventory and Eating Attitudes Test (Marshall, Palmer, & Stretch, 1993).

Treatment Outcomes and Prognosis of Children and Adolescents Diagnosed with Anorexia Nervosa

Course of Illness and Remission

The course and outcome of eating disorders, and anorexia nervosa in particular, have only recently been studied in depth. To date several studies have found that the outcome and prognosis for anorexia nervosa is the poorest and most chronic when compared to bulimia nervosa and eating disorder not otherwise specified. For instance, Herzog, Dorer, Keel, Selwyn, Ekelad, Flores, et al. (1999) followed a group of anorexic and bulimic patients over a 7.5 year period and found that full recovery was much higher for individuals diagnosed with bulimia nervosa when compared to those with anorexia nervosa. At two-year

follow-up, only 8% of restrictors and 13% of binge-purgers had reached full recovery at some point. Although the rate of full recovery increased over the duration of the study, only 33% of those with anorexia nervosa achieved full recovery during the seven year study, as opposed the 74% recovery rate among bulimic subjects. Given this marked difference in recovery, it comes as no surprise that having a diagnosis of anorexia nervosa was the strongest predictor of poor outcome in this sample. Although Herzog and colleagues were unable to identify predictors of relapse, findings indicated that within the anorexic sample, 40% of the 83.7% who achieved full recovery eventually relapsed during the study.

A similar 7-year study of outcome was conducted by Bryant-Waugh, Knibbs, Fosson, Kaminski, and Lask (1988) with a pediatric sample. They found that outcome over a 7-year period was poor in terms of psychological and/or physical functioning for one-half to one-third of the patients, with over 25% requiring readmission to treatment facilities after discharge. A subsequent study by Bryant-Waugh, Cooper, Taylor, and Lask (1996) followed 22 anorexic children over a 2 to 5-year period. Results from this more recent study suggest that longer-term outcome for children diagnosed with anorexia nervosa are poor, with only 55.5% of the participants achieving good outcome. Bryant-Waugh and colleagues (1996) did not identify any indicators of outcome over a 3-year period, as was the case in the earlier 7-year study. Although reported relapse rates vary,

recent studies suggest that between 30% and 50% in adolescents who have been treated for an eating disorder relapse within three years of treatment (Norring & Sohlberg, 1993; Pike, 1998; Strober, Freeman, & Morrell, 1997). Strober and colleagues also found that recovery of adolescents treated for anorexia nervosa was generally protracted over a 10 to 15 year follow-up period, with partial and full recovery occurring in less than 10% of cases in the immediate two years following treatment. Although 76% of this sample eventually met criteria for full recovery, 30% of these patients relapsed after a period of recovery.

The time course of recovery and symptom remission is a factor that is inextricably linked to the study of treatment outcome. A plethora of studies have suggested that physical and psychological symptoms of anorexia nervosa do not remit simultaneously. Some clinicians and researchers have hypothesized that the high rate of relapse, specifically following acute inpatient treatment, may be due in part to the lack of psychological recovery accompanying physical recovery during acute phases of treatment (Fennig, Fennig, & Roe, 2002). Clausen (2004) studied a group of eating disordered patients over a two and a half year period following outpatient treatment. Results from this study indicated that physical symptoms were the first to remit, followed by behavioral symptoms, and lastly psychological symptoms. In efforts to address this pattern of recovery, several treatment facilities, including Children's Medical Center of Dallas, have

developed continuum of care programs to adapt to changing treatment needs of recovering individuals.

Limitations of the Current Outcome Literature

Although outcome literature of eating disorders has increased in recent years, a clear and consistent understanding of treatment outcome has not emerged for a number of reasons. In her 1998 paper, Pike cites several methodological issues that make generalizability across various outcome studies difficult. Perhaps the most important factor in such studies is the variability in the definition of outcome utilized across studies. A more consistently utilized definition of outcome is the Morgan-Russell Criteria, which categorizes outcome on three levels according to physical recovery: good, intermediate, and poor. However, Pike contends that this criterion does not account for behavioral and psychological aspects of eating disorders, which may lead to an inaccurate classification of outcome. Despite the wide-spread use of the Morgan-Russell Criteria, no benchmark for measuring treatment outcomes has been well established, leading to the use of several different definitions of outcome. For instance, some studies have defined treatment outcome as the absence of symptoms for a minimum of eight weeks, while others have used the number of re-hospitalizations as means for classifying outcome.

Although comprehensive assessment of physical, psychological, and behavioral symptoms appear quit appropriate for measuring long-term outcomes, such criteria are inappropriate for measuring acute, short-term outcome (Treat, 2005). As mentioned previously, the course of symptom remission is typically protracted over a number of months and years. As such, criteria for classifying acute treatment outcome must be modified to reflect the stage of symptom remission expected. In her 1998 article, Pike begins to delineate criteria for outcome over a short-term period. Such criteria are as follows: weight status, eating behavior, menstrual functioning, and medical stabilization.

There also appears to be a large range in the length of follow-up periods utilized within the outcome literature. Follow-up periods within the literature range from three months to 20 years, with variable points of measurement in between. Additionally, there appears to be a dearth of literature focused on acute treatment outcomes following intensive inpatient treatment. Most studies of outcome and anorexia nervosa have followed patients during and after a period of outpatient treatment, while neglecting to study the more severe cases involved in inpatient treatment. Moreover, studies that do include inpatient participants often study them with outpatients as a single homogenous group, despite the differing levels of illness severity and treatment needs characteristic of these very different groups. Other studies have also investigated different types of eating disorders as a single group, with no differentiation between diagnoses, such as anorexia and

bulimia nervosa. Although studying anorexia nervosa and bulimia nervosa as a single group may appear logical and compelling in terms of increased sample size and power within a study, doing so may be inappropriate given the differences in clinical presentation and outcome characteristic of the two groups.

Outcome and Weight

Low body weight is a core clinical feature of anorexia nervosa and has been linked to treatment outcome in numerous studies. A three-year follow-up study of children and adolescents diagnosed with anorexia nervosa revealed that low percent body weight at admission to an intensive outpatient treatment facility was related to higher rates of subsequent relapse (D. A. Waller, Mugan, M. N., Moreshed, T., Setnick, J., Cummings, M., Hynan, L. S., 2003). Additionally, it appears that even patients who recover from their illness continue to maintain a body mass index below that of the general population (Tozzi, Sullivan, Fear, McKenzie, & Bulik, 2003).

Meanwhile, other studies have found that weight and body mass index at the time of referral is a strong predictor of treatment outcome. In 1993, Herzog, Sacks, Keller, Lavori, Ranson, and Gray followed 225 eating disordered women every 3 months over the course of one year to assess the course and outcome of their eating disorders. Of this sample, 41 met DSM-III-R criteria for anorexia

nervosa and had a mean age of 24.9 years. During this one year period, only 10% of those diagnosed with anorexia nervosa achieved a full recovery (defined as an asymptomatic state for 8 consecutive weeks), while 56% of the bulimics reached full recovery. At the completion of the study, Herzog and colleagues found percent ideal body weight at the beginning of the study to be the best predictor of outcome among the anorexics, such that those with lower percent ideal body weight with a diagnosis of anorexia had the worst outcomes at one year.

Studies within the pediatric population have also supported the predictive power of intake body weight in relation to treatment outcomes. A study conducted by Gowers, Weetman, Shore, Hossain, and Elvins (2000) followed 75 cases of adolescent-onset anorexia nervosa from an outpatient treatment facility and assessed these participants at 2 and 7 year follow-up. Criteria for good outcome were established as weight equal to or above 85% ideal body weight, resumption of menstrual cycle, and satisfactory psychosocial functioning. Results indicated that percent body weight and Morgan-Russell Outcome Assessment Schedule at intake were predictive of 2 and 7-year outcome, and that lower body weights were predictive of a longer period to recovery.

Outcome and Eating Behavior

Various aspects of eating behavior have been studied in relation to treatment outcome, with purging being one of the most widely studied behaviors related to eating disorders. Several studies have suggested that the presence of purging a negative treatment indicator, although this finding has not always been consistent. For instance, Gowers et al. (2000) did not find that the presence of purging was predictive of good or poor outcome over a 7 year-period among adolescents diagnosed with anorexia nervosa. Additional studies have investigated the nature of a variety other eating behaviors as they relate to treatment outcome. A study conducted by Commerford, Licinio, and Halmi (1997) followed-up with 31 adolescent eating disordered patients by telephone 5-years after discharge from an inpatient treatment facility to assess outcome. Commerford and colleagues found that patients who were able to successfully choose their own foods, eat at home during passes, and refrain from purging at the time of discharge were more likely to report a higher percent ideal body weight at the time of follow-up and were less likely to report a relapse of illness. Although numerous behaviors, such as purging and hoarding food, have been linked to the development and maintenance of eating disorders, there continues to be a paucity of research exploring such behaviors within the context of treatment outcomes.

Outcome and Comorbid Psychopathology

The literature investigating treatment outcome and comorbid psychopathology has yielded inconsistent results. In 1999, Herzog et al. found that adult anorectics with a comorbid diagnosis of major depressive disorder achieved partial recovery at a more rapid pace. However, in an earlier study, Herzog and colleagues (1993) found that the presence of comorbid Axis I diagnoses at intake were not predictive of outcome. Conversely, some studies of pediatric anorexia nervosa and outcome have revealed that poor prognosis was more likely to occur in conjunction with a variety of factors, comorbid depression being one of them. Furthermore, a three-year follow-up study of children and adolescents diagnosed with anorexia nervosa found that low percent average body weight and comorbid depression at admission to an intensive outpatient treatment facility was related to higher rates of relapse (D. A. Waller, Mugan, M. N., Moreshed, T., Setnick, J., Cummings, M., Hynan, L. S., 2003). As such, comorbid depression within the adult population may be related to more rapid recovery. However, research within the pediatric population suggests that comorbid depression may be a negative treatment indicator.

Outcome and Age of Onset

Various studies have examined age of onset as a variable of treatment outcome; however, findings are inconclusive. While some studies have found that earlier age of onset coupled with early treatment is related to better outcome, other studies have not found this to be true (S. G. Gowers, Weetman, Shore, Hossain, & Elvins, 2000; Herzog et al., 1993). For instance, a study conducted by Strober et al. (1997) followed a group of adolescents diagnosed with anorexia nervosa over a 10-15 year period and found that early age of onset was not related to treatment outcome. However, an earlier study conducted by Bryant-Waugh et al. (1988) found that poor outcome was more likely to occur for those with an age of onset below 11 years. Conversely, other studies have found that participants who were younger at the time of inpatient treatment were more likely to have a better outcome, as they were able to maintain a higher percent body weight (Commerford, 1997). Commerford and colleagues hypothesize that this finding may be due to the parental role of reinforcing and ensuring that patients adhere to treatment recommendations.

Outcome and Anorectic Cognitions and Attitudes

It appears that much of the focus within the outcome literature has been on illness variables that are easily quantifiable and operationalized, such as body mass index. However, a core clinical feature of anorexia nervosa lies within the cognitive content and attitudes espoused by individuals affected by the disorder. While the attention has shifted in recent years to the cognitive content of anorexics, it does not appear that cognitions and core beliefs have been studied extensively in relation to treatment outcomes. Meanwhile, it appears that the outcome literature has recently begun to examine eating disordered attitudes in relation to treatment outcomes.

For instance, Castro, Toro, and Cruz (2000) followed 108 outpatient adolescent females between the ages of 12 and 18 years who met DSM-IV criteria for anorexia nervosa. Participants completed the Eating Attitudes Test (EAT—26) and the Egna Minnen Betraffande Uppfostram: 'My Memories of Upbringing' questionnaire at the beginning of treatment. Good and poor outcome was determined by the following criteria: no more than one hospital admission and achievement and maintenance of at least 90% ideal body weight. After two years of treatment, Castro and colleagues found that the EAT—26 total score was an independent predictor of treatment response. A subsequent study conducted by Castro, Gila, Puig, Rodriguez, and Toro (2004) followed-up with 101 inpatient

adolescents diagnosed with anorexia nervosa one year after initial inpatient treatment. They found that abnormal eating attitudes (measured by the EAT—26 total score) were predictive of rehospitalization after a period of full weight restoration, as well as young age at admission (less than 15 years) and slow rate of weight gain during treatment. In addition to the predictive utility of abnormal eating attitudes toward general treatment outcome, other studies have found that a period of inpatient hospitalization fosters significant improvement in abnormal eating attitudes (Lowe, Davis, Annunziato, & Lucks, 2003).

Outcome and Family Functioning

General family functioning. Although a great deal of research has begun to explore the family dynamics of individuals diagnosed with anorexia nervosa, relatively little research has explored the relationship between family functioning and treatment outcomes. To date, research within this area has yielded inconsistent results, with some studies suggesting that family functioning is one of the strongest indicators of short-term outcome within the adolescent population. For instance, numerous studies have suggested that troubled family relations and hostile attitudes toward family members at the time of treatment intake are correlated with, and may predict longer periods to recovery and poor outcome (Morgan & Russell, 1975; Strober, Freeman, & Morrell, 1997). Additionally,

other studies have found that children and adolescents with anorexia nervosa who come from non-traditional family structures, such as single parent families and blended families, are more likely to experience poor prognosis when compared to those from more traditional family structures (Bryant-Waugh, Knibbs, Fosson, Kaminski, & Lask, 1988). This particular finding may be influenced by a myriad of factors, such as increased stress within the family and a perceived lack of control within the family. In 1997, North, Gowers, and Byram found that female adolescents rated their overall family functioning as less healthy than their mothers, and that adolescent and clinician-rated perceptions of overall family functioning at the beginning of inpatient treatment were predictive of 2-year outcome.

Parental factors. Castro, Toro, and Cruz (2000) investigated the relationship between perceptions of parents and short-term outcome in a sample of 108 outpatient adolescent females between the ages of 12 and 18 years who met DSM-IV criteria for anorexia nervosa. Good versus poor outcome was determined by the following criteria: no more than one hospital admission and achievement and maintenance of at least 90% of their ideal body weight. After two years of treatment, Castro and colleagues found that patients with poor outcome reported more parental rejection and control-overprotection from both of

their parents when compared to those with good outcome. Additionally, perceived paternal rejection was predictive of poor treatment outcome.

Other research has focused on elucidating the nature of familial communication in relation to treatment outcome. For instance, van Furth, van Strien, Martina, van Son, Hendrick, and van Engeland (1996) examined expressed emotion ratings of parents with eating disordered adolescents being treated in a variety of inpatient and outpatient facilities. Van Furth and colleagues found that mothers' Critical Comments ratings accounted for 28% to 34% of the variance in immediate and follow-up outcome. Furthermore, this rating was the best predictor of outcome when compared to traditional outcome measures of diagnosis, duration of illness, body weight, body mass index, age of onset, and gender.

Improvement in family functioning. Some research projects have focused on elucidating the nature of change in family functioning over the course of treatment. Wallin and Kronvall (2002) followed 26 families of adolescent anorexic females who had been admitted for treatment to an eating disorders program on an inpatient or outpatient basis. Family therapy was provided for two years, after which time outcome was determined by the Global Clinical Rating Scale, which accounts for social functioning, psychiatric functioning, and eating disordered symptoms. Based on clinician observations, these families had improved on dimensions of competency, cohesion, adaptability, enmeshment, and

rigidity. Moreover, self-reported ratings of family functioning yielded improved expressiveness. However, Wallin and Kronvall did not detect any differences between those who had recovered and those who had not recovered based on baseline observational ratings of family functioning. Conversely, they did find that the recovered group reported less enmeshment and more competence at the completion of the study than those who had failed to recover.

However, other studies have found that perceptions and ratings of family functioning do not improve over the course of treatment. Gowers and North (1999) examined the relationship between severity of illness in adolescent anorexic individuals and correlates in perception of family functioning. They found that improvement in clinical condition was not accompanied by improvements in self-reported family functioning over a one year period, suggesting that perception of family functioning does not improve over the course of short-term treatment. Additionally, Gowers and North found that as the severity of illness increased, (as measured by the Morgan-Russell Schedule) families tended to report less family dysfunction, suggesting that family dysfunction may not precede or become heightened by illness severity. However, this finding is in contrast to those of other research projects, necessitating further investigation of family functioning in relation to illness severity and treatment outcome.

CHAPTER THREE

RATIONALE, AIMS, AND HYPOTHESES

Rationale and Aims

Although the treatment outcome literature on pediatric anorexia nervosa has increased in recent years, studies have yielded variable results. Some studies have found that outcome is generally poor, characterized by a chronic course of illness and high relapse rates, while other studies have found that outcome may be better in certain cases. For instance, several studies have found that higher body weight at admission to treatment, fewer familial disturbances, and healthier psychological functioning are predictive of good outcome. Although there appears to be a relationship between treatment outcomes and baseline factors related to family functioning and specific eating disorder symptoms, a clear and consistent relationship has not emerged for a number of reasons. Additionally, much focus has been on evaluating short-term and long-term outcomes of anorexia nervosa, with little attention toward acute treatment outcomes despite evidence suggesting that those who require hospitalization have the poorest outcomes (S. G. Gowers, Weetman, Shore, Hossain, & Elvins, 2000). As such, little is known about what aspects of family functioning and eating disordered symptomatology are related to

or predict outcome following a period of acute and intensive treatment, or what cognitive and attitudinal symptoms improve.

Given the lack of research within this area, the current study aims to do the following:

1. The primary aim of this study is to identify aspects of family functioning, anorectic cognitions, and eating attitudes at admission to inpatient or partial hospitalization that predict acute treatment outcome at discharge within a pediatric population.
2. The secondary aim of this study is to evaluate what aspects of family functioning, anorectic cognitions, and eating attitudes improve over the course of inpatient or partial hospitalization for pediatric anorexia nervosa.

Questions and Hypotheses

Predictors of Outcome

Research Question One: Do family functioning, eating disordered cognitions, and eating attitudes at admission to an intensive eating disorder program predict outcome at discharge?

Hypothesis One: Poor family functioning at intake (as measured by standardized clinical observation on the Timberlawn Couple and Family Evaluation Scale [TCFES], parent Self-Report Family Inventory—2 [SFI—2], and patient SFI—2) will predict poor outcome at discharge, as measured by three separate indicators of outcome: 1) discharge body mass index [BMI]; 2) improvement of BMI at discharge of at least one standard deviation from baseline (indicating positive outcome); and 3) the Clinical Global Impressions Scale global improvement [CGI-I] at discharge.

Note: Standardized clinician-rated observation of family functioning will be measured by the TCFES's sum of scales score and the following TCFES domain scores: structure, autonomy, problem-solving, affect regulation, and conflict. Additionally, patient and parent self-reported family functioning will be measured by the following SFI—2 scales: health competence, cohesion, conflict, leadership, and expressiveness.

Hypothesis Two: The presence of more eating disordered cognitions at intake (as measured by the Mizes Anorectic Cognitions Scale—Revised [MAC-R] total score, self-control subscale, rigid weight regulation subscale, and weight and approval subscale) will predict poor outcome at discharge (as measured by the same three separate indicators of outcome described above).

Hypothesis Three: The presence of more eating disordered attitudes at intake (measured by the Eating Attitudes Test—26 [EAT—26] total score, dieting subscale, bulimia and food preoccupation subscale, and oral control subscale) will predict poor outcome at discharge (as measured by the same three separate indicators of outcome described above).

Research Question Two: Do family functioning, eating disordered cognitions, and eating disordered attitudes at admission to an intensive eating disorder program contribute non-overlapping variance to the prediction of outcome at discharge?

Hypothesis Four: Aspects of family functioning, eating-disordered cognitions, and eating attitudes at intake that were predictive of treatment outcome will contribute independently to treatment outcome (as measured by the three separate indicators of outcome described above).

Cognitive and Attitudinal Symptom Improvement

Research Question Three: Do family functioning, eating-disordered cognitions, and eating attitudes at admission improve over the course of treatment?

Hypothesis Five: Family functioning (as measured by clinical observation on the TCFES, parent SFI—2, and patient SFI—2) will improve over the course of hospitalization.

Note: Family functioning will be measured in the same fashion explicated above: Standardized clinician-rated observation of family functioning will be measured by the TCFES's sum of scales score and the following TCFES domain scores: structure, autonomy, problem-solving, affect regulation, and conflict. Additionally, patient and parent self-reported family functioning will be measured by the following SFI—2 scales: health competence, cohesion, conflict, leadership, and expressiveness.

Hypothesis Six: Eating disordered cognitions (as measured by the MAC—R total score, self-control subscale, rigid weight regulation subscale, and weight and approval subscale) will improve over the course of hospitalization.

Hypothesis Seven: Eating disordered attitudes (as measured by the EAT—26 total score, dieting subscale, bulimia and food preoccupation subscale, and oral control subscale) will improve over the course of hospitalization.

CHAPTER FOUR

METHODOLOGY

Participants

Participants for this study were taken from the University of Texas Southwestern Medical Center at Dallas' IRB approved study entitled "Family Functioning in Children and Adolescents with Eating Disorders". A total of 43 families enrolled in the study, and 35 of these 43 agreed to complete at least part of the follow-up protocol at discharge (a complete discussion of participant adherence to study protocol and study non-completers is provided in Chapter Five). All patients enrolled in the study were admitted to inpatient or partial hospitalization for treatment of anorexia nervosa or eating disorder not otherwise specified (with below healthy weight, i.e., body mass index [BMI] below 18.5) at Children's Medical Center of Dallas.

Inclusion Criteria

1. Participants must have been between the ages of 8 and 18 years of age at the time of hospital admission.

2. Participants must have been living with at least one parent or legal guardian who has been the child's primary caregiver for a minimum of one year.
3. Participants must have had at least one primary caregiver willing to participate in the study with his or her child.
4. Participants must have had a primary DSM-IV diagnosis of anorexia nervosa or eating disorder not otherwise specified with below healthy weight at admission to the unit. Patients who had comorbid diagnoses (e.g. non-psychotic depression, anxiety, obsessive compulsive disorder, etc) were not excluded from the study, so long as the primary reason for treatment was an eating disorder.

Exclusion Criteria

1. Patients who had a diagnosis of a psychotic disorder were excluded from this study.
2. Patients were excluded from the study if they had below normal intellectual functioning (i.e. IQ lower than 80 based on the history given by the primary caregiver, medical chart notes, observation, or WISC-IV if a concern about intellectual capacities arose during clinical assessment).

Design and Procedure

Participants were recruited by direct solicitation upon admission to inpatient or partial hospitalization at the psychiatry unit of Children's Medical Center of Dallas. A research assistant attended the intake interview or contacted the primary caregiver at the time of admission to solicit consent. Informed consent was obtained from patients and their caregivers prior to the collection of any data and included an explanation regarding the purpose, procedures, possible risks and benefits, and confidentiality related to the study. Participants were not monetarily compensated for their participation in this research project, and their treatment was not altered in any manner as a result of their participation. Participants were informed of the alternatives to participation in this study and were given the opportunity to ask questions before consenting or assenting to participate in this research study.

At the time of informed consent, the research associate ensured that all aspects of the study were fully understood by all participants and explained the study in developmentally appropriate terms to the patient. Once patients and their caregivers voluntarily chose to participate in the study, they were asked to sign the informed consent form (See Appendix A) and the HIPPA authorization form (See Appendix B), which detailed instances in which the participants' protected health information may be disclosed. Once these forms were signed, copies were

provided to the primary caregiver/guardian and placed in the medical chart. Additionally, the following demographic and illness variables were obtained from the medical record: patient age, date of birth, ethnicity, date of admission, height, and weight.

The primary caregiver was then asked to complete the Self-Report of Family Functioning—Second Edition (SFI-II); while the patient was asked to complete the SFI-II, Eating Attitudes Test (EAT-26), and the Mizes Anorectic Cognitions Scale—Revised (MAC-R). These measures were completed by the primary caregiver and patient within one week of admission to the unit. At the time of admission, a trained research associate also interviewed the patient and parent/legal guardian separately to complete the Schedule for Affective Disorders and Schizophrenia for School Aged Children, Present and Lifetime Version (K-SADS-PL) (Klein, 1993), a semi-structured DSM-IV based clinical interview, in order to obtain valid psychiatric diagnoses.

Families (consisting of at least one parent and the patient) also participated in the Timberlawn Couple and Family Evaluation Scale (TCFES) video taping within one week of admission. During this video taping, families were presented with the following prompts via audiotape and asked to discuss each for eight minutes: 1) discuss as a family what is strong about your family; 2) discuss the major source of disagreement in your family; and 3) plan a family activity that involves all of you and takes at least an hour to do. These tapes were

subsequently rated by research associates who have been trained according to the *Timberlawn Couple and Family Evaluation Scales: A Rater Training Guide*. Additionally, all raters obtained direct training by an expert on the TCFES, and interrater reliability coefficients were calculated for independent raters on available training tapes. Once the raters were trained and adequate interrater reliability was established (a kappa of .80 or higher), tapes for this study were rated. Every 5th tape was double rated to ensure rater drift did not occur. After each tape was double rated, interrater reliability was calculated. When adequate interrater reliability (kappa $\geq$ .80) was not found, raters discussed the rationale behind their ratings together, and a training tape was independently rated and discussed according to rating criteria for additional training. This rating and discussion process was repeated until adequate interrater reliability was reestablished. Additionally, the primary rater for each tape was not blind, as this individual had administered the K-SADS-PL and worked with the family throughout the process of data collection. However, the individual providing double ratings was blind to the family and had not worked with the family in any capacity.

All research patients participated in the standard treatment program offered by the CMC psychiatry unit (which is the treatment all patients receive, regardless of their decision to participate in this research project). All patients were placed on an individualized meal plan and expected to consume an

increasing amount of calories over the course of their treatment in order to restore their physical health and weight. Family members also participated in meal education sessions, during which they learned skills for supporting the patient during mealtimes and practiced utilizing these skill on the unit with the patient. Additionally, all patients participated in individual therapy and family therapy twice a week. Although a standard therapeutic protocol was not utilized by the therapists, a combination of cognitive-behavioral, supportive, and insight-oriented techniques were utilized according to the individual needs of patients. Patients and their families were also encouraged to express their thoughts and feelings to each other in appropriate ways, as well as to identify different methods for ensuring that patients could ask for and receive support within the family system. In addition to individual and family therapy, patients participated in a daily support group, coping skills group, and nutrition group. They also attended an ED process group twice a week.

Within one week of discharge, all self-report measures were re-administered to the primary caregiver and patient, and the family (consisting of at least one caregiver and the patient) participated in the TCFES taping a second time. Additionally, the date of discharge, height, and weight were recorded from the discharge summary of the medical record. All data obtained (including self-report measures, clinician-rated measures, and TCFES tapes) was stored in a locked file cabinet within a locked room at the UT Southwestern Research Center

for Pediatric Psychiatry. Data was removed from the locked cabinet for entry into a confidential database and immediately returned to the cabinet after data entry. All data was double-checked to ensure accuracy prior to data analysis.

Materials and Measures

Clinician Rated Measures

a. The Schedule for Affective Disorders and Schizophrenia for School-aged Children- Present and Lifetime Versions (KSADS-PL; Kaufman et al., 1997) is an updated version of the K-SADS (Chambers et al., 1985) and uses DSM-IV criteria to assess present episode and lifetime history of psychiatric illness in children and adolescents between the ages of 6 and 17 years. It is a semi-structured parent-child integrated clinical interview that utilizes an 82-symptom screen portion. To address differential diagnosis, it includes the following five supplemental sections: 1) affective disorders, 2) psychotic disorders, 3) anxiety disorders, 4) behavioral disorders, and 5) substance abuse, eating disorders, and tic disorders. Data from parents and children are collected separately, and responses are recorded on the same answer sheet

by the same clinician to allow for a comparison of responses. The data from parents and children are synthesized based on the interviewer's clinical judgment in order to generate DSM-IV Axis I diagnoses. The K-SADS-PL uses a 0-3 point rating scale and provides global and diagnostic-specific impairment ratings. Convergent validity with the Beck Depression Rating Scale has been found to be .90 and with the Children's Depression Rating Scale it is .89 (Ambrosini, 2000). Test-retest reliability has been established as ranging from .63 for attention deficit disorder to .90 for major depression (Ambrosini, 2000). Additionally, interrater reliability has been established as .8 (Ambrosini, 2000).

b. The Clinical Global Impressions Scale (CGI) (National Institute of Mental Health, 1985) is used as a clinician assessment of overall symptom severity and improvement, each with a seven point scale, with lower values denoting more favorable and healthy outcomes. At intake, only severity of illness is rated. However, in subsequent assessments, both severity and improvement can be rated. This is a standard scale utilized within treatment outcome research, and a CGI improvement (CGI-I) of 1 (very much) or 2 (much) is considered to be an acceptable response to treatment within the

treatment outcome literature. As such, CGI-I scores for the current study were converted to categorical data accordingly (i.e., scores of 1 and 2 denoting good treatment response/outcome, and scores from 3 to 7 denoting poor treatment response/outcome). The CGI was developed during the PRB collaborative schizophrenia studies. The items on the CGI are considered universal, and are therefore appropriate for use in pediatric as well as adult populations. The intraclass correlation coefficient for CGI improvement as a continuous variable in the above study was 0.93 and .95 as a categorical variable. For the current study, CGI-I scores were provided by two independent raters: the clinical psychologist (rater 1) and psychiatrist (rater 2) of the psychiatry unit at CMC. Interrater reliability based upon simple proportion of agreement between rater 1 and rater 2 on the CGI-I was good, with 77% agreement between the two expert raters. However, when chance agreement was accounted for by Cohen's Kappa (K), the interrater reliability figure was poor (K=.51). As such, each rater's CGI-I scores were used as independent and separate indicators of outcome to explore systematic differences between their ratings in relation to potential predictors of outcome in the present study.

c. Timberlawn Couple and Family Evaluation Scales (TCFES) (Lewis, Gossett, Housson, & Owen, 1999) is a revision of the Beavers Timberlawn Family Evaluation, and is a standardized clinician rated observational measure which consists of 18 scales that measure competence in the larger domains of system structure, autonomy, affect regulation, conflict, and problem-solving (see table below). Families and couples are prompted via audiotape to discuss strengths in their family, sources of disagreement in their family, and to plan a family activity. These three discussions take eight minutes a piece and are video recorded so that family interactions can be coded by trained clinicians or researchers. Recent reliability and validity studies on the TCFES provide support for its use with clinical populations (Lewis, Gossett, Housson, & Owen, 1999). For instance, reliabilities for the family summary scores ranged from .83 to 87; the five a priori family domains ranged from .74 to .85; and reliabilities of the family interaction individual scales ranged from .57 to .85, with a median reliability of .71. Family scales significantly distinguished between clinical and nonclinical samples [Sum of Scales ($F(1,72) = 14.75$, $p < , .001$] . Detailed procedures for training raters and establishing interrater reliability for the present study are explicated in the

Design and Procedure section above. Intraclass correlations between the primary rater (who was not blind to the family) and the double rater (who was blind to the family) for the five domains of family functioning, Sum of Scales, and Global Competence ranged from .80 to .94, denoting excellent interrater reliability on the TCFES ratings of this study. A summary of intraclass correlations for the TCFES is provided in Appendix C, Table 1.

TCFES Domains and Subscales:

Structure • Overt Power • Adult Leadership • Inappropriate Parent-Child Coalition • Closeness	**Affect Regulation** • Expressiveness • Responsiveness • Positive Regard • Negative Regard • Mood and Tone • Empathy
Autonomy • Clarity of Expression • Respect for Subjective Reality • Responsibility	**Disagreement/Conflict** • Frequency • Affective Quality • Generalization and Escalation
Problem Solving • Closure • Negotiation	**Global Competence**

Self-Report Measures of Family Functioning

d. Self-Report Family Inventory-2 (SFI-2) (Hampson, Beavers, & Hulgus, 1989) is a 36-item self-report instrument that evaluates family members' perspectives of the domains of health competence, conflict, cohesion, directive leadership, and emotional expressiveness. The health competence subscale includes nineteen content items involving family affect, parental coalitions, problem-solving abilities, autonomy and individuality, optimistic versus pessimistic views, and acceptance of family members. The conflict subscale includes 12 content items dealing with overt versus covert conflict, including arguing, blaming, fighting openly, acceptance of responsibility, unresolved conflict and negative affect/tone. The cohesion subscale includes five content items involving family togetherness, satisfaction received from inside the family versus outside the family, and spending family time together. The leadership subscale includes three content items involving parental leadership, directiveness, and the degree of rigidity of control. The emotional expressiveness subscale includes six content items dealing with verbal and nonverbal expression of warmth, caring and closeness (Hampson, Beavers, &

Hulgus, 1989). The scale is designed for family members 11 years of age and older, and all items with the exception of the last two (overall family rating and family independence rating) are answered on the following likert scale: 1- "Yes: Fits our family well;" 3- "Some: Fits our family some;" and 5- "No: Does not fit our family." Participants of the current study were instructed to rate aspects of family functioning based upon their perceptions at intake and to re-rate their perceptions once again at discharge in order to assess changes and similarities in perceptions over the course of treatment. Internal consistency for the SFI-II has been assessed at a Cronbach's alpha of 86. Test-retest reliability coefficients (for 30 to 90 days) range from .84 to .87 for family health/competence, .50 to .59 for conflict, .50 to .70 for cohesion, .79 to .89 for expressiveness, and .41 to .49 for directive leadership. Additionally, the SFI has demonstrated adequate concurrent validity through high correlations with other family self report instruments (Hampson, et al, 1989). For example, the SFI health/competence scale correlates at r=.87 with the general functioning factor of the McMaster Family Assessment Device (Wood, Waller, Miller, & Slade, 1992), and the SFI cohesion

subscale correlates r=-.82with the cohesion scale from the FACES III (Olson, 1982).

Health Competence	• family affect, • parental coalitions, • problem-solving abilities, • autonomy and individuality, • optimistic versus pessimistic views • acceptance of family members
Conflict	• overt versus covert conflict (arguing, blaming, fighting openly, acceptance of responsibility, unresolved conflict, and negative feeling tone)
Cohesion	• family togetherness, • satisfaction received from inside the family versus outside, • spending time together
Directive Leadership	• parental leadership • directiveness • degree of rigidity and control
Emotional Expressiveness	• verbal and nonverbal expression of warmth, caring and closeness

Self-Report Measures of Eating Disordered Cognitions and Attitudes

e. The Mizes Anorectic Cognition Scale—Revised (MAC-R) (Mizes, 1994) is an updated revision of the original Mizes Anorectic Cognitions Scale, and is a self-report questionnaire that assesses cognitions related to both anorexia and bulimia nervosa. The MAC-R consists of 33 items from the original MAC and 24 new questions, equaling a total of 57 items. The MAC-R examines

three specific areas related to cognitions: rigid weight and eating regulation, weight and eating behavior as the basis of approval from others, and excessive self-control as a component of self-esteem (Mizes, 1990, 1992). The MAC-R is written at a sixth grade level and is suitable for persons in middle school through adulthood. Internal consistency for the MAC-R has been assessed at a Chronbach's alpha of .90 for the total score, .84 for the self-control scale, .85 for the weight and approval scale, and .82 for the rigid weight regulation scale. Concurrent validity was also demonstrated on the MAC-R and is significantly correlated with the EDI total score (derived by summing all the EDI subscales) and with the EDI Restraint scale. More specifically, the EDI-2 summary score and MAC-R total score were significantly correlated (r=.69, p=.00), and the restraint scale was also significantly correlated with the MAC-R total score (r=.62, p=.00), self-control scale (r=.70, p=.00), weight and approval scale (r=.43, p=.00), and rigid weight regulation scale (r=.40, p=.00) (Mizes et al., 2000).

f. Eating Attitudes Test (EAT-26) (Garner, Olmsted, Bohr, & Garfinkel, 1982) is a 26-item self-report measure of symptoms and

concerns characteristic of eating disorders. The EAT-26 is widely used as a screening tool for eating disorders and has been found to be sensitive to identifying eating disorders as well as partial syndrome eating disorders. Additionally, its use has been validated in the adolescent population (Wood, Waller, Miller, & Slade, 1992). Individuals report their agreement with statements concerning attitudes of weight food based on the following 6-point likert scale: 1-always, 2-usually, 3-often, 4-sometimes, 5-rarely, and 6 never. The EAT-26 yields a total score, as well as three subscales consisting of dieting, bulimia and food preoccupation, and oral control. The dieting subscale assesses avoidance of high calorie foods and concerns with being thinner. Additionally, the bulimia and food preoccupation subscale assesses obsessive thoughts about food, as well as tendencies to binge and purge. Finally, the oral control subscale evaluates the degree of restraint one engages in to restrict food intake and perceived feedback from others to gain weight. Internal consistency of the EAT has been reported to range from .79 to .94. Additionally, while Garner and Garfinkel did not report test-retest reliability coefficients, the children's version of the EAT has been reported to have a test-retest reliability of .81 (Allison, 1995).

Objective Measures

g. Body Mass Index (BMI) is an objective mathematical indication of relative body weight based on an individual's weight and height. BMI is calculated by dividing an individual's weight in kilograms by the individual's height in meters squared. A BMI below 18.5 is considered underweight.

CHAPTER FIVE

RESULTS

Screening of Outcome Variables

The following were initially identified as outcome variables of interest: discharge BMI, improvement of BMI from intake to discharge (improvement of at least one standard deviation denoting good outcome), and CGI-I ratings. As explicated in the preceding interrater reliability section, both CGI-I raters' scores were regarded as independent indicators of outcome due to poor interrater reliability. To ensure that all intended measures of outcome were indeed independent constructs which measure unique aspects of outcome (i.e., did not overlap significantly with each other), chi square tests for independence were run between dichotomous outcome variables, and independent t-tests were carried out between discharge BMI and dichotomous outcome variables. A Bonferroni correction was applied to repeated independent t-tests to control for Type I error. As seen in Appendix C, Tables 2-4, all variables of outcome were independent of each other. Therefore, these four variables (CGI-I rater 1, CGI-I rater 2, BMI improvement of at least one standard deviation, and discharge BMI) were included, as planned, in analyses of treatment outcome as indicators of outcome. Appendix C, Table 5 provides frequency and percent rates for outcome (improved

versus not improved) based upon the three categorical indicators of outcome (CGI-I rater 1, CGI-I rater 2, and improvement in BMI of at least one standard deviation).

Descriptive Statistics

Demographic and Illness Variables

The total sample consisted of 43 patients (41 inpatients and 2 partial hospitalization patients) and their families. The two subjects admitted to partial hospitalization were compared to the rest of the sample to determine if they presented as outliers on demographic and illness variables. However, these two cases did not present as outliers to the overall sample and, therefore, were not excluded from the sample for subsequent analyses.

A summary of demographic and illness variables (age at admission, intake BMI, discharge BMI, and length of treatment) is provided in Appendix C, Table 6. Patients ranged in age from 10 to 17 years, with a mean age of 14.21 years (SD=1.73). As expected, the mean BMI was much lower at admission (M=15.64, SD=1.38) when compared to discharge (M=17.58, SD=1.27), and BMI improved significantly over the course of hospitalization, $t(42)=-10.06$, $p<.001$. Additionally, the average length of hospitalization was 28.75 days (SD=13.92).

Approximately eighty-eight percent of the sample was female (n= 38), while the rest were male (n=5). This sample also consisted primarily of Caucasians, with only 2 African-Americans, 4 Latinas, and 1 Asian (Appendix C, Table 7). Additionally, the majority of the sample consisted of restricting anorexics (n=30), with far fewer purging anorexics (n=5) and individuals with ED NOS (n=8). This sample was also characterized by a high rate of comorbid psychopathology in addition to ED, which is consistent with comorbidity rates depicted in the literature. Specifically, 69.77% of the sample met criteria for a depressive disorder and 27.91% met criteria for an anxiety disorder. Frequency tables detailing specific DSM-IV diagnoses are provided in Appendix C, Tables 8 and 9. Although this sample appears largely homogeneous in terms of demographic variables, these characteristics are congruent with other studies which have investigated ED samples. Please see Appendix G for tables summarizing correlations among study dependent variables.

Study Completers versus Non-Completers

Although 43 families consented to participate in this study, several participants did not complete all components of the study. However, as seen in Appendix F, Table 1, many families did not cite a specific reason for discontinuing or limiting their participation throughout course of the study. A

comparison of study completers versus non-completers was performed to assess any systematic differences between the two groups. For the purpose of these analyses, study completers were defined as families who participated in the TCFES taping at intake and discharge. The TCFES was chosen as the criterion for these analyses due to the unique nature of the data it provides (i.e., clinical observations), as opposed to the myriad of self-report measures utilized for this study. According to this criterion, the attrition rate of participants from intake to discharge during the course of this study was 26.8%, with 11 families failing to complete data collection at discharge. However, two families were not included in these analyses because they refused to participate in the TCFES at intake and discharge, citing discomfort with the nature of the task and being video taped as their reason for refusal. As seen in Appendix F, Tables 2-9, study completers did not differ significantly from study non-completers by chi squares and independent *t*-tests in terms of demographic variables (p=.30 to .97), outcome variables (p=.14 to .87), or baseline characteristics (p=.08 to .98).

Given that there was additional missing data not captured by the above analyses, study completers versus non-completers were also defined in the following manners for exploratory purposes: 1) missing *all* discharge data (attrition rate of 18.6%), and 2) missing *any* discharge data (attrition rate of 51.16%). When completers versus non-completers were compared to each other

based upon these criteria, there continued to be no significant differences between the groups in relation to demographic, outcome, and baseline characteristics.

Measures of Family Functioning

All measures of family functioning were compared to non-clinical, normative samples to assess relative levels of pathology. On the TCFES, one sample *t*-tests were conducted to compare the ED intake mean scores on the subscales, sum of scales, and global competence to the mean scores of a group of 28 non-clinical families who participated in the development of the scale (Housson, 1996). Domain scores of the ED sample could not be compared to a non-clinical sample, as mean domain scores have not been published for any non-clinical samples to date. A Bonferroni correction was applied to these multiple one sample *t*-tests to control for Type I error, resulting in the following adjusted alpha levels: $p<.0025$ for significance at the .05 alpha level and $p<.0005$ for significance at the .01 alpha level. Results revealed that the non-clinical sample was significantly healthier than the ED sample on the following subscales: Responsibility, Expressiveness, Responsiveness, and Mood and Tone. However, the ED sample was rated as healthier than the non-clinical sample on the following scales: Negative Regard, Conflict Frequency, Conflict Affective Quality, and Conflict Generalization/Escalation. Appendix D, Table 1 contains a

summary of these results, and Appendix C, Table 10 contains descriptive statistics for the TCFES domains, sum of scales, and global competence on all available ED data at intake and discharge.

Mean scores on patient and parent SFI-II subscales were compared by one sample *t*-tests to the mean scores of a non-clinical normative group published by Hampson, Beavers, & Hulgus (1989). A Bonferroni correction was applied to these multiple one sample *t*-tests to control for Type I error, resulting in the following adjusted alpha levels: $p<.01$ for significance at the .05 alpha level and $p<.002$ for significance at the .01 alpha level. ED patients reported significantly more family dysfunction on the SFI-II subscale of Leadership than children and adolescents in the healthiest group of the normative sample. However, there were no differences between the ED group and the healthiest non-clinical group by child/adolescent self-reports on the four remaining subscales. Additionally, when compared to the least healthy group of the normative sample, patients in the ED group rated their families significantly healthier on all SFI-II subscales.

Comparisons between parent ratings of the normative sample and ED sample were similar to the child/adolescent ratings described above. When compared to the healthiest normative sample, ED parents did not rate their families as significantly healthier or unhealthier. However, when compared to the least healthy normative sample, parents in the ED group rated their families as significantly healthier. Appendix D, Table 2 contains a summary of these results,

and Appendix C, Tables 11 and 12 contain descriptive statistics at intake and discharge for all available patient and parent SFI-II subscales.

Measures of Eating Cognitions and Attitudes

Mean EAT-26 scores of the ED sample were compared by one sample *t*-tests to the mean scores of a non-clinical adolescent female group, aged 11-16 years (Wood, Waller, Miller, & Slade, 1992). Bonferroni corrections were employed to control for Type I error and indicated that $p<.0025$ was needed for significance at the .01 alpha level. As seen in Appendix D, Table 3, all EAT-26 subscales were significantly higher in the ED sample than the non-clinical group, such that the ED group endorsed unhealthier eating attitudes. (Please refer to Appendix C, Table 13 for descriptive statistics at intake and discharge for all available EAT-26 subscales.)

Given the absence of adolescent normative data on the MAC-R within the literature, the total score of the ED sample was compared to a non-clinical sample of 290 undergraduates by a one sample *t*-test (Osman, Chiros, Gutierrez, Kopper, & Barrios, 2001). The remaining MAC-R subscales could not be compared to a non-clinical sample, as no normative data on these subscales have been published. As seen in Appendix D, Table 4, the ED sample reported significantly more anorectic cognitions than the non-clinical sample ($p<.001$). Please refer to

Appendix C, Table 14 for a summary of descriptive statistics at intake and discharge for all available MAC-R data.

Predictors of Outcome

Inferential statistics were utilized to explore and analyze the study hypotheses. A combination of chi squares, *t*-tests, and regressions were utilized for this purpose. Data were screened to ensure that the assumptions of these statistics were fulfilled. Additionally, power analyses were conducted to determine the sample size needed to detect significant findings for hypotheses in which no significance was yielded. As explicated above, four indicators of outcome were utilized to explore potential predictors of outcome. These indicators were: 1) discharge BMI (as a continuous variable), 2) improvement in BMI by at least one standard deviation at discharge (as a categorical variable of "good" versus "poor" outcome), 3) CGI-I clinical psychologist ratings/CGI-I rater 1 (as a categorical variable of "good" versus "poor" outcome), and 4) CGI-I psychiatrist ratings/CGI-I rater 2 (as a categorical variable of "good" versus "poor" outcome).

Hypothesis One

Poor family functioning at intake (as measured by standardized clinical observation on the Timberlawn Couple and Family Evaluation Scale [TCFES], parent Self-Report Family Inventory—2 [SFI—II], and patient SFI—II) will predict poor outcome at discharge, as measured by four separate indicators of outcome. Standardized clinician-rated observation of family functioning was measured by the TCFES's sum of scales score and the following TCFES domain scores: Structure, Autonomy, Problem-Solving, Affect Regulation, and Conflict. Additionally, patient and parent self-report of family functioning were measured by the following SFI—II scales: Health Competence, Cohesion, Conflict, Leadership, and Expressiveness.

Potential predictors of outcome were identified by correlations and independent *t*-tests between the indicators of outcome and baseline variables of family functioning. The bivariate relationship between discharge BMI and the specified variables of family functioning at baseline were assessed by Pearson Product Moment correlation coefficients. Additionally, the bivariate relationship between the three categorical indicators of outcome (Improvement in BMI by at least 1 SD, CGI-I rater 1, and CGI-I rater 2) and variables of family functioning were assessed by independent *t*-tests. Bonferroni corrections were applied to these

analyses to control for Type I error, resulting in the following adjusted alpha levels for significance at the .05 level: TCFES=$p<.007$, SFI-II=$p<.01$.

As seen in Appendix C, Tables 15-17, variables of family functioning were not significantly correlated with discharge BMI. Similarly, variables of family functioning did not differ between those categorized as having a poor outcome versus a good outcome based upon improvement in BMI by at least one SD (Appendix C, Tables 18 and 19) or CGI-I (Rater 1) (Appendix C, Tables 20 and 21). Additionally, standardized clinician-rated family functioning on the TCFES and patient reports of family functioning on the SFI-II did not differ between those with poor versus good outcome based upon the CGI-I (Rater 2) (Appendix C, Tables 22 and 23). However, parent ratings on several subscales of the SFI-II differed significantly after Bonferroni corrections between those categorized as having good outcome versus poor outcome on the CGI-I by rater 2 (Appendix C, Table 23). More specifically, parents of patients with good outcome reported greater familial Health Competence, $t(40)=-2.96$, $p<.05$, greater familial Cohesion, $t(40)=-2.84$, $p<.05$, and less Conflict, $t(40)=-3.15$, $p<.05$.

Prior to conducting a logistic regression to examine the predictive power of family functioning by parent report on outcome determined by the CGI-I (Rater 2), variables were assessed for multicollinearity, and potential covariates were screened. To assess for multicollinearity, correlations were conducted among potential predictor variables of parent reported Health Competence, Cohesion,

and Conflict on the SFI-II. These analyses yielded significant correlations among the predictor variables, $p<.001$ (Appendix C, Table 24). Given this extreme degree of multicollinearity, it was determined that each variable would not contribute unique variance in predicting outcome, as these variables are highly related with one another. Theoretically, it appears that Health Competence assesses family functioning in a much broader sense than Cohesion and Conflict on the SFI-II. Therefore, parent reported Health Competence was the only potential predictor variable to be included in the logistic regression with CGI-I (Rater 2).

The relationship between demographic variables (admit BMI, LOT, and age) and outcome based upon the CGI-I (Rater 2) was explored by independent sample *t*-tests to identify potential covariates for the logistic regression. Theoretically speaking, it was anticipated that intake BMI and LOT would be significantly related to outcome. As one would expect, intake BMI and LOT were negatively correlated, $r(41) = -.36$, $p < .017$, such that individuals with lower intake BMI's had longer hospital stays. However, intake BMI and LOT were not significantly related to CGI-I (Rater 2) outcome, $t\ (39) = 9.17$, $p = .37$ and $t\ (39) = -2.78$, $p = .78$, respectively. Unexpectedly, age was significantly related to CGI-I (Rater 2), $t\ (39) = -2.47$, $p = .02$, such that younger patients tended to have better outcome than older patients. Therefore, patient age was controlled for in the logistic regression between CGI-I (Rater 2) and parent reported Health

Competence on the SFI-II. As seen in Appendix C, Table 25, parent reported Health Competence on the SFI-II significantly predicted patient outcome (good versus poor) based upon the CGI-I (Rater 2), β=1.15, Wald(1) = 4.68, p = .03.

Hypothesis Two

The presence of more eating disordered cognitions at intake (as measured by the Mizes Anorectic Cognitions Scale—Revised [MAC-R] total score, self-control subscale, rigid weight regulation subscale, and weight and approval subscale) will predict poor outcome at discharge (as measured by the same four separate indicators of outcome described above).

The bivariate relationship between discharge BMI and the MAC-R subscale scores at baseline were assessed by Pearson Product Moment correlation coefficients to identify potential predictors of outcome. Additionally, the bivariate relationship between the three categorical indicators of outcome (improvement in BMI by at least 1 SD, CGI-I rater 1, and CGI-I rater 2) and MAC-R subscales were assessed by independent *t*-tests to identify potential predictors as well. Bonferroni corrections were applied to these analyses to control for Type I error, resulting in an adjusted alpha level of $p<.0125$ for significance at the .05 level.

As seen in Appendix C, Table 26, MAC-R subscale scores were not significantly correlated with discharge BMI. Similarly, MAC-R subscales did not

differ between those categorized as having a poor outcome versus a good outcome based upon improvement in BMI by at least one SD or either rater's CGI-I ratings (Appendix C, Tables 27-29). Therefore, no potential predictor variables of outcome emerged from these analyses with intake MAC-R scores.

Hypothesis Three

The presence of eating disordered attitudes at intake (measured by the Eating Attitudes Test—26 [EAT—26] total score, dieting subscale, bulimia and food preoccupation subscale, and oral control subscale) will predict poor outcome at discharge (as measured by the same four separate indicators of outcome described above).

The relationship between potential predictors of outcome on the EAT-26 and indicators of outcome were assessed by correlations and independent *t*-tests. The bivariate relationship between discharge BMI and the EAT-26 subscale scores a baseline were assessed by Pearson Product Moment correlation coefficients. Additionally, the bivariate relationship between the three categorical indicators of outcome (Improvement in BMI by at least 1 SD, CGI-I rater 1, and CGI-I rater 2) and EAT-26 subscales were assessed by independent *t*-tests. Bonferroni corrections were applied to these analyses to control for Type I error, resulting in an adjusted alpha level of $p<.0125$ for significance at the .05 level.

As seen in Appendix C, Table 30, EAT-26 subscale scores were not significantly correlated with discharge BMI. Similarly, EAT-26 subscales did not differ between those categorized as having a poor outcome versus a good outcome based upon improvement in BMI by at least one SD or either rater's CGI-I ratings (Appendix C, Tables 31-33). Therefore, no viable potential predictors of outcome emerged from these analyses with intake EAT-26 scores.

Hypothesis Four

Aspects of family functioning, eating-disordered cognitions, and eating attitudes at intake that were predictive of treatment outcome will contribute independently to treatment outcome (as measured by the four separate indicators of outcome described above). The aim of this hypothesis was to determine whether family functioning, eating disordered cognitions, and eating disordered attitudes at admission to an intensive eating disorder treatment facility contributed non-overlapping variance to the prediction of outcome at discharge. However, only one variable (parent reported familial Health Competence on the SFI-II) was found to be predictive of outcome in the preceding set of analyses. Therefore, an insufficient number of predictor variables were identified in order to investigate this hypothesis.

Power Analyses

Given the limited findings regarding predictors of acute treatment outcome, power analyses were conducted to determine the sample sizes needed for a power of .80 at an alpha level of .01 to approximate the adjusted alpha levels imposed by Bonferroni corrections. As one would expect, results varied a great deal among the different analyses. Therefore, the following results pertain to the smallest sample sizes needed on each measure (for which significant predictors were not identified) to detect predictors of outcome.

Based on the current TCFES data, a minimum sample size of 100 is needed for a power of .80, which would potentially yield significant predictors of outcome based upon improvement in BMI by at least one standard deviation. Additionally, a sample size of 58 would be required on the patient SFI-II form to achieve a power of .80 for identification of predictors of outcome based upon the CGI-I (Rater 2). Finally, sample sizes of 66 on the MAC-R and 70 on the EAT-26 are needed to obtain a power of .80 to identify potential indicators of outcome based upon improvement in BMI by at least one standard deviation.

Improvement in Family Functioning and Psychological Symptoms

Hypothesis Five

Family functioning (as measured by clinical observation on the TCFES, parent SFI—II, and patient SFI—II) will improve over the course of hospitalization. Paired samples *t*-tests were conducted to evaluate whether clinical observations of family functioning on the TCFES improved over the course of acute hospitalization. Bonferroni corrections were applied to these multiple *t*-tests to control for Type I error, resulting in an adjusted alpha level of $p<.007$ for significance at the .05 level. As seen in Appendix C, Table 34, there were no significant changes or improvements in standardized clinician-rated family functioning during the course of acute treatment, with *p*-values ranging from .14 to .99.

Paired samples *t*-tests were also conducted on patient and parent SFI-II subscale scores to assess change from intake to discharge in self-reported family functioning. After Bonferroni corrections were applied to control for Type I error, a p-value$<.01$ was needed to detect significant change at the .05 alpha level. With regard to patient self-report, Expressiveness was the only scale to improve significantly from intake (M=1.88, SD=.73) to discharge (M=1.61, SD=.67), $t(27)=2.91$, $p<.05$. However, similar improvements over the course of treatment

were not observed on the parent SFI-II, as *p*-values ranged from .38 to .89. A summary of these results are provided in Appendix C, Tables 35 and 36.

As noted in the descriptive statistics section, ED intake Expressiveness scores on the patient SFI-II were healthier than the "least healthy" normative group and commensurate with Expressiveness scores reported by the "healthiest" normative group. As such, an additional analysis was conducted to compare the mean ED Expressiveness score at discharge to the mean of the "healthiest" non-clinical sample in order to determine whether the improved score inflated beyond the mean of the healthy normative group. However, a one-sample *t*-test revealed no significant difference between the mean of the ED group at discharge (M=1.62, SD=.67) and the mean of the "healthiest" non-clinical normative group (M=1.80), $t(27)=-1.53$, $p=.14$.

Hypothesis Six

Eating disordered cognitions (as measured by the MAC—R total score, self-control subscale, rigid weight regulation subscale, and weight and approval subscale) will improve over the course of hospitalization. Paired samples *t*-tests were conducted on intake and discharge scores of the four MAC-R scales to assess change over the course of acute treatment. A Bonferroni correction was applied to these analyses to control for Type I error, resulting in an adjusted alpha

level of .013 for significance at the .05 level. As seen in Appendix C, Table 37, there were no significant improvements in eating disordered cognitions over the course of acute treatment, with *p*-values ranging from .08 to .59.

Hypothesis Seven

Eating disordered attitudes (as measured by the EAT—26 total score, dieting subscale, bulimia and food preoccupation subscale, and oral control subscale) will improve over the course of hospitalization. Paired samples *t*-tests were conducted on intake and discharge scores of the EAT-26. A Bonferroni correction was applied to these analyses to control for Type I error, resulting in adjusted alpha levels of .013 for significance at the .05 level and .003 for significance at the .01 level. Results yielded significant improvement in eating attitudes on the Oral Control subscale from intake (M=9.37, SD=4.14) to discharge (M=5.67, SD=4.60), $t(26)=4.78$, $p<.01$, as well as improvement on the Bulimia and Food Preoccupation subscale from intake (M=5.59, SD=3.39) to discharge (M=4.11, SD=3.71), $t(26)=2.86$, $p<.05$. Additionally, significant improvement was noted on the Total Score of the EAT-26 from intake (M=31.74, SD=16.47) to discharge (M=22.56, SD=18.35), $t(26)=3.54$, $p<.01$. However, no significant change was noted on the Dieting subscale after Bonferroni corrections, although *p*-values appear to approach significance at the .05 level.

As noted in the preceding descriptive statistics section, all intake EAT-26 subscales were significantly unhealthier than those of a non-clinical adolescent group. As such, additional analyses were conducted to compare improved discharge EAT-26 subscales (Oral Control, Bulimia and Food Preoccupation, and Total Score) to the mean of the non-clinical group to determine if they remained significantly more unhealthy in comparison, despite their improvement. Although these three scales improved significantly over the course of acute treatment, one-sample *t*-tests revealed that the ED sample continued to endorse significantly more unhealthy eating attitudes than the non-clinical group on the Oral Control subscale, $t(27)=4.50$, $p<.001$; the Bulimia and Food Preoccupation subscale, $t(27)=5.16$, $p<.001$; and the Total Score, $t(27)=4.41$, $p<.001$.

Power Analyses

Admittedly, the preceding results were likely limited by the small sample size and power of the current study. As such, power analyses were conducted to determine the sample sizes needed for a power of .80 at an alpha level of .01 (to approximate adjusted alpha levels due to Bonferroni corrections) for detecting significant changes over the course of treatment. As one would expect, results varied a great deal among the different analyses. Therefore, the following results

pertain to the smallest sample sizes needed to detect significant differences on measures in which no significant change occurred.

Based upon the current TCFES data, a sample size of 89 pairs would be needed in order to achieve a power of .80. Additionally, one would need approximately 134 participants to fill out the Parent SFI-II form at intake and discharge in order to obtain adequate power. As Expressiveness was the only subscale to improve significantly on the SFI-II by patient self-report, power analyses were conducted to determine the sample size needed in order to detect significant improvement on additional subscales. According to these analyses, approximately 112 patients would need to complete the SFI-II form at intake and discharge for adequate power. Finally, with regard to the MAC-R, a sample size of 62 pairs would be needed in order to obtain a power of .80.

Exploratory Analyses

Given the limited findings pertaining to predictors of acute treatment outcome, exploratory analyses were conducted to investigate two additional, unplanned indicators of outcome: median split of percent BMI gain and discharge EAT-26 total scores. For the purpose of the first exploratory analysis, good versus poor outcome was based upon a median split of percent gain in BMI over the course of treatment. Independent *t*-tests were conducted to evaluate baseline

characteristics of family functioning, eating cognitions, and eating attitudes as potential predictors of outcome based upon this additional indicator of outcome. Additionally, Bonferroni corrections were applied to these analyses in order to control for Type I error. As seen in Appendix E, Tables 1-4, outcome based upon a median split of percent BMI gain was not significantly related to any baseline measures of family functioning, eating disordered cognitions, or eating attitudes. Therefore, no potential indicators of outcome emerged from these analyses.

As mentioned previously, the Oral Control subscale, Bulimia and Food Preoccupation subscale, and Total Score of the EAT-26 improved significantly over the course of acute hospitalization. As such, exploratory analyses were conducted to evaluate whether aspects of family functioning at intake (measured by the TCFES, patient SFI-II, and parent SFI II) or eating cognitions at intake predicted outcome at discharge, as defined by eating attitudes. For the purpose of these analyses, the total score of the EAT-26 was utilized as the indicator of outcome, as both Oral Control and Bulimia and Food Preoccupation load onto the total score, and thus, were highly correlated with the total score (r=.88 and r=.89, respectively). In order to account for individual differences on intake total scores of the EAT-26, a percent change on EAT-26 total scores from intake to discharge was utilized (rather than raw EAT-26 total scores at discharge). Given that decreases in EAT-26 scores over the course of treatment signified improvements

in eating attitudes, higher negative change scores were more desirable and indicative of improvement.

Pearson Product Moment correlations were conducted between the percent change in total score on the EAT-26 and baseline measures of eating cognitions and family functioning. Bonferroni corrections were applied to these correlations to control for Type I error, resulting in the following adjusted alpha levels for significance at the .05 level: MAC-R=.0125, TCFES=.007, SFI-II=.01. As seen in Appendix E, Tables 5 and 6, patient reported Expressiveness on the SFI-II was the only baseline variable of family functioning that was significantly correlated with the percent change in EAT-26 total score, $r(24)=.58$, $p<.05$. Additionally, no aspects of eating cognitions at intake were significantly related to percent change in EAT-26 total score (Appendix E, Table 7).

Prior to regressing patient reported Expressiveness on percent change in EAT-26 total score by linear regression, the relationships between percent change in EAT-26 total score and baseline demographic and illness variables were explored in order to identify potential covariates. Pearson Product Moment correlation coefficients were conducted between percent change in the EAT-26 total score at discharge and the following variables: age, admit BMI, length of treatment, percent BMI gain, and duration of ED episode. Additionally, the relationship between the percent change in EAT-26 total score and the following variables were analyzed by independent samples *t*-tests: Ethnicity, ED diagnosis,

gender, and previous ED treatment. As seen in Appendix E, Tables 8 and 9, no potential covariates were identified, as no demographic or illness variables were significantly related to the change in pathology of eating attitudes. When patient reported Expressiveness at intake was regressed on percent change in EAT-26 total score, healthier Expressiveness scores predicted better outcome (i.e., higher, negative percent change in EAT-26 total scores), R^2=.34, F (1, 24)= 12.20, p<.01 (Appendix E, Table 10).

Further exploratory analyses were also conducted by defining good versus poor outcome based upon a median split of percent change in EAT-26 total score. The relationships between outcome and intake variables of eating cognitions and family functioning were explored by independent samples *t*-tests. Additionally, the relationship between outcome and demographic/illness variables was assessed. Chi square analyses were conducted between outcome and the following variables: ethnicity, ED diagnosis, gender, and previous ED treatment. Independent samples *t*-tests were conducted between outcome and the following variables: age, length of treatment, admit BMI, percent weight gain over the course of treatment, and duration of ED episode. As seen in Appendix E, Tables 11-15, none of these variables were significantly related to outcome based upon a median split of percent change in EAT-26 total score. As such, no variables of family functioning or eating cognitions were identified as predictors of outcome from these analyses.

CHAPTER SIX

DISCUSSION

Overview of the Study

The present study was designed to systematically evaluate various aspects of acute treatment outcome within a sample of children and adolescents hospitalized for anorexia nervosa and subthreshhold anorexia nervosa. The current ED literature is unfortunately characterized by inconsistent results regarding predictors of treatment outcome, as well as the course of illness and recovery. Although there appears to be a relationship between treatment outcome and baseline factors related to family functioning and the severity of psychological symptoms, a clear and consistent relationship has not yet emerged. Additionally, the existing literature appears to focus on the evaluation of short and long-term outcomes in heterogeneous samples of patients participating in different levels of care (e.g., outpatient, intensive outpatient, day treatment, and inpatient). As such, the purpose of the current study was to evaluate acute or immediate treatment outcomes within a homogeneous sample of severely ill individuals who have been hospitalized for treatment.

The primary aim of the present study was to identify aspects of family functioning and psychological symptoms (ED related cognitions and attitudes)

that predict acute treatment outcome. A secondary aim of this study was to assess improvement in family functioning and psychological symptoms over the course of acute hospitalization. Anorectic cognitions and eating attitudes were assessed by patient self-report (MAC-R and EAT-26), and the nature of family functioning was assessed by multisource and multimethod data. Specifically, aspects of family functioning were examined by a standardized clinician-rated, observational measure of family functioning (TCFES), as well as patient and parent self-report (SFI-II). Outcome was also defined by the following multisource and multimethod data: weight restoration (BMI) and clinician ratings of improvement by the CMC psychiatry unit clinical psychologist and psychiatrist (CGI-I, Rater 1 and Rater 2). Multisource and multimethod data was utilized in the present study to better account for the complex nature of family functioning and various aspects of treatment outcome.

Family Functioning as a Predictor of Outcome

The first hypothesis posited that the presence of poor family functioning upon admission to treatment would predict poor outcome at discharge, as defined by weight restoration and clinical judgment by two independent raters on the CGI-I. Although the family dynamics of individuals diagnosed with AN have been an area of great interest over the past few decades, relatively little research

has explored the relationship between family functioning and treatment outcome. Despite the paucity of research within this specific area, some studies have suggested that the nature of family functioning might be one of the strongest indicators of short-term outcome within the adolescent population (Morgan & Russell, 1975; North, Gowers, & Byram, 1997; Strober, Freeman, & Morrell, 1997).

Although family functioning and outcome was measured by multimethod and multisource data, parental ratings of family functioning were the only variables of family functioning at intake that were significantly related to outcome in this sample. Moreover, the only indicator of outcome that was significantly related to these baseline variables of family functioning was the unit psychiatrist's CGI-I ratings of outcome. Initial analyses suggested that healthier parental ratings of SFI-II Health Competence (a general measure of the family's affective climate and structure), Cohesion (family togetherness and satisfaction within the family), and Conflict (the presence of overt markers of conflict) were significantly related to the psychiatrist's assessment of improvement at discharge. However, all three of these scales were significantly correlated with each other and, therefore, likely measured a common construct of family functioning. Upon closer examination, the high degree of overlap among these scales appears conceptually sound, as there would likely be little overt conflict within a family system in which

members feel a strong sense of togetherness and satisfaction, and within which the family structure and affective climate is healthy.

Theoretically, it can be argued that Health Competence assesses family functioning in a broader sense, as opposed to the Cohesion and Conflict scales. More specifically, Health Competence measures the following broad domains of family functioning: affective tone of the family, parental coalitions, problem-solving abilities, autonomy and individuality, optimistic versus pessimistic views, and acceptance of family members. In light of this broad-based description of the Health Competence scale, it can be argued that aspects of family Cohesion and Conflict might be subsumed under the umbrella of Health Competence. As such, parent-reported Health Competence was the only variable of family functioning to be tested as a predictor of outcome based upon the psychiatrist's CGI-I assessment. Eliminating Cohesion and Conflict from the analysis prevented redundancy, as these three variables could not contribute unique variance to predicting outcome.

In exploring whether parental ratings of family functioning at intake predicted outcome at discharge, age of the patient was controlled for, as the psychiatrist tended to rate younger patients as having good outcome and older patients as having poor outcome at discharge (which is commensurate with some previous studies examining outcome in a pediatric population). When age was controlled for in the logistic regression analysis, adaptive parental ratings of

Health Competence significantly predicted good outcome based upon the psychiatrist's ratings.

This finding is somewhat discrepant from previous studies which have identified clinician and patient perceptions of family functioning at baseline as predictive of outcome. However, the fact that parental perception of family functioning was predictive of outcome in this study does not provide evidence against the predictive validity of clinician and patient perceptions of family functioning. Simply stated, clinician and patient perception of family functioning at admission to treatment may not be predictive of acute treatment outcome, but predictive of short-term or long-term outcome. Meanwhile, the current findings serve as evidence that parent perception of family functioning may be predictive of acute treatment outcome, which is an area that has not been studied to the extent that short-term and long-term outcomes have been studied. Similarly, the lack of relationship between baseline family functioning and the other indicators of outcome (e.g., the clinical psychologist's CGI-I ratings and the BMI-based indicators) does not invalidate these variables as indicators of outcome. For instance, it is quite possible that these indicators of outcome are related to variables that were not evaluated by the current project or that these outcome variables predict longer-term outcome, which was beyond the scope of this study.

Psychological Symptoms as a Predictor of Outcome

The next set of hypotheses posited that the presence of more eating disordered cognitions and attitudes at admission would predict poor outcome at discharge, as defined by different measures of weight restoration (BMI) and clinical judgment (CGI-I) by two independent raters. Although unhealthy cognitions regarding food and weight have been identified as a core clinical feature of anorexia nervosa, these cognitions have not been evaluated as predictors of outcome. Moreover, only in recent years have the nature of eating attitudes been studied in relation to treatment outcome. Such studies have suggested that eating attitudes at admission to treatment are predictive of outcome two-years following discharge. However, no studies have examined the predictive utility of eating attitudes with regard to outcome over an acute period of treatment.

Despite the expectation that the degree of anorectic cognitions and eating attitudes at admission would predict outcome over an acute period of treatment, these hypotheses were not supported. It is difficult to ascertain why cognitions and attitudes were not related to the identified variables of outcome in this study. However, the limited power of this sample size may be a contributory factor. Additionally, it is possible that these cognitions and attitudes are predictive of more prolonged periods of outcome, rather than acute treatment outcomes. As

such, it will be important to examine baseline anorectic cognitions and pathological eating attitudes as predictors of short and long-term outcome in future studies.

Improvement in Family Functioning

The present study also hypothesized that the nature of family functioning would improve over the course of acute hospitalization, as measured by standardized clinical observation, patient report, and parent report. Although improvement in family functioning has been studied more in depth in recent years, such studies have focused on improved family functioning at long-term follow-up and have yielded inconsistent results. As such, relatively little is known about the improvements made in family functioning over an intensive and acute period of treatment—a shortcoming in the literature that the present study aimed to address.

The present study failed to find significant changes in family functioning from intake to discharge by standardized clinical observation and parent report. However, Expressiveness on the SFI-II improved significantly over the course of treatment by patient self-report. As such, it appears that patients perceived improvement in verbal and nonverbal expression of warmth, caring, and closeness within their families at discharge. This improvement is especially interesting, as

patients rated familial Expressiveness as quite healthy at baseline, prior to treatment. Given their perception of adaptive levels of familial expressiveness at intake, one might expect there to be little room for improvement within this particular area of family functioning. However, it is possible that patients perceived greater degrees of expressiveness within their families by the end of treatment due to the high degree of emphasis the treatment program exerts on "expression of feelings" as a treatment goal. Thus, the emphasis on appropriate expression of feelings throughout the course of treatment may have indeed increased family expressiveness to some degree and primed patients to notice and report increased levels of familial expressiveness.

Additionally, it is worth noting that patients rated their families as extremely healthy at intake on every scale of the SFI-II, with the exception of the Leadership scale—which they rated as less healthy than the "healthiest" normative group but healthier than the "least healthy" normative group. Leadership on the SFI-II measures the degree to which parents provide directive within the family system. This finding appears somewhat contradictory to previous depictions of ED parents as controlling and over-involved in their children's lives, as one might expect such parents to be rated as extremely directive in their leadership style. However, such an interpretation must be made with caution based upon the current data, as the ED patients rated Leadership as

fairly healthy (i.e., healthier than the least healthy normative group), although not as healthy as the other SFI-II scales.

The lack of improvement in parent reports of family functioning and patient reported Leadership, Health Competence, Cohesion, and Conflict on the SFI-II may have been due to many different factors. For instance, there may have been little room for significant improvement to occur, as patients and parents rated their families as fairly healthy at intake. Moreover, it is also possible that significant improvement within these domains of family functioning, no matter how healthy at baseline, cannot be realistically achieved within such a short period of time—especially when the focus of treatment is not on improving these domains of family functioning. Additionally, family members may have had relatively fewer opportunities to practice, or engage in, more adaptive family interactions over the course of treatment, as patients spent the majority of their time away from their families while participating in treatment.

Although patients and parents tended to view their families as functioning quite adaptively at admission to treatment, such findings were not consistently supported by standardized clinical observation on the TCFES. Overall family functioning (as measured by Global Competence) was no different than that of the healthy control group; however, ED families were rated as more dysfunctional on the following subscales: Responsibility, Expressiveness, Responsiveness, and Mood and Tone. As such, these families appeared to have difficulties accepting

responsibility for their own feelings and thoughts, clearly expressing themselves, and responding to and acknowledging other people's feelings. Moreover, the overall affective climate of these familial interactions was less warm and caring than the non-clinical sample. Although it appears that these families functioned less adaptively within the preceding domains of family functioning at intake, the present study failed to identify improvements within these areas over the course of acute treatment. As such, families may require a longer period of therapeutic intervention before significant improvements in family functioning are noticeable. Although standardized observations of these families yielded areas of dysfunction, it is worth noting that they appeared healthier than the non-clinical sample on the following scales: Negative Regard, Conflict Frequency, Conflict Affective Quality, and Conflict Generalization/Escalation. As such, these family interactions tended to be characterized by the near absence of conflict, disagreements, and negative regard for each other.

Collectively, these descriptions of family interactions are commensurate with some of the descriptions provided within the current literature. For instance, many studies have characterized families of individuals with AN as being extremely conflict avoidant and less open to forms of overt self-expression and direct communication (Humphrey, 1989; Latzer, Hochdorf, Bachar, & Canetti, 2002). In AN families where these descriptions hold true, it therefore makes sense that families would exhibit fewer forms of open expressiveness and affect, in both

positive and negative forms. Moreover, individuals within families whose affective climate is restricted are likely less inclined to accept responsibility for one's own feelings and respond to, or acknowledge, affects shared by others in the system. Such familial characteristics have been described as potential contributors to the development and maintenance of ED, as patients may learn to express themselves indirectly through the restriction of caloric intake.

Additionally, these results support studies which have found that patients and parents tend to report healthy family functioning, while clinicians tend to rate them as unhealthy (North, Gowers, & Byram, 1995). Although this finding has not been a consistent one within the literature, it appears to apply within this sample of families. Many clinicians and researchers have noted that commensurate perceptions of family functioning among family members in the face of incongruent clinical ratings may be the result of enmeshed family systems—a characteristic which has been observed in many ED samples. Moreover, the discrepancy between family ratings and clinician ratings may be the result of different world views of what is "healthy". For instance, patients and parents may refer to their own family systems as the frame of reference for normalcy, thereby resulting in healthier self-reports of family functioning. However, clinicians who have a broader frame of reference from working with a variety of families, might view the same behaviors and characteristics as unhealthy. Additionally, it is quite feasible that families wished to present

themselves in a positive light on a very face-valid measure of family functioning (SFI-II) during a period of time in which they felt vulnerable to judgment by others as a result of a family member requiring psychiatric hospitalization.

Improvement in Psychological Symptoms

The last set of hypotheses posited that eating disordered cognitions and attitudes would also improve over the course of treatment. Although eating disordered cognitions and attitudes continued to be extremely unhealthy at discharge (when compared to a non-clinical group), it appears that many aspects of eating attitudes improved significantly over the course of treatment. More specifically, Oral Control and Bulimia and Food Preoccupation on the EAT-26 improved significantly over an acute period of treatment, thereby resulting in significant change in the Total Score (as these subscales are two of three that comprise the Total Score). Although the Dieting subscale did not improve at a statistically significant level, it is worth noting that the subscale was approaching significance and might yield statistically significant change with a larger sample size. Collectively, these eating attitude findings are congruent with previous studies which have found that patient eating attitudes improve significantly over the course of hospitalization (Lowe, Davis, Annunziato, & Lucks, 2003).

Upon closer examination of EAT-26 items, it was determined that the scale assesses eating attitudes based upon *recent* eating *behavior*. The Oral Control subscale measures attitudes regarding the degree to which one engages in restriction of food intake and receives perceived feedback from others to gain weight. For example, some of the items that comprise the Oral Control subscale are as follows: "[I] avoid eating when I am hungry", "[I] take longer than others to eat my meals", and "[I] cut my food into small pieces". Additionally, the Bulimia and Food Preoccupation subscale assesses obsessive thoughts about food and tendencies to binge or purge through items such as: "[I] vomit after I have eaten" and "[I] give too much time and thought to food". It is important to note that patients are encouraged to engage in more adaptive behaviors while on the unit, and their eating behaviors are closely monitored throughout the course of treatment. As such, it only follows that patients would endorse fewer items in the pathological direction at the end of hospitalization when eating attitudes are assessed by examination of *current* behaviors. In this respect, it appears that the treatment program is quite successful in helping patients learn to engage in more adaptive eating behaviors.

Although eating attitudes appear to improve significantly over the course of hospitalization, it appears that pathological eating cognitions do not improve and continue to remain extremely unhealthy at discharge. Upon examination of the items on the MAC-R, it was found that many of the items assess cognitions by

examination of one's general worldview about food and weight (as opposed to one's thoughts about *recent behaviors*). For instance, some of the items on the MAC-R are as follows: "no one likes fat people, so I must remain thin to be liked by others", "If I eat a sweet, it will be converted instantly into stomach fat", and "If I can cut out all carbohydrates, I will never be fat". Therefore, it appears that patients' general worldviews about food, weight, and eating habits do not improve significantly during an acute period of hospitalization, despite significant improvements in eating behaviors and weight. In light of these results, it appears that patients must participate in further treatment over a longer period of time to experience improvements in eating disordered cognitions regarding food and weight.

Exploratory Analyses

Additional analyses were conducted to explore unplanned indicators of treatment outcome. One indicator of outcome was based upon the median split of percent gain in BMI over the course of treatment as an indicator of good versus poor outcome. However this indicator of outcome was not significantly related to any baseline measures of family functioning, eating disordered cognitions, or eating attitudes. Retrospectively, the non-significant relationship between potential predictors of outcome and the BMI median split was not completely

unexpected, as discharge BMI and good versus poor outcome based upon one standard deviation improvement in BMI did not yield significant relationships either. Therefore, acute treatment outcome based upon BMI indicators may not be related to baseline factors of family functioning, eating cognitions, or eating attitudes. Alternatively, BMI indicators of outcome at a later phase in treatment and recovery may be related to baseline factors, which was beyond the scope of this particular study.

In addition to examining the median split of percent gain in BMI as an indicator of outcome, eating attitudes at discharged were examined as a potential indicator of outcome. As mentioned previously, the Oral Control subscale, Bulimia and Food Preoccupation subscale, and total score of the EAT-26 improved significantly over the course of treatment. As such, the percent change in total score and a median split of percent change in total score (yielding good and poor outcome) were treated as psychological indicators of outcome. Baseline variables of family functioning, eating cognitions, and demographic/illness variables were examined in relation to these two indicators of outcome. However, no significant relationships emerged between these variables and outcome based upon a median split of percent change in eating attitudes. Similarly, no demographic/illness variables or variables of eating cognitions were related to percent change in eating attitudes. Additionally, patient reported Expressiveness on the SFI-II at admission was the only variable of family functioning that

significantly predicted percent change in eating attitudes, such that patients who reported healthier levels of familial expressiveness reported more improvements in eating attitudes at discharge. As such, patient perception of expressiveness within the family appears to play a vital role in outcome based upon eating attitudes, more so than parent and clinician perception. However, these results do not nullify the importance of parent and clinician perception of family functioning, as their perceptions may be significantly related to short-term or long-term outcome, rather than immediate treatment outcome. This finding is especially interesting, as the current literature has focused more so on indicators of outcome that are easily quantifiable, such as body mass index. Although psychological variables, manifest through behaviors, attitudes, and cognitions may not easily lend themselves to quantifiable measurement, it appears that they play an important role in outcome that warrants further study, specifically in relation to baseline family functioning.

Methodological Considerations

The present study has a number of methodological limitations that warrant discussion. For instance, the current study lacked adequate power on many analyses to detect significant predictors of outcome and change within family functioning and cognitions. Moreover, the largely homogenous demographic

nature of the sample precluded more sophisticated analyses differentiating gender, ethnicity, and subtype of anorexia—which may have yielded information of interest to researchers and clinicians who work with the ED population. Although the homogenous demographic nature of this sample is quite consistent with the demographic makeup of other studies investigating the ED population, the generalizability of these results to ethnic minorities, males, and AN purging type is nonetheless limited. As such, it will be important for future studies to examine differences in severity of illness, family functioning, psychological functioning, and multimethod/multisource outcome among these groups.

Another methodological limitation of this study pertains to the manner in which the TCFES was administered. Although all immediate family members were encouraged to participate in the taping at intake and discharge, this was not always pragmatically feasible due to difficulties coordinating everyone's schedules. Consequently, some tapes included dyads (typically the patient and mother), while other tapes included the patient, both parents, and periodically siblings of the patient. As such, the number of family members present and which individuals were able to participate in the study may have influenced the family dynamics caught on tape, subsequently impacting the clinical ratings of family functioning. Several studies utilizing clinician ratings of family functioning have noted that communication within the family may differ a great deal, based upon who is involved in the communication. Therefore, the inconsistent participation

among families in this study is a major limitation which may have impacted the results.

Additionally, only mothers completed the self-report measure of family functioning (with the exception of one case wherein the father was the child's primary caregiver). The current literature has suggested that perceptions of family functioning might vary among different individuals within the family system. As such, there may be differences in the way mothers and fathers view family functioning, subsequently impacting treatment outcome differentially. Given this potential difference in parental perspective, the current findings may be limited to maternal perception of family functioning, rather than generalizing to paternal perceptions as well.

The poor interrater reliability on the CGI-I is also an issue that must be addressed. This methodological issue can easily be resolved in future studies by employing consensus training prior to data collection in order to ensure that raters base their assessments on common criteria, thereby increasing interrater reliability. However, there is inherent value in gaining different perspectives on outcome from different expert raters, despite the value of consensus training. For instance, the clinical psychologist and psychiatrist who provided ratings for the current study may have had different, but equally valid perspectives on outcome based upon differences in training experiences. Such multisource data can potentially enrich a study and capture the multidimensional and complex nature of

treatment outcome, rather than simplifying the measure (through consensus training) to make the process of research cleaner. For instance, the present study yielded results that suggest the psychiatrist's CGI-I ratings were highly related to parental perceptions of family functioning at intake. However, as this research continues to grow, it is quite possible that the clinical psychologist's ratings might emerge as a predictor of outcome or an indicator of outcome in ways which were beyond the scope of the current study. Such information would be lost with consensus training on this measure.

Another limitation of the current study is the absence of a clinical comparison group. Although aspects of family functioning were compared to non-clinical groups, they were not compared to a non-ED psychiatric control group. As such, it is difficult to ascertain whether differences between the ED group and non-clinical groups are a function of the unique ED family environment. In other words, it is quite possible that the differences in family functioning noted between the ED and non-clinical groups also exist between non-clinical groups and families who have a child with any psychiatric disorder which required intensive treatment.

Clinical Implications and Areas for Future Research

Although the present study is limited by the aforementioned methodological issues, it contributes valuable information to the current body of literature on pediatric AN. It is one of very few studies that have evaluated treatment outcome in a homogeneous group of severely ill patients requiring hospitalization. Moreover, these results underscore the importance of family functioning (by patient and parent self-report), as healthier family functioning appears to predict better outcome by psychiatrist ratings and eating attitudes. Thus, variables of family functioning should continue to be studied as baseline predictors of outcome and correlates with the course of illness, recovery, and relapse. Given that parent perception of family functioning and patient perception of familial expressiveness were both predictors of immediate outcome in the present study, future studies might continue to investigate the nature of these variables in relation to longer-term outcome. Such studies might show that the perception of healthier family functioning at baseline is related to better outcome in the short and long-term. Thus, aspects of family functioning that appear to be related with outcome might then be targeted with specific interventions earlier in the course of treatment in order to increase the likelihood of successful treatment and recovery.

Additionally, the present study found that weight restoration and change in eating attitudes (in relation to current eating behaviors) were the first symptoms to remit. However, more ingrained psychological symptoms (i.e., eating cognitions), were resistant to change, which is congruent with the current literature. As such, it appears that an acute period of hospitalization is most effective in treating emaciation and eating disordered behavior. However, restructuring the pathological cognitive content of patients appears to require a longer period of therapeutic intervention. Given that the cognitive content of patients appears resistant to change in earlier phases of treatment, the severity of these symptoms may play a vital role in vulnerability to relapse after discharge from the hospital.

Future studies should employ a more sophisticated longitudinal design in order to monitor patients as they transition back into their natural environments and continue treatment on an outpatient basis. These studies should continue to monitor physical symptoms (such as BMI) and eating behaviors to evaluate whether their initial progress continues to grow, maintain, or regress. For instance, it will be important to examine whether improvements in eating behaviors are maintained once discharged from the hospital, as parents may find it challenging to monitor their child's eating behaviors as closely as the hospital staff. Additionally, it will be important to study the course of change in cognitive symptoms. In doing so, short-term and long-term predictors of treatment outcome might be identified, as well as factors that contribute to relapse. As cognitive

symptoms appear to persist after weight restoration, treatment interventions following discharge from the hospital should be targeted toward these psychological symptoms. As such, it will be worthwhile for future studies to examine the effectiveness of different types of therapy (i.e., cognitive behavioral therapy) in treating these persisting symptoms, both within the hospital setting and outpatient setting.

In addition to addressing some of the methodological issues inherent in this study, future studies might also continue to utilize a multimethod and multisource approach to data collection. Although these approaches were utilized within the present study, future studies might examine different sources of data. For instance, bone density scans and echocardiograms might be used as additional physical indicators of outcome, in addition to BMI. Additionally, family therapists might be used as raters of family functioning, as they work quite intimately with these families and likely have a unique perspective on family dynamics. Given the complex nature of family functioning and anorexia nervosa (i.e., the interaction between psychological functioning and physical health), continued work with multisource and multimethod data will likely better elucidate the nature of this debilitating and life-threatening psychiatric disorder.

APPENDIX A

Consent Form

The University of Texas Southwestern Medical Center at Dallas

Children's Medical Center at Dallas

CONSENT TO PARTICIPATE IN RESEARCH

Title of Research: Family Functioning in Children and Adolescents with Eating Disorders

Sponsor: Timberlawn Research Foundation

Investigators:	Telephone No. (regular office hours)	Telephone No. (other times)
Betsy Kennard, Psy.D.	214.648.4403	214.648.4403
Stephanie Setliff, M.D.	214.456.6471	214.456.6471
Maryann Hetrick, B.A.	214.648.4456	214.648.4456

PURPOSE: The primary purpose of this study is to investigate family interaction patterns in children and adolescents who have been diagnosed with an eating disorder (ED) and to determine whether these patterns respond to treatment. The second aim is to determine whether family functioning at admission to the

hospital predicts response to inpatient, partial hospitalization, day treatment, and intensive outpatient psychiatric treatment. The third aim will be to compare families who have a child with an eating disorder to families with a depressed child to determine characteristics unique to ED families. The final aim is to assess the effectiveness of the eating disorder treatment program at Children's Medical Center in changing dysfunctional family interaction patterns.

PROCEDURES: This study will assess patients recently admitted to a program for treatment of Eating Disorders via self report questionnaires as well as videotaped interactions between patients and either one or both parents. Questionnaires and videotaped interactions will help assess weight characteristics, behaviors, interaction styles, symptoms of eating disorders, and family functioning. Diagnostic evaluations and assessments of behavioral and cognitive aspects of disordered eating will also be obtained. The diagnostic evaluations and assessments will measure the general cause, development, and outcome of an eating disorder in the patient, as well as measure depressive symptoms and the amount of change from entry to discharge in the patient's performance. The diagnostic evaluation will only be done at study entry.

Initial Visit

During the first evaluation you and your child will be asked questions about your child's eating habits, and a variety of symptoms that adolescents sometimes have. These questions will be in the form of an interview and written questionnaires. In addition, your family will be asked to discuss three topics for eight minutes each. Your discussion will be videotaped so that they can later be coded on a measure of family interaction. This visit will last approximately three hours.

Follow- up

Follow-up assessments of you and your child, including all measures other than the diagnostic interview, will be conducted at discharge, 6 months and 12 months after discharge. In addition, your child will also be assessed for any depressive symptoms after discharge, as well as response to the prior treatment of the eating disorder. The data collected from your child will be compared to existing data from children with the diagnosis of Major Depressive Disorder for this study.

Study Duration

The individual subject duration in the study is based on their length of treatment in this continuum of care eating disorder program. Most patients remain in the treatment program anywhere from 4 to 8 weeks. However, some patients remain in the program much longer. After discharge from treatment, two follow-ups will be conducted at 6 months and 12 months. Depending upon the length of

treatment, the study can last from 52 weeks up to 56 weeks. Again this could be longer, if the patient remains in treatment for an extended period of time.

POSSIBLE RISKS: The risk of this study involves discussing information that you or your child may feel uncomfortable talking about. All participants will be told that they do not have to answer any questions if they are uncomfortable. Subjects who appear or express any discomfort with the procedure will be interviewed by the research coordinator or Dr. Kennard to determine the need for intervention. All data will be password protected. Participation in this study does require you to be videotaped and to sign a consent form. The consent form, which will have the patient's signature, as well as the videotape of the parent-child interaction could be linked to subjects. The videotapes will be labeled with identification numbers only and only Dr. Kennard and her research assistants will have access to and be able to view the tapes. The consent form will be kept in a locked cabinet inside charts with only identification numbers labeled on cover, which only Dr. Kennard and her research assistants will have access.

POSSIBLE BENEFITS: While there is no specific benefit to subjects for participation, the results of this research may help determine factors that

contribute to treatment response, which may help others in the future who have the same disorder.

PAYMENT TO TAKE PART IN THIS RESEARCH: Subjects will not be paid for participation in this research.

VOLUNTARY PARTICIPATION IN RESEARCH: Your child has the right to agree or refuse to participate in this research. If your child decides to participate and later changes his/her mind, he/she is free to discontinue participation in the research at any time.

Refusal to participate will involve no penalty or loss of benefits to which your child is otherwise entitled. Refusal to participate will not affect your child's legal rights or the quality of health care that your child receives at this center.

ALTERNATIVES TO PARTICIPATION IN THIS RESEARCH: Your child does not have to participate in this research to receive care for your medical problem. Please ask Dr. Kennard as many questions as you and your child wish.

Dr. Kennard's answers to your questions could help you decide whether to participate in this research or receive the standard care that is currently available for your child's medical problem.

If your child decides to participate in research now, and later changes his/her mind, your child may stop his/her participation in the research then and receive the alternative care.

RECORDS OF YOUR PARTICIPATION IN THIS RESEARCH: You have the right to privacy. Any information about you that is collected for this research will remain confidential as required by law. In addition to this consent form, you will be asked to sign an "Authorization for Use and Disclosure of Protected Health Information for Research Purposes," which will contain more specific information about who is authorized to review, use, and/or receive your protected health information for the purposes of this study.

YOUR QUESTIONS: Dr. Kennard is available to answer you and your child's questions about this research at 214.648.4403. The Chairman of the IRB is available to answer questions about your child's rights as a participant in research. You may telephone the Chairman of the IRB during regular office hours at 214-648-3060.

YOU WILL HAVE A COPY OF THIS CONSENT FORM TO KEEP.

Your signature below certifies the following:

- You have read (or been read) the information provided above.
- You have received answers to all of your questions.
- You have freely decided to participate in this research.
- You understand that you are not giving up any of your legal rights.

_____

Participant's Name (printed)

_____ _____

Participant's Signature Date

_____

Legally authorized representative's name

(printed) (if applicable)

_____ _____

Legally authorized representative's Date

Signature (if applicable)

_____

Name (printed) of person obtaining

Consent

_____ _____

Signature of person obtaining consent Date

ASSENT OF A MINOR:

I have discussed my participation in this research with my mother or father or legal guardian and my study doctor, and I agree to participate in this research.

_____ _____

Signature (participants from 10 to 18 years old) Date

APPENDIX B

HIPAA Notification Form

The University o Texas Southwestern Medical Center at Dallas

Children's Medical Center, Parkland Health & Hospital System

Retina Foundation of the Southwest, Texas Scottish Rite Hospital for Children

Zale Lipshy University Hospital, St. Paul University Hospital

The University of Texas Southwestern Moncrief Cancer Center

Authorization for Use and Disclosure of

Health Information for Research Purposes

NAME OF RESEARCH PARTICIPANT:

_____

1. You agree to let Children's Medical Center at Dallas share your health information with Dr. Betsy Kennard and her staff at the University of Texas Southwestern Medical Center at Dallas for the purpose of the following research study: *Family Functioning of Children and Adolescents with Eating Disorders*, IRB# 0603-364

2. You agree to let the Researchers use your health information for this Research Project. You also agree to let the Researchers share your health information with

others who may be working with the Researchers on the Research Project ("Recipients") as follows.

- The UT Southwestern institutional Review Board (IRB). This is a group of people who are responsible for assuring that the rights of participants in research are respected. Members and staff of the IRB at UT Southwestern may review the records of your participation in this research. A representative of the IRB may contact you for information about your experience with this research. If you do not want to answer their questions, you may refuse to do so.

- Representatives of the Office of Human Research Protections (OHRP). The OHRP may oversee the Research Project to confirm compliance with laws, regulations and ethical standards.

3. Whenever possible your health information will be kept confidential. Federal privacy laws may not apply to some institutions outside of UT Southwestern. There is a risk that the Recipients could share your information with others without your permission. UT Southwestern cannot guarantee the confidentiality of your health information after it has been shared with the Recipients.

4. You agree to permit the Researchers to use and share your health information as listed below. Information related to your psychiatric history, such as previous diagnosis, substance abuse history and any previous psychiatric hospitalizations may be used. This information also refers to any medical conditions you may have whether or not it is related to your eating disorder. In addition, questionnaires regarding mental health will be obtained.

5. The Researchers may use your health information to create research data that does not identify you. Research data that does not identify you may be used and shared by the Researchers (for example, in a publication about the results of the Research Project); it may also be used and shared by the Researchers and Recipients for other research purposes not related to the Research Project.

6. This authorization is voluntary. Your health care providers must continue to provide you with health care services even if you choose not to sign this authorization. However, if you choose not to sign this authorization, you cannot take part in this Research Project.

7. This Authorization has no expiration date.

8. If you change your mind and do not want us to collect or share your health information, you may cancel this authorization at any time. If you decide to

cancel this authorization, you will no longer be able to take part in the Research Project. The Researchers may still us and share the health information that they have already collected before you cancelled the authorization. To cancel this authorization, you must make this request in writing to:

Dr. Betsy Kennard

5323 Harry Hines Blvd.

Dallas, Texas 75390-8589

9. A copy of this authorization form will be provided to you.

_____ _____

Signature of Research Participant Date

***For Legal Representatives of Research Participants* (if applicable):**

Printed Name of Legal Representative: _____

Relationship to Research Participant: _____

I certify that I have the legal authority under applicable law to make this Authorization on behalf of the Research Participant identified above. The basis for this legal authority is:

_____.

(e.g. parent, legal guardian, person with legal power of attorney, etc.)

_____ _____

Signature of Legal Representative Date

APPENDIX C

Tables

Table 1

Interrater Reliability for TCFES

Domain/Scale	Rater 1 M (SD)	Rater 2 M (SD)	Intraclass Correlation
Structure	13.56 (3.33)	14.33 (3.22)	.87
Autonomy	9.89 (2.35)	9.83 (2.39)	.80
Problem-Solving	6.17 (2.04)	6.28 (1.84)	.82
Affect Regulation	19.67 (3.74)	19.94 (3.40)	.82
Conflict	13.39 (2.25)	13.39 (2.40)	.81
Sum of Scales	62.67 (11.77)	63.78 (11.45)	.88
Global Competence	10.50 (3.02)	10.61 (2.55)	.94

Note: Higher scores denote greater health.

Table 2

Chi Square Test for Independence between CGI-I Rater 1 and BMI Improvement of at least 1 SD

		CGI-I (Rater 1)			
		Improved	Not Improved	Chi Square	*p*-value
BMI Improvement (of at least 1 SD)	Improved	20	9	1.45	*ns*
	Not Improved	7	7		

Table 3

Chi Square Test for Independence between CGI-I Rater 2 and BMI Improvement of at least 1 SD

		CGI-I (Rater 2)			
		Improved	Not Improved	Chi Square	*p*-value
BMI Improvement (of at least 1 SD)	Improved	21	8	.99	*ns*
	Not Improved	8	6		

Table 4

Independent t-tests between Discharge BMI and Dichotomous Outcome Variables

Outcome Variable	Discharge BMI: Good Outcome M (SD)	Discharge BMI: Poor Outcome M (SD)	df	*t*	*p*
CGI-I Rater One	17.61 (1.20)	17.52 (1.43)	41	.23	*ns*
CGI-I Rater Two	17.74 (1.35)	17.24 (1.07)	41	1.22	*ns*
BMI Improvement (of at least 1 SD)	17.86 (1.31)	17.00 (.99)	41	-2.16	.04[a]

[a] Not significant after Bonferroni correction

Table 5

Dichotomous Indicators of Outcome Frequency Table

	Improved		Not Improved	
Indicator of Outcome	Frequency	Percent	Frequency	Percent
CGI-I (Rater 1)	27	62.8	16	37.2
CGI-I (Rater 2)	29	67.4	14	32.6
BMI Improvement (of at least 1 SD)	29	67.4	14	32.6

Table 6

Demographic and Outcome Variable Characteristics

Variable	Mean	SD	Range
Age in Years	14.21	1.73	10-17
Intake BMI	15.64	1.38	13.0-18.3
Discharge BMI	17.58	1.27	15.1-20.2
Length of Treatment in Days	28.75	13.92	4-62

Table 7

Gender/Ethnicity Frequency Table

	Frequency (%)		
Ethnicity	Male	Female	Total
Caucasian	4 (9.25)	32 (74.45)	36 (83.7)
African-American	1 (2.35)	1 (2.35)	2 (4.7)
Latino	0 (0.0)	4 (9.3)	4 (9.3)
Asian	0 (0.0)	1 (2.3)	1 (2.3)
Total	5 (11.6)	38 (88.4)	43 (100)

Table 8

ED Diagnosis by Gender Frequency Table

	Frequency (%)		
ED Diagnosis	Male	Female	Total
AN, Restricting	4 (9.3)	26 (60.5)	30 (69.8)
AN, Purging	0 (0)	5 (11.6)	5 (11.6)
ED NOS	1 (2.3)	7 (16.3)	8 (18.6)
Total	5 (11.6)	38 (88.4)	43 (100)

Note: Under the diagnostic category of ED NOS, 5 females engaged in purging, 2 females engaged in restricting, and 1 male engaged in restricting.

Table 9

Frequency of Child/Adolescent Comorbid DSM-IV Diagnoses

DSM-IV Diagnosis	Frequency	Percent
Mood Disorders	**30**	**69.77**
Major Depressive Disorder	16	37.21
Dysthymic Disorder	2	4.65
Depression NOS	12	27.91
Anxiety Disorders	**12**	**27.91**
Obsessive Compulsive Disorder	3	6.98
Social Phobia	2	4.65
Generalized Anxiety Disorder	1	2.33
Anxiety Disorder NOS	6	13.95
Miscellaneous Disorders	**4**	**9.31**
Oppositional Defiant Disorder	3	6.98
Attention Deficit/Hyperactivity Disorder	1	2.33

Table 10

Sample Descriptive Statistics for TCFES (for all available data)

	Intake n=41		Discharge n=30	
Domain/Scale	Mean (SD)	Range	Mean (SD)	Range
Structure	13.54 (3.25)	6-18	14.47 (3.14)	8-18
Autonomy	9.88 (2.28)	5-14	10.63 (2.17)	7-15
Problem-Solving	6.66 (1.85)	2-10	6.50 (1.83)	4-10
Affect Regulation	19.56 (4.22)	10-27	19.33 (4.13)	13-27
Conflict	13.56 (2.73)	3-15	13.57 (2.61)	5-15
Sum of Scales	63.20 (12.52)	26-81	64.50 (11.44)	43-82
Global Competence	10.54 (3.15)	4-16	10.50 (3.19)	2-15

Note: Higher scores denote greater health.

Table 11

Sample Descriptive Statistics for Patient SFI-II (for all available data)

	Intake n=41		Discharge n=28	
Subscale	Mean (SD)	Range	Mean (SD)	Range
Health Competence	2.18 (.85)	1.21-4.74	2.00 (.52)	1.21-3.11
Cohesion	2.70 (.80)	1.60-4.80	2.58 (.58)	1.60-3.80
Conflict	2.18 (.67)	1.33-3.75	2.05 (.66)	1.33-4.00
Leadership	2.21 (.68)	1.00-4.33	2.41 (.75)	1.00-4.00
Expressiveness	1.99 (1.00)	1.00-4.60	1.61 (.67)	1.00-3.40

Note: Lower scores denote greater health.

Table 12

Sample Descriptive Statistics for Parent SFI-II (for all available data)

	Intake n= 42		Discharge n= 27	
Subscale	Mean (SD)	Range	Mean (SD)	Range
Health Competence	2.23 (.79)	1.21-4.37	2.16 (.82)	1.21-4.37
Cohesion	2.69 (.95)	1.00-4.80	2.65 (.92)	1.20-4.80
Conflict	2.24 (.63)	1.33-3.75	2.19 (.65)	1.42-4.00
Leadership	2.27 (.77)	1.00-3.67	2.24 (.79)	1.00-3.67
Expressiveness	1.95 (.88)	1.00-4.20	1.94 (.89)	1.00-4.20

Note: Lower scores denote greater health.

Table 13

Sample Descriptive Statistics for Patient EAT-26 (for all available data)

	Intake n=39		Discharge n=28	
Subscale	Mean (SD)	Range	Mean (SD)	Range
Dieting	16.97 (12.36)	0-39	12.43 (11.32)	0-39
Bulimia & Food Preoccupation	5.97 (4.37)	0-18	4.00 (3.69)	0-13
Oral Control	9.67 (4.71)	0-20	5.46 (4.64)	0-16
Total Score	32.62 (18.97)	0-67	21.89 (18.34)	0-68

Note: Higher scores denote the presence of more unhealthy eating attitudes.

Table 14

Sample Descriptive Statistics for Patient MAC-R (for all available data)

	Intake n=41		Discharge n=28	
Subscale	Mean (SD)	Range	Mean (SD)	Range
Self-Control	30.42 (7.52)	16-40	27.75 (8.71)	11-40
Rigid Weight-Regulation	22.44 (8.54)	8-39	20.29 (8.55)	8-38
Weight and Approval	21.27 (6.72)	11-38	20.00 (6.77)	8-36
Total Score	74.12 (19.21)	43-115	68.04 (21.32)	28-114

Note: Higher scores denote the presence of more anorectic cognitions.

Table 15

Bivariate Correlations between Baseline TCFES and Discharge BMI (n=41)

	Discharge BMI (Continuous)	
Domain/Scale	*r*	*p*
Structure	-.152	*ns*
Autonomy	-.183	*ns*
Problem-Solving	-.163	*ns*
Affect Regulation	-.283	.07
Conflict	-.058	*ns*
Sum of Scales	-.207	*ns*
Global Competence	-.210	*ns*

Note: *r*=Pearson Product Moment Correlation Coefficient

Table 16

Bivariate Correlations between Baseline Patient SFI-II and Discharge BMI (n=41)

	Discharge BMI (Continuous)	
Subscale	*r*	*p*
Health Competence	.202	*ns*
Cohesion	.171	*ns*
Conflict	.345	.03[a]
Leadership	.219	*ns*
Expressiveness	.244	*ns*

Note: *r*=Pearson Product Moment Correlation Coefficient

[a] Non-significant after Bonferroni Correction

Table 17
Bivariate Correlations between Baseline Parent SFI-II and Discharge BMI (n=42)

	Discharge BMI (Continuous)	
Subscale	*r*	*p*
Health Competence	.115	*ns*
Cohesion	.115	*ns*
Conflict	.136	*ns*
Leadership	-.048	*ns*
Expressiveness	.106	*ns*

Note: *r*=Pearson Product Moment Correlation Coefficient
[a]Non-significant after Bonferroni correction

Table 18

Independent t-tests of Intake TCFES and 1 SD Improvement of BMI as an Indicator of Poor vs. Good Outcome

Domain/Subscale	n=13 Poor Outcome M (SD)	n=28 Good Outcome M (SD)	df	*t*	*p*
Structure	14.54 (2.96)	13.07 (3.32)	39	1.36	*ns*
Autonomy	11.00 (1.92)	9.36 (2.28)	39	2.25	.03[a]
Problem-Solving	7.00 (1.63)	6.50 (1.95)	39	.80	*ns*
Affect Regulation	21.00 (3.49)	18.89 (4.42)	39	1.51	*ns*
Conflict	13.92 (2.29)	13.39 (2.94)	39	.57	*ns*
Sum of Scales	67.39 (10.60)	61.25 (13.04)	39	1.48	*ns*
Global Competence	11.46 (2.79)	10.11 (3.26)	39	1.29	*ns*

Note: Higher scores on the TCFES denote greater health.
[a] Non-significant after Bonferroni corrections.

Table 19

Independent t-tests of Intake SFI-II and 1 SD Improvement of BMI as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=13 Poor Outcome M (SD)	n=28 Good Outcome M (SD)	df	*t*	*p*
Patient					
Health Competence	2.28 (.99)	2.14 (.79)	39	.48	*ns*
Cohesion	2.72 (.93)	2.69 (.75)	39	.11	*ns*
Conflict	2.17 (.73)	2.18 (.66)	39	-.02	*ns*
Leadership	1.92 (.68)	2.35 (.65)	39	-1.92	.06
Expressiveness	1.92 (1.08)	2.02 (.99)	39	-.30	*ns*
Parent					
Health Competence	2.51 (.92)	2.09 (.69)	40	1.64	*ns*
Cohesion	2.90 (1.02)	2.58 (.91)	40	1.04	*ns*
Conflict	2.19 (.61)	2.27 (.65)	40	-.39	*ns*
Leadership	2.38 (.87)	2.21 (.72)	40	.66	*ns*
Expressiveness	2.31 (1.09)	1.76 (.71)	40	1.72	.10

Note: Lower SFI-II scores denote greater health.

Table 20

Independent t-tests of Intake TCFES and CGI-I (Rater 1) as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=26 Good Outcome M (SD)	n=15 Poor Outcome M (SD)	df	*t*	*p*
Structure	13.85 (3.02)	13.00 (3.67)	39	.80	*ns*
Autonomy	9.89 (2.25)	9.87 (2.42)	39	.02	*ns*
Problem-Solving	6.77 (1.99)	6.47 (1.64)	39	.50	*ns*
Affect Regulation	19.92 (4.47)	18.93 (3.81)	39	.72	*ns*
Conflict	13.50 (3.10)	13.67 (2.02)	39	-.19	*ns*
Sum of Scales	63.92 (13.48)	61.93 (11.00)	39	.49	*ns*
Global Competence	10.96 (3.29)	9.80 (2.83)	39	1.14	*ns*

Note: Higher TCFES scores denote greater health.

Table 21

Independent t-tests of Intake SFI-II and CGI-I (Rater 1) as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=26 Good Outcome M (SD)	n=15 Poor Outcome M (SD)	df	*t*	*p*
Patient					
Health Competence	2.24 (1.00)	2.07 (.54)	39	.61	*ns*
Cohesion	2.75 (.90)	2.63 (.63)	39	.46	*ns*
Conflict	2.17 (.76)	2.19 (.51)	39	-.13	*ns*
Leadership	2.22 (.73)	2.20 (.60)	39	.08	*ns*
Expressiveness	2.03 (1.15)	1.91 (.72)	39	.35	*ns*
Parent					
Health Competence	2.18 (.81)	2.31 (.78)	40	-.48	*ns*
Cohesion	2.62 (.94)	2.79 (.99)	40	-.54	*ns*
Conflict	2.19 (.64)	2.33 (.63)	40	-.67	*ns*
Leadership	2.32 (.67)	2.19 (.92)	40	.54	*ns*
Expressiveness	1.90 (.92)	2.03 (.85)	40	-.44	*ns*

Note: Lower scores on the SFI-II denote greater health.

Table 22

Independent t-tests of Intake TCFES and CGI-I (Rater 2) as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=29 Good Outcome M (SD)	n=12 Poor Outcome M (SD)	df	*t*	*p*
Structure	13.83 (3.33)	12.83 (3.07)	39	.89	*ns*
Autonomy	9.76 (2.42)	10.17 (1.99)	39	-.52	*ns*
Problem-Solving	6.59 (1.99)	6.83 (1.53)	39	-.39	*ns*
Affect Regulation	19.72 (4.80)	19.17 (2.41)	39	.49	*ns*
Conflict	13.52 (2.95)	13.67 (2.23)	39	-.16	*ns*
Sum of Scales	63.41 (13.89)	62.67 (8.85)	39	.17	*ns*
Global Competence	10.69 (3.38)	10.17 (2.59)	39	.48	*ns*

Note: Higher scores denote greater health on the TCFES.

Table 23

Independent t-tests of Intake SFI-II and CGI-I (Rater 2) as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=28 Good Outcome M (SD)	n=13 Poor Outcome M (SD)	df	*t*	*p*
Patient					
Health Competence	1.98 (.76)	2.61 (.90)	39	-2.34	.03[a]
Cohesion	2.54 (.72)	3.06 (.87)	39	-2.03	.05
Conflict	2.02 (.60)	2.51 (.72)	39	-2.29	.03[a]
Leadership	2.18 (.71)	2.28 (.64)	39	-.45	*ns*
Expressiveness	1.89 (1.05)	2.21 (.91)	39	-.97	*ns*
Parent					
Health Competence	2.00 (.72)	2.70 (.73)	40	-2.96	.01*
Cohesion	2.41 (.90)	3.23 (.82)	40	-2.84	.01*
Conflict	2.04 (.64)	2.64 (.38)	40	-3.15	.00*
Leadership	2.26 (.60)	2.29 (1.05)	40	-.09	*ns*
Expressiveness	1.76 (.82)	2.33 (.91)	40	-2.06	.05

*$p<.05$ (with Bonferroni correction)

[a]Non-significant after Bonferroni correction

Note: Lower scores denote greater health on the SFI-II.

Table 24

Test for Multicollinearity Correlation Matrix: Potential Intake Parent SFI-II Predictor Variables (n=42)

Parent SFI-II Subscale		Health Competence	Cohesion	Conflict
Health Competence	*r*	1.00		
	p-value			
Cohesion	*r*	.89		
	p-value	.00**		
Conflict	*r*	.79	.68	1.00
	p-value	.00**	.00**	

**$p<.01$ (with Bonferroni corrections)

Table 25

Summary of Logistic Regression for Intake Family Functioning Predicting Discharge BMI (n=42)

Parent SFI-II Subscale	β	SE	Wald	df	*p*
Health Competence	1.15	.53	4.68	1	.03*

Note: Patient's age is controlled for in the above analysis.
*$p<.05$

Table 26
Bivariate Correlations between Baseline Patient MAC-R and Discharge BMI (n=41)

	Discharge BMI (Continuous)	
Subscale	*r*	*p*
Self-Control	.026	*ns*
Rigid Weight Regulation	.017	*ns*
Weight and Approval	-.032	*ns*
Total Score	.006	*ns*

Note: *r*=Pearson Product Moment Correlation Coefficient

Table 27

Independent t-tests of Intake MAC-R and Improvement of BMI as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=13 Poor Outcome M (SD)	n=28 Good Outcome M (SD)	df	*t*	*p*
Self-Control	34.00 (5.57)	28.75 (7.81)	39	2.18	.04[a]
Rigid Weight Regulation	25.08 (8.40)	21.21 (8.47)	39	1.36	*ns*
Weight and Approval	22.85 (8.36)	20.54 (5.85)	39	1.03	*ns*
Total Score	81.92 (19.42)	70.50 (18.34)	39	1.82	.08

[a]Non-significant after Bonferroni corrections

Note: Higher scores on the MAC-R denote the presence of more anorectic cognitions.

Table 28
Independent t-tests of Intake MAC-R and CGI-I (Rater 1) as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=26 Good Outcome M (SD)	n=15 Poor Outcome M (SD)	df	*t*	*p*
Self-Control	30.69 (8.04)	29.93 (6.77)	39	.31	*ns*
Rigid Weight Regulation	22.00 (9.14)	23.20 (7.63)	39	-.43	*ns*
Weight and Approval	22.39 (6.54)	19.33 (6.82)	39	1.42	*ns*
Total Score	75.08 (19.75)	72.47 (18.81)	39	.42	*ns*

Note: Higher scores on the MAC-R denote the presence of more anorectic cognitions.

Table 29

Independent t-tests of Intake MAC-R and CGI-I (Rater 2) as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=28 Good Outcome M (SD)	n=13 Poor Outcome M (SD)	df	*t*	*p*
Self-Control	30.21 (8.05)	30.85 (6.50)	39	-.25	*ns*
Rigid Weight Regulation	21.93 (9.10)	23.54 (7.41)	39	-.56	*ns*
Weight and Approval	20.96 (6.47)	21.92 (7.47)	39	-.42	*ns*
Total Score	73.11 (19.50)	76.31 (19.17)	39	-.49	*ns*

Note: Higher scores on the MAC-R denote the presence of more anorectic cognitions.

Table 30

Bivariate Correlations between Baseline Patient EAT-26 and Discharge BMI (n=39)

	Discharge BMI (Continuous)	
Subscale	*r*	*p*
Dieting	.01	*ns*
Bulimia and Food Preoccupation	-.06	*ns*
Oral Control	.01	*ns*
Total Score	-.01	*ns*

Note: *r*=Pearson Product Moment Correlation Coefficient

Table 31

Independent t-tests of Intake EAT-26 and Improvement of BMI as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=12 Poor Outcome M (SD)	n=27 Good Outcome M (SD)	df	*t*	*p*
Dieting	23.25 (10.63)	14.19 (12.22)	37	2.22	.03 [a]
Bulimia and Food Preoccupation	7.92 (4.62)	5.11 (4.04)	37	1.92	.06
Oral Control	11.17 (4.53)	9.00 (4.72)	37	1.34	*ns*
Total Score	42.33 (17.18)	28.30 (18.39)	37	2.24	.03 [a]

[a] Non-significant after Bonferroni correction.

Note: Higher EAT-26 scores denote the presence of more eating disordered attitudes.

Table 32

Independent t-tests of Intake EAT-26 and CGI-I (Rater 1) as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=25 Good Outcome M (SD)	n=14 Poor Outcome M (SD)	df	*t*	*p*
Dieting	16.24 (13.31)	18.29 (10.81)	39	-.49	*ns*
Bulimia and Food Preoccupation	5.96 (4.89)	6.00 (3.44)	39	-.03	*ns*
Oral Control	9.84 (5.01)	9.36 (4.29)	39	.30	*ns*
Total Score	32.04 (20.62)	33.64 (16.28)	39	-.25	*ns*

Note: Higher EAT-26 scores denote the presence of more eating disordered attitudes.

Table 33

Independent t-tests of Intake EAT-26 and CGI-I (Rater 2) as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=27 Good Outcome M (SD)	n=12 Poor Outcome M (SD)	df	*t*	*p*
Dieting	15.48 (12.46)	20.33 (11.96)	37	-1.14	*ns*
Bulimia and Food Preoccupation	5.41 (4.20)	7.25 (4.65)	37	-1.22	*ns*
Oral Control	9.67 (5.14)	9.67 (3.77)	37	.00	*ns*
Total Score	30.56 (19.26)	37.25 (18.23)	37	-1.02	*ns*

Note: Higher EAT-26 scores denote the presence of more eating disordered attitudes.

Table 34

Intake and Discharge TCFES Paired Samples t-test (n=30)

Domain/Subscale	Intake M (SD)	Discharge M (SD)	df	Mean Difference	*t*	*p*
Structure	13.73 (3.32)	14.47 (3.14)	29	-.73	-1.53	*ns*
Autonomy	10.10 (2.51)	10.63 (2.17)	29	-.53	-1.38	*ns*
Problem-Solving	6.70 (1.93)	6.50 (1.83)	29	.20	.69	*ns*
Affect Regulation	19.73 (4.32)	19.33 (4.13)	29	.40	.71	*ns*
Conflict	13.57 (2.87)	13.57 (2.61)	29	.00	.00	*ns*
Sum of Scales	63.80 (13.03)	64.50 (11.44)	29	-.70	-.36	*ns*
Global Competence	10.83 (3.20)	10.50 (3.19)	29	.33	.74	*ns*

Note: Higher TCFES scores denote greater health.

Table 35

Intake and Discharge Patient SFI-II Paired Samples t-test (n=28)

Subscale	Intake M (SD)	Discharge M (SD)	df	Mean Difference	*t*	*p*
Health Competence	2.03 (.57)	2.00 (.52)	27	.03	.32	*ns*
Cohesion	2.59 (.66)	2.57 (.58)	27	.01	.14	*ns*
Conflict	2.11 (.54)	2.05 (.66)	27	.06	.60	*ns*
Leadership	2.13 (.53)	2.41 (.75)	27	-.27	-1.80	.08
Expressiveness	1.88 (.73)	1.61 (.67)	27	.27	2.91	.007*

*$p<.05$ (with Bonferroni correction)
Note: Lower scores on the SFI-II denote greater health.

Table 36

Intake and Discharge Parent SFI-II Paired Samples t-test (n=27)

Subscale	Intake M (SD)	Discharge M (SD)	df	Mean Difference	*t*	*p*
Health Competence	2.21 (.84)	2.16 (.82)	26	.06	.74	*ns*
Cohesion	2.64 (.92)	2.65 (.92)	26	-.02	-.14	*ns*
Conflict	2.21 (.64)	2.19 (.65)	26	.02	.30	*ns*
Leadership	2.36 (.70)	2.24 (.79)	26	.12	.90	*ns*
Expressiveness	2.00 (.92)	1.94 (.89)	26	.06	.56	*ns*

Note: Lower scores on the SFI-II denote greater health.

Table 37

Intake and Discharge Patient MAC-R Paired Samples t-test (n=29)

Subscale	Intake M (SD)	Discharge M (SD)	df	Mean Difference	*t*	*p*
Self-Control	30.17 (7.62)	27.76 (8.55)	28	2.41	1.73	.10
Rigid Weight Regulation	23.17 (8.07)	20.31 (8.40)	28	2.86	1.84	.08
Weight and Approval	20.50 (6.44)	20.00 (6.77)	28	0.50	0.55	*ns*
Total Score	73.36 (18.72)	68.04 (21.32)	28	5.32	1.62	*ns*

Note: Higher scores on the MAC-R denote the presence of more eating disordered cognitions.

Table 38

Intake and Discharge Patient EAT-26 Paired Samples t-test (n=27)

Subscale	Intake M (SD)	Discharge M (SD)	df	Mean Difference	*t*	*p*
Dieting	16.78 (11.27)	12.78 (11.38)	26	4.00	2.38	.03[a]
Bulimia and Food Preoccupation	5.59 (3.39)	4.11 (3.71)	26	1.48	2.86	.01*
Oral Control	9.37 (4.14)	5.67 (4.60)	26	3.70	4.78	<.000**
Total Score	31.74 (16.47)	22.56 (18.35)	26	9.19	3.54	.002**

*p<.05 (with Bonferroni correction)
**p<.01 (with Bonferroni correction)
[a]Non-significant after Bonferroni correction
Note: Higher scores on the EAT-26 denote the presence of more eating disordered attitudes.

APPENDIX D

Normative and Comparison Data

Table 1

Comparison of ED Sample at Intake (n=41) to Non-Clinical Sample (n=28) for TCFES by One-Sample t-tests

	ED Group	Non-Clinical Group	
Scale	M (SD)	M (SD)	*p*
I. Structure			
Overt Power	3.44 (.90)	3.86 (.93)	.005[a]
Adult Leadership	3.27 (1.05)	3.46 (1.07)	*ns*
Inappropriate Parent Child Coalition	3.49 (1.19)	3.29 (.94)	*ns*
Closeness	3.34 (1.15)	3.64 (.87)	*ns*
II. Autonomy			
Clarity of Expression	3.42 (.92)	3.71 (.81)	.05
Respect for Subjective Reality	3.34 (1.04)	3.50 (.79)	*ns*
Responsibility	3.12 (.93)	3.69 (.91)	.000**
III. Problem-Solving			
Closure	3.29 (1.08)	3.46 (1.11)	*ns*
Negotiation	3.37 (.99)	3.29 (.86)	*ns*
IV. Affect Regulation			
Expressiveness	2.93 (.82)	3.61 (.74)	.000**
Responsiveness	2.88 (.87)	3.54 (.88)	.000**
Positive Regard	3.49 (1.05)	3.57 (.92)	*ns*
Negative Regard	4.05 (1.14)	3.36 (.91)	.000**
Mood and Tone	3.32 (.82)	3.93 (.77)	.000**
Empathy	2.90 (.70)	3.11 (.83)	.07
V. Disagreement/Conflict			
Frequency	4.34 (1.06)	3.11 (.96)	.000**
Affective Quality	4.59 (.87)	3.39 (.79)	.000**
Generalization/Escalation	4.63 (.92)	3.75 (.52)	.000**
Sum of Scales	63.20 (12.52)	68.25 (11.79)	.013[a]
Global Competence	10.54 (3.15)	11.12 (3.94)	*ns*

Note: Higher scores denote greater health.
***p*<.01 (with Bonferroni Correction)
[a]Non-significant after Bonferroni corrections.

Table 2

Comparison of ED Sample at Intake to Non-Clinical Sample on the SFI-II

	ED Group	Normative Sample (Healthiest)		Normative Sample (Least Healthy)	
Subscale	M	M	*p*	M	*p*
Child (n=41)					
Health Competence	2.18	2.06	.37	3.03	.000**
Cohesion	2.70	2.72	.89	3.56	.000**
Conflict	2.18	2.16	.87	3.34	.000**
Leadership	2.21	1.91	.007*	2.63	.000**
Expressiveness	1.99	1.80	.24	2.50	.002*
Parent (n=42)					
Health Competence	2.23	1.96	.03 [a]	3.01	.000**
Cohesion	2.69	2.29	.01 [a]	3.20	.001**
Conflict	2.24	2.17	.45	3.37	.000**
Leadership	2.27	2.00	.03 [a]	2.68	.001**
Expressiveness	1.95	1.65	.04 [a]	2.55	.000**

Note: Lower scores denote greater health.
*$p<.05$, **$p<.01$ (with Bonferroni corrections)
[a] Non-significant after Bonferroni corrections

Table 3

Comparison of ED Group at Intake (n=39) to Non-Clinical Sample (n=475) for EAT-26

	ED Group	Normative Sample	
Subscale	M (SD)	M (SD)	*p*
Dieting	16.97 (12.36)	4.67 (6.23)	.000**
Bulimia and Food Preoccupation	5.97 (4.37)	0.40 (1.37)	.000**
Oral Control	9.67 (4.71)	1.52 (2.22)	.000**
Total Score	32.62 (18.97)	6.60 (8.09)	.000**

***p*<.01 (with Bonferroni corrections)

Note: Higher scores on the EAT-26 denote the presence of more eating disordered attitudes.

Table 4

Comparison of ED Sample (n=41) at Intake to Non-Clinical Sample for MAC-R

	ED Group	Normative Sample	
	M (SD)	M (SD)	*p*
Total Score	74.12 (19.21)	62.40 (15.23)	.000**

**$p<.01$

Note: Higher scores denote the presence of more anorectic cognitions.

APPENDIX E

Exploratory Analyses

Table 1

Independent Samples t-tests of Intake TCFES and Percent Increase in BMI Median Split as an Indicator of Poor versus Good Outcome

Domain/Subscale	n=20 Poor Outcome M (SD)	n=21 Good Outcome M (SD)	df	*t*	*p*
Structure	13.95 (3.86)	13.14 (2.58)	39	.79	*ns*
Autonomy	10.65 (2.30)	9.14 (2.06)	39	2.21	.03[a]
Problem-Solving	6.85 (2.16)	6.48 (1.54)	39	.64	*ns*
Affect Regulation	20.10 (4.66)	19.05 (3.80)	39	.80	*ns*
Conflict	13.05 (3.49)	14.05 (1.69)	39	-1.18	*ns*
Sum of Scales	64.60 (15.09)	61.86 (9.67)	39	.70	*ns*
Global Competence	10.90 (3.34)	10.19 (2.99)	39	.72	*ns*

Note: Higher scores on the TCFES denote greater health.
[a] Non-significant after Bonferroni correction.

Table 2

Independent Samples t-tests of Intake SFI-II and Percent Increase in BMI Median Split as an Indicator of Poor versus Good Outcome

SFI-II Subscale	Poor Outcome M (SD)	Good Outcome M (SD)	df	*t*	*p*
Patient					
Health Competence	2.26 (.99)	2.10 (.70)	39	.60	*ns*
Cohesion	2.76 (.92)	2.65 (.68)	39	.45	*ns*
Conflict	2.19 (.77)	2.17 (.58)	39	1.00	*ns*
Leadership	2.22 (.81)	2.21 (.54)	39	.05	*ns*
Expressiveness	1.93 (1.13)	2.05 (.89)	39	-.38	*ns*
Parent					
Health Competence	2.31 (.89)	2.15 (.69)	40	.67	*ns*
Cohesion	2.70 (.86)	2.68 (.97)	40	.06	*ns*
Conflict	2.23 (.62)	2.25 (.65)	40	-.10	*ns*
Leadership	2.43 (.94)	2.11 (.52)	40	1.36	*ns*
Expressiveness	1.98 (1.07)	1.91 (.67)	40	.24	*ns*

Note: Lower scores on the SFI-II denote greater health.

Table 3

Independent Samples t-tests of Intake EAT-26 and Percent Increase in BMI Median Split as an Indicator of Poor versus Good Outcome

EAT-26 Subscale	n=18 Poor Outcome M (SD)	n=21 Good Outcome M (SD)	df	*t*	*p*
Dieting	20.39 (10.76)	14.05 (13.13)	37	1.63	*ns*
Bulimia and Food Preoccupation	6.78 (4.36)	5.29 (4.36)	37	1.07	*ns*
Oral Control	10.22 (4.39)	9.19 (5.03)	37	.68	*ns*
Total Score	37.39 (17.55)	28.52 (19.60)	37	1.48	*ns*

Note: Higher scores on the EAT-26 denote the presence of more eating disordered attitudes.

Table 4

Independent Samples t-tests of Intake MAC-R and Percent Increase in BMI Median Split as an Indicator of Poor versus Good Outcome

MAC-R Subscale	n=20 Poor Outcome M (SD)	n=21 Good Outcome M (SD)	df	*t*	*p*
Self-Control	33.35 (5.65)	27.62 (8.12)	39	2.61	.06
Rigid Weight Regulation	23.50 (8.77)	21.43 (8.40)	39	.77	*ns*
Weight and Approval	22.65 (7.63)	19.95 (5.61)	39	1.30	*ns*
Total Score	79.50 (18.34)	69.00 (19.04)	39	1.80	.08

Note: Higher scores on the MAC-R denote the presence of more anorectic cognitions.

Table 5

Pearson Correlation between Intake TCFES and Percent Change in EAT-26 Total Score (n=24)

	EAT-26 Total Score	
TCFES Domain	*r*	*p*
Structure	-.09	*ns*
Autonomy	-.31	*ns*
Problem-Solving	-.20	*ns*
Affect Regulation	-.33	*ns*
Conflict	.29	*ns*
Sum of Scales	-.18	*ns*
Global Competence	-.16	*ns*

Table 6

Pearson Correlation between Intake SFI-II and Percent Change in EAT-26 Total Score (n=26)

	EAT-26 Total Score	
SFI-II Subscales	*r*	*p*
Child		
Health Competence	.21	*ns*
Cohesion	.08	*ns*
Conflict	.46	.02[a]
Leadership	-.13	*ns*
Expressiveness	.58	.002*
Parent		
Health Competence	-.03	*ns*
Cohesion	-.04	*ns*
Conflict	-.10	*ns*
Leadership	-.09	*ns*
Expressiveness	-.07	*ns*

*$p<.05$ (With Bonferroni Correction)
[a]Non-significant after Bonferroni Correction

Table 7

Pearson Correlation between Intake MAC-R and Percent Change in EAT-26 Total Score (n=26)

	EAT-26 Total Score	
MAC-R Subscales	*r*	*p*
Self-Control	-.03	*ns*
Rigid Weight Regulation	-.07	*ns*
Weight and Approval	.15	*ns*
Total Score	.01	*ns*

Table 8

Pearson Correlation between Demographic and Illness Variables and Percent Change in EAT-26 Total Score (n=26)

	EAT-26 Total Score	
Demographic/Illness Variables	*r*	*p*
Age	-.27	*ns*
Admit BMI	.01	*ns*
Length of Treatment	.29	*ns*
Percent BMI Gain	.23	*ns*
Duration of ED Episode	.02	*ns*

Table 9

Independent Samples t-tests between Percent Change in EAT-26 Total Score and Demographic Variables

Demographic Variables	Percent Change in EAT-26 Total Score		df	*t*	*p*
	M (SD)	M (SD)			
Ethnicity	**Caucasian (n=21)**	**Other (n=5)**			
	-.33 (.29)	-.15 (.74)	24	-.52	*ns*
ED Diagnosis	**AN, Restricting (n=22)**	**Other (n=4)**			
	-.30 (.43)	-.27 (.21)	24	-.11	*ns*
Gender	**Female (n=22)**	**Male (n=4)**			
	-.27 (.39)	-.43 (.49)	24	-.71	*ns*
Previous ED Treatment	**Yes (n=8)**	**No (n=18)**			
	-.23 (.17)	-.32 (.47)	24	.55	*ns*

Table 10

Linear Regression between Percent Change in EAT-26 Total Score and Intake Patient SFI-II Expressiveness Scale

	β	S.E.	df	t	*p*
Expressiveness	.31	.09	1	3.49	.002**

**$p<.01$

Table 11

Independent Samples t-tests of Intake TCFES and Median Split of Percent Change in EAT-26 Total Score

	Median Split of Percent Change In EAT-26 Total Score				
Domain/Scale	Improved M (SD)	Not improved M (SD)	df	*t*	*p*
Structure	13.50 (2.91)	14.17 (3.30)	22	-.53	*ns*
Autonomy	10.25 (1.91)	10.17 (2.95)	22	.08	*ns*
Problem-Solving	6.83 (1.59)	6.42 (1.68)	22	.62	*ns*
Affect Regulation	20.17 (2.89)	19.00 (4.64)	22	.74	*ns*
Conflict	13.42 (2.28)	14.08 (2.31)	22	-.71	*ns*
Sum of Scales	64.17 (9.07)	63.75 (12.95)	22	.09	*ns*
Global Competence	10.83 (10.75)	10.75 (3.39)	22	.07	*ns*

Table 12

Independent Samples t-tests of Intake SFI-II and Median Split of Percent Change in EAT-26 Total Score

SFI-II Scale	Median Split of Percent Change In EAT-26 Total Score: Improved M (SD)	Median Split of Percent Change In EAT-26 Total Score: Not improved M (SD)	df	*t*	*p*
Child					
Health Competence	2.01 (.64)	2.10 (.65)	24	-.22	*ns*
Cohesion	2.69 (.72)	2.46 (.68)	24	.92	*ns*
Conflict	2.03 (.58)	2.09 (.53)	24	-.72	*ns*
Leadership	2.21 (.50)	2.51 (.52)	24	.37	*ns*
Expressiveness	1.69 (.64)	1.97 (.80)	24	-1.31	*ns*
Parent					
Health Competence	2.22 (.83)	2.10 (.65)	24	.43	*ns*
Cohesion	2.68 (1.02)	2.46 (.68)	24	.64	*ns*
Conflict	2.30 (.75)	2.09 (.53)	24	.83	*ns*
Leadership	1.94 (.77)	2.51 (.52)	24	-2.29	.03[a]
Expressiveness	1.94 (.83)	1.97 (.80)	24	-.10	*ns*

[a] Non-significant after Bonferroni correction

Table 13

Independent Samples t-tests of Intake MAC-R and Median Split of Percent Change in EAT-26 Total Score

	Median Split of Percent Change In EAT-26 Total Score				
MAC-R Scale	Improved M (SD)	Not improved M (SD)	df	*t*	*p*
Self-Control	29.77 (7.56)	30.46 (7.81)	24	-.23	*ns*
Rigid Weight Regulation	23.31 (7.16)	23.46 (7.80)	24	-.05	*ns*
Weight and Approval	18.62 (6.36)	21.69 (6.29)	24	-1.24	*ns*
Total Score	71.69 (17.01)	75.62 (20.02)	24	-.54	*ns*

Table 14

Chi Square of Demographic Variables and Median Split of Percent Change in EAT-26 Total Score

Demographic Variable		Median Split of Percent Change In EAT-26 Total Score: n=13 Improved	Median Split of Percent Change In EAT-26 Total Score: n=13 Not Improved	Chi Square	*p*
Ethnicity	Caucasian	11	10	.25	*ns*
	Other	2	3		
ED Diagnosis	AN, Restricting	11	11	.00	*ns*
	Other	2	2		
Gender	Male	2	2	.00	*ns*
	Female	11	11		
Previous ED Tx	Yes	3	5	.72	*ns*
	No	10	8		

Table 15

Independent Samples t-tests of Demographic/Illness Variables and Median Split of Percent Change in EAT-26 Total Score

	Median Split Percent Change In EAT-26 Total Score				
Demographic/Illness Variable	Improved M (SD)	Not improved M (SD)	df	*t*	*p*
Age in Years	14.46 (1.71)	14.23 (1.30)	24	.39	*ns*
Length of Treatment	30.38 (13.06)	31.62 (16.49)	24	-.21	*ns*
Admit BMI	15.47 (1.45)	15.76 (1.41)	24	-.52	*ns*
Percent Weight Gain	14.64% (9.76%)	14.37% (10.92%)	24	.07	*ns*
Duration of ED Episode (Months)	6.3 (6.06)	9.39 (7.73)	24	-1.04	*ns*

APPENDIX F

Missing Data

Table 1

Reasons for Missing Data at Discharge (Defined as missing TCFES)

Reason	Frequency (%)
Family would not schedule an appointment for discharge TCFES taping but gave no reason for not doing so	8 (19.5%)
Parent too overwhelmed with treatment or busy to schedule an appointment for discharge TCFES taping	2 (4.9%)
Patient refused to participate in discharge TCFES taping due to discomfort with the task	1 (2.4%)
Total Missing Discharge TCFES	**11 (26.8)**

Note: Two families refused to participate in the TCFES taping at intake and discharge. Thus, they were excluded from this frequency table and subsequent analyses between study completers and non-completers.

Table 2

Comparison of Completers vs. Non-completers on Demographic Variables (non-completers defined as missing discharge TCFES)

Demographics		Completers n=30	Non-completers n=11	Chi Square	*p*-value
Gender	Male	4	1	.14	*ns*
	Female	26	10		
Ethnicity	Caucasian	26	9	3.55	*ns*
	Hispanic	1	2		
	African-American	2	0		
	Asian	1	0		
Diagnosis	AN, Restricting	22	8	3.64	*ns*
	AN, Purging	3	0		
	ED NOS	5	3		

Table 3

Comparison of Completers vs. Non-completers on Demographic Variables (non-completers defined as missing discharge TCFES)

Demographics		n=30 Completers	n=11 Non-completers	Chi Square	*p*-value
Ethnicity	Caucasian	26	9	.15	*ns*
	Other	4	2		
Diagnosis	AN, Restricting	22	8	.00	*ns*
	Other	8	3		

Note: Ethnicity-"Other" category includes Hispanics, African-Americans, and Asians.
Diagnosis-"Other" category includes AN, Purging and ED NOS.

Table 4

Comparison of Completers vs. Non-completers on Outcome Measures (non-completers defined as missing discharge TCFES)

		Completers	Non-completers	Chi Square	*p*-value
CGI-I Rater 1	Improved	17	9	2.20	*ns*
	Not Improved	13	2		
CGI-I Rater 2	Improved	21	8	.03	*ns*
	Not Improved	9	3		
Improvement in BMI (1 Standard Deviation)	Improved	21	7	.15	*ns*
	Not Improved	9	4		

Table 5

Independent t-tests of Demographic Variables and Completers vs. Non-completers (defined as missing discharge TCFES data)

Demographic Variable	n=30 Completers M (SD)	n=11 Non-completers M (SD)	df	*t*	*p*
Intake BMI	15.63 (1.34)	15.61 (1.56)	39	-.03	*ns*
Discharge BMI	17.61 (1.18)	17.52 (1.65)	39	-.19	*ns*
Percent BMI Gain	13.18 (9.11)	12.68 (10.17)	39	-.15	*ns*
LOT	28.10 (13.59)	30.75 (16.30)	39	.52	*ns*
Age	14.27 (1.84)	13.91 (1.58)	39	-.57	*ns*

Table 6

Independent t-tests of Intake TCFES and Completers versus Non-completers (defined as missing all discharge TCFES data)

Domain/Subscale	n=30 Completers M (SD)	n=11 Non-completers M (SD)	df	*t*	*p*
Structure	13.73 (3.32)	13.00 (3.13)	39	-.64	*ns*
Autonomy	10.01 (2.51)	9.27 (1.42)	39	-1.03	*ns*
Problem-Solving	6.70 (1.93)	6.55 (1.70)	39	-.23	*ns*
Affect Regulation	19.73 (4.32)	19.09 (4.09)	39	-.43	*ns*
Conflict	13.57 (2.87)	13.55 (2.42)	39	-.02	*ns*
Sum of Scales	63.80 (13.03)	61.55 (11.44)	39	-.51	*ns*
Global Competence	10.83 (3.20)	9.73 (3.00)	39	-1.00	*ns*

Note: Higher scores on the TCFES denote greater health.

Table 7

Independent t-tests of Intake SFI-II and Completers versus Non-completers (defined as missing discharge TCFES data)

Domain/Subscale	Completers M (SD)	Non-completers M (SD)	df	*t*	*p*
Patient (n=29, 10)					
Health Competence	2.00 (.68)	2.54 (1.14)	37	1.43	*ns*
Cohesion	2.54 (.70)	2.98 (.93)	37	1.58	*ns*
Conflict	2.05 (.51)	2.39 (.98)	37	1.05	*ns*
Leadership	2.16 (.63)	2.33 (.86)	37	.68	*ns*
Expressiveness	1.81 (.86)	2.42 (1.36)	37	1.31	*ns*
Parent (n=30, 10)					
Health Competence	2.10 (.79)	2.60 (.68)	38	1.77	.08
Cohesion	2.59 (.95)	2.92 (.97)	38	.96	*ns*
Conflict	2.14 (.63)	2.51 (.59)	38	1.64	*ns*
Leadership	2.20 (.76)	2.53 (.83)	38	1.17	*ns*
Expressiveness	1.89 (.88)	2.10 (.91)	38	.66	*ns*

Note: Lower scores on the SFI-II denote greater health.

Table 8

Independent t-tests of Intake EAT-26 and Completers versus Non-completers (defined as missing discharge TCFES data)

Domain/Subscale	n=28 Completers M (SD)	n=9 Non-completers M (SD)	df	*t*	*p*
Dieting	16.04 (11.02)	17.22 (16.51)	35	.25	*ns*
Bulimia and Food Preoccupation	5.93 (4.03)	5.44 (5.68)	35	-.28	*ns*
Oral Control	9.67 (4.55)	8.78 (5.43)	35	-.49	*ns*
Total Score	31.64 (16.41)	31.44 (26.25)	35	-.02	*ns*

Note: Higher scores on the EAT-26 denote the presence of more eating disordered attitudes.

Table 9

Independent t-tests of Intake MAC-R and Completers versus Non-completers (defined as missing discharge TCFES data)

Subscale	n=29 Completers M (SD)	n=10 Non-completers M (SD)	df	*t*	*p*
Self-Control	30.00 (7.65)	30.40 (7.79)	37	.14	*ns*
Rigid Weight Regulation	22.62 (8.72)	21.80 (9.00)	37	-.26	*ns*
Weight and Approval	21.41 (7.22)	21.60 (5.93)	37	.07	*ns*
Total Score	74.03 (19.92)	73.80 (19.97)	37	-.03	*ns*

Note: Higher scores on the MAC-R denote the presence of more anorectic cognitions.

APPENDIX G

Correlations Among Dependent Variables

Table 1

Bivariate Pearson Correlations between Intake TCFES and Intake Patient SFI-II (n=39)

		Patient SFI-II Scales				
TCFES Domains		Health Competence	Cohesion	Conflict	Leadership	Expressiveness
Structure	*r (p)*	-.27 (.10)	-.17 (*ns*)	-.38 (.02) [a]	-.47 (.003) [a]	-.41 (.01) [a]
Autonomy	*r (p)*	-.23 (*ns*)	-.20 (*ns*)	-.39 (.02) [a]	-.37 (.02) [a]	-.41 (.01) [a]
Problem-Solving	*r (p)*	-.09 (*ns*)	.07 (*ns*)	-.19 (*ns*)	-.34 (.04) [a]	-.38 (.02) [a]
Affect Regulation	*r (p)*	-.33 (.04) [a]	-.22 (*ns*)	-.49 (.001) [a]	-.55 (<.000)*	-.27 (.09)
Conflict	*r (p)*	-.20 (*ns*)	-.09 (*ns*)	-.23 (*ns*)	-.44 (.01) [a]	-.46 (.003) [a]
Sum of Scales	*r (p)*	-.28 (.08)	-.17 (*ns*)	-.41 (.01) [a]	-.52 (.001) [a]	-.21 (*ns*)
Global Competence	*r (p)*	-.36 (.02) [a]	-.29 (.08)	-.43 (.01) [a]	-.58 (<.000)*	-.42 (.008) [a]

*$p<.05$ (with Bonferroni corrections)
[a]Non-significant after Bonferroni corrections

Table 2

Bivariate Pearson Correlations between Intake TCFES and Intake Parent SFI-II (n=40)

		Parent SFI-II Scales				
TCFES Domains		Health Competence	Cohesion	Conflict	Leadership	Expressiveness
Structure	*r (p)*	-.23 (*ns*)	-.14 (*ns*)	-.21 (*ns*)	-.19 (*ns*)	-.31 (.05)
Autonomy	*r (p)*	-.16 (*ns*)	-.12 (*ns*)	-.28 (.08)	-.15 (*ns*)	-.26 (.10)
Problem-Solving	*r (p)*	-.04 (*ns*)	.09 (*ns*)	.02 (*ns*)	-.32 (.05)	-.06 (*ns*)
Affect Regulation	*r (p)*	-.23 (*ns*)	-.10 (*ns*)	-.22 (*ns*)	-.32 (.05)	-.27 (.10)
Conflict	*r (p)*	-.09 (*ns*)	.03 (*ns*)	-.16 (*ns*)	-.33 (.04) [a]	-.06 (*ns*)
Sum of Scales	*r (p)*	-.19 (*ns*)	-.07 (*ns*)	-.21 (*ns*)	-.30 (.06)	-.25 (*ns*)
Global Competence	*r (p)*	-.23 (*ns*)	-.10 (*ns*)	-.27 (.10)	-.24 (*ns*)	-.30 (.06)

[a]Non-significant after Bonferroni corrections

Table 3

Bivariate Pearson Correlations between Intake TCFES and Intake MAC-R (n=39)

		MAC-R Scales			
TCFES Domains		Self-Control	Rigid Weight Regulation	Weight and Approval	Total
Structure	*r (p)*	.21 (*ns*)	.24 (*ns*)	-.18 (*ns*)	.12 (*ns*)
Autonomy	*r (p)*	.28 (.08)	.31 (.06)	-.16 (*ns*)	.19 (*ns*)
Problem-Solving	*r (p)*	.11 (*ns*)	.25 (*ns*)	-.03 (*ns*)	.14 (*ns*)
Affect Regulation	*r (p)*	.19 (*ns*)	.31 (.06)	-.20 (*ns*)	.14 (*ns*)
Conflict	*r (p)*	-.07 (*ns*)	.06 (*ns*)	-.30 (.06)	-.10 (*ns*)
Sum of Scales	*r (p)*	.17 (*ns*)	.27 (.10)	-.22 (*ns*)	.11 (*ns*)
Global Competence	*r (p)*	.12 (*ns*)	.20 (*ns*)	-.17 (*ns*)	.07 (*ns*)

Table 4

Bivariate Pearson Correlations between Intake TCFES and Intake EAT-26 (n=37)

		EAT-26 Scales			
TCFES Domains		Dieting	Bulimia and Food Preoccupation	Oral Control	Total
Structure	*r (p)*	.27 (.10)	.29 (.08)	.34 (.04) [a]	.33 (.05)
Autonomy	*r (p)*	.41 (.01) [a]	.48 (.003) [a]	.37 (.03) [a]	.47 (.003) [a]
Problem-Solving	*r (p)*	.14 (*ns*)	.19 (*ns*)	.20 (*ns*)	.19 (*ns*)
Affect Regulation	*r (p)*	.32 (.05)	.27 (*ns*)	.33 (.05)	.36 (.03) [a]
Conflict	*r (p)*	.09 (*ns*)	.15 (*ns*)	.23 (*ns*)	.15 (*ns*)
Sum of Scales	*r (p)*	.29 (.08)	.32 (.06)	.35 (.04) [a]	.35 (.03) [a]
Global Competence	*r (p)*	.21 (*ns*)	.20 (*ns*)	.33 (.04) [a]	.27 (*ns*)

[a]Non-significant after Bonferroni corrections

Table 5

Bivariate Pearson Correlations between Intake Patient SFI-II and Intake Parent SFI-II (n=40)

		Parent SFI-II Scales				
Patient SFI-II Scales		Health Competence	Cohesion	Conflict	Leadership	Expressiveness
Health Competence	*r (p)*	.59 (<.000)*	.44 (.01) [a]	.47 (.002) [a]	.18 (*ns*)	.53 (<.000)*
Cohesion	*r (p)*	.54 (<.000)*	.46 (.003) [a]	.49 (.001)*	.11 (*ns*)	.49 (.001)*
Conflict	*r (p)*	.53 (<.000)*	.41 (.009) [a]	.41 (.01) [a]	.05 (*ns*)	.46 (.003) [a]
Leadership	*r (p)*	.41 (.01) [a]	.29 (.07)	.37 (.02) [a]	.33 (.04) [a]	.36 (.02) [a]
Expressiveness	*r (p)*	.50 (.001)*	.29 (.07)	.41 (.01) [a]	.23 (*ns*)	.49 (.001)*

**p*<.05 (with Bonferroni corrections)
[a]Non-significant after Bonferroni corrections

Table 6

Bivariate Pearson Correlations between Intake Patient SFI-II and Intake MAC-R (n=41)

		Patient SFI-II Scales				
MAC-R Scales		Health Competence	Cohesion	Conflict	Leadership	Expressiveness
Self-Control	*r (p)*	.34 (.03) [a]	.32 (.05)	.28 (.08)	.25 (*ns*)	.15 (*ns*)
Rigid Weight Regulation	*r (p)*	.19 (*ns*)	.20 (*ns*)	.08 (*ns*)	.03 (*ns*)	.09 (*ns*)
Weight and Approval	*r (p)*	.42 (.01) [a]	.34 (.03) [a]	.41 (.01) [a]	.31 (.05)	.33 (.04) [a]
Total	*r (p)*	.37 (.02) [a]	.33 (.03) [a]	.29 (.07)	.22 (*ns*)	.21 (*ns*)

[a]Non-significant after Bonferroni corrections

Table 7

Bivariate Pearson Correlations between Intake Patient SFI-II and Intake EAT-26 (n=39)

		Patient SFI-II Scales				
EAT-26 Scales		Health Competence	Cohesion	Conflict	Leadership	Expressiveness
Dieting	*r (p)*	.26 (*ns*)	.30 (.07)	.15 (*ns*)	.06 (*ns*)	.04 (*ns*)
Bulimia and Food Preoccupation	*r (p)*	.34 (.03) [a]	.31 (.06)	.19 (*ns*)	.12 (*ns*)	.19 (*ns*)
Oral Control	*r (p)*	-.01 (*ns*)	-.02 (*ns*)	-.04 (*ns*)	-.15 (*ns*)	-.02 (*ns*)
Total	*r (p)*	.25 (*ns*)	.26 (*ns*)	.13 (*ns*)	.03 (*ns*)	.06 (*ns*)

[a]Non-significant after Bonferroni corrections

Table 8

Bivariate Pearson Correlations between Parent SFI-II and Intake MAC-R (n=40)

		Parent SFI-II Scales				
MAC-R Scales		Health Competence	Cohesion	Conflict	Leadership	Expressiveness
Self-Control	*r (p)*	.09 (*ns*)	-.02 (*ns*)	-.04 (*ns*)	-.06 (*ns*)	.01 (*ns*)
Rigid Weight Regulation	*r (p)*	.08 (*ns*)	-.01 (*ns*)	-.07 (*ns*)	-.12 (*ns*)	.14 (*ns*)
Weight and Approval	*r (p)*	.21 (*ns*)	.16 (*ns*)	.05 (*ns*)	.20 (*ns*)	.30 (.06)
Total	*r (p)*	.14 (*ns*)	.04 (*ns*)	-.03 (*ns*)	-.01 (*ns*)	.17 (*ns*)

Table 9

Bivariate Pearson Correlations between Parent SFI-II and Intake EAT-26 (n=38)

		Parent SFI-II Scales				
EAT-26 Scales		Health Competence	Cohesion	Conflict	Leadership	Expressiveness
Dieting	*r (p)*	.04 (*ns*)	-.03 (*ns*)	-.07 (*ns*)	-.13 (*ns*)	<-.01 (*ns*)
Bulimia and Food Preoccupation	*r (p)*	.11 (*ns*)	.07 (*ns*)	-.09 (*ns*)	-.03 (*ns*)	.08 (*ns*)
Oral Control	*r (p)*	-.21 (*ns*)	-.13 (*ns*)	-.22 (*ns*)	-.26 (*ns*)	-.20 (*ns*)
Total	*r (p)*	-.01 (*ns*)	-.03 (*ns*)	-.12 (*ns*)	-.15 (*ns*)	-.03 (*ns*)

Table 10

Bivariate Pearson Correlations between MAC-R and EAT-26

		MAC-R Scales			
EAT-26 Scales		Self-Control	Rigid Weight Regulation	Weight and Approval	Total
Dieting	*r (p)*	.78 (<.000)*	.76 (<.000)*	.26 (*ns*)	.74 (<.000)*
Bulimia and Food Preoccupation	*r (p)*	.60 (<.000)*	.66 (<.000)*	.32 (.05)	.64 (<.000)*
Oral Control	*r (p)*	.35 (.03) [a]	.35 (.03) [a]	-.07 (*ns*)	.27 (.10)
Total	*r (p)*	.73 (<.000)*	.73 (<.000)*	.22 (*ns*)	.69 (<.000)*

*p<.05 (with Bonferroni corrections)
[a]Non-significant after Bonferroni corrections

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VITAE

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