THE RELATIONSHIP BETWEEN MATERNAL PSYCHOPATHOLOGY AND ACUTE TREATMENT OUTCOMES OF CHILDREN AND ADOLESCENTS DIAGNOSED WITH ANOREXIA NERVOSA

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by

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Recent studies have suggested that maternal psychopathology influences the psychiatric status of children. However, there is a lack of research in the eating disorder literature pertaining to the impact of maternal psychopathology, specifically related to depression and eating disordered cognitions, on a child or adolescent diagnosed with anorexia nervosa. Therefore, this study investigated the

relationship between maternal psychopathology and eating disorders. Specifically, this study examined the relationship between maternal eating disordered cognitions and depression and severity of child's psychopathology, as well as the relationship between maternal eating disordered cognitions and acute treatment outcomes of a child or adolescent diagnosed with anorexia nervosa. The sample consisted of 43 children and adolescents between the ages of ten and seventeen years of age, with a diagnosis of anorexia nervosa or eating disorder not otherwise specified. All subjects were being treated at Children's Medical Center psychiatric unit as an inpatient or partial hospitalization patient. At entry to treatment, all patients were administered a structured clinical interview to obtain comprehensive psychiatric diagnoses. Additionally, subjects and their mothers or female caregivers completed self-report measures of eating disordered cognitions and depressive symptomatology. The relationship between maternal psychopathology, child eating disordered psychopathology, and relationship to treatment outcome was assessed. Results revealed a significant relationship between maternal depressive symptoms and the severity of the child's eating disordered cognitions. However, despite the expectation that the degree of maternal eating disordered cognitions at admission would predict the child's outcome over an acute period of treatment, no significant relationship was found. Results from this study suggest that maternal depression may play a more

influential role in the child's eating disorder psychopathology than maternal eating disordered cognitions.

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CHAPTER I

INTRODUCTION

Statement of the Problem

Anorexia nervosa is a serious psychiatric disorder that is associated with the highest mortality risk among all mental disorders and is one of the most difficult to treat (Crisp, Callender, Halek, & Hsu, 1992). Adolescence is the life stage associated with the greatest risk of onset of anorexia nervosa due to the convergence of physical changes and psychosocial challenges at this time (Gowers & Shore, 2001). However, recent reports have shown an increase in children as young as five years of age expressing weight and shape concerns (Feldman, Feldman, & Goodman, 1998). The lifetime prevalence rate of eating disorders among female adolescents is 23 per 1,000. Furthermore, female children and adolescents before the age of 19 years have the highest rate of first incidence of anorexia nervosa at approximately 1.3% (Lewinsohn, Striegel-Moore, & Seely, 2000).

Anorexia nervosa is frequently associated with a chronic course and factors that determine the course and outcome are largely unknown as a result of the disorders heterogeneity. In fact, one of the prominent features of the disorder is its variable course and outcome (Pike, 1998). However, a recent review of the

literature found several factors to be predictive of anorexia nervosa outcomes: type of eating disorder, body weight, duration of illness, age of onset, familial disturbances, and comorbid psychiatric diagnoses (Herzog, Dorer, Keel, Selwyn, Ekeblad, & Flores, 1999). Unfortunately, the ability to generalize across outcome studies is extremely difficult due to the varied methodological procedures employed across studies including: sample characteristics and size, clarity of diagnostic criteria, assessment procedures, outcome criteria, and follow-up procedures (Pike, 1998).

Comorbid psychiatric diagnoses are also reported in a large percentage of patients with anorexia nervosa. According to Herpetz-Dahlmann, Wewetzer, Schulz, and Remschmidt (1995), anxiety and affective disorders are the most common comorbid psychiatric diagnoses associated with anorexia nervosa in both the acute and long-term phases of the disorder. Some researchers estimate that approximately 45% of anorexic patients report depressive symptoms (Santonastaso, Pantaro, Panarotto, & Silverstri, 1991). Additionally, researchers have reported a significant association between outcome of anorexia nervosa and depressive psychopathology, with patients with a poorer outcome displaying increased levels of comorbid psychopathology (Herpetz-Dahlmann, Wewetzer, Schulz, & Remschmidt, 1995).

Although maternal psychopathology has been found to be important to the psychiatric status of children (Weissman, et al., 2006), there is a lack of research

in the eating disorder literature pertaining to the impact of maternal psychopathology (i.e. depression and eating disordered cognitions) on a child or adolescent with anorexia nervosa. The aim of the present study was to assess the relationship between maternal psychopathology and severity of psychopathology in the child, specifically related to anorexia nervosa and depression. Furthermore, this study aimed to identify relationships between maternal psychopathology and acute treatment outcomes in a homogeneous group of severely ill patients requiring hospitalization for anorexia nervosa.

CHAPTER II

LITERATURE REVIEW

Significance and Background

Eating disorders hinder physical, emotional, and behavioral growth in children and adolescents, and can be life-threatening (Robin, Gilroy, & Dennis, 1998). In addition, eating disorders are associated with substantial impairment in health and interpersonal adjustment, and have elevated relapse rates (Herzog et al., 1999). The prevalence of anorexia nervosa in 15 to 19 year old girls has been reported to be 0.48% (Robin, Gilroy, & Dennis, 1998), and the prevalence of eating disorders among children is lower. However, an exact figure remains unknown as a result of a lack of methodologically sound epidemiological studies (Lask & Bryant-Waugh, 1993). According to Lewinsohn, Striegel-Moore, and Seeley (2000), adolescent females make up the population group with the highest rate of current eating disorders. Eating disorders are less common among males than females, and it has been estimated that a female to male ratio of 10:1 exists (Garfinkel et al., 1995). Anorexia nervosa is not restricted to middle and upper class Caucasian children and adolescents, but can arise in children and adolescents from a range of cultural, ethnic, and socioeconomic backgrounds (Lask & Bryant-Waugh, 1993).

Anorexia nervosa has been depicted in medical literature for approximately 300 years, but was not recognized as a formal diagnosis until 1980, when it was included in the Diagnostic and Statistical Manual of Mental Disorders- 3rd Edition (DSM) (Mizes, 1998). The DSM-IV (American Psychiatric Association, 1994) lists the following criteria for a diagnosis of anorexia nervosa: refusal to maintain body weight at or above a minimum normal weight for age and height (less than 85% of expected) or failure to grow, a strong fear of gaining weight or becoming fat, body image disturbance, excessive influence of body weight on self – assessment, or denial of the seriousness of the low weight, and the absence of three successive menstrual cycles in postmenarcheal females (American Psychiatric Association, 1994).

Special Considerations for Children and Adolescents

The DSM-IV criteria for anorexia nervosa are not sensitive with regard to development and other factors intrinsic in child and adolescent eating disorders, and strictly applying these criteria may result in an underestimation of appropriate diagnosis. For example, numerous children and adolescents with eating disorders will not exhibit weight loss, and a physician must determine whether they have failed to make the expected weight gain for a particular growth period (Robin, Gilroy, & Dennis, 1998). According to Beaumont, Al-Alami, and Touyz (1988),

calculating an individual's weight to height association or body mass index (BMI), and then comparing the calculated BMI to national reference data for children and adolescents is the favored method of measuring body size and gaining insight into whether the individual has failed to meet growth requirements.

In addition, the absence of three consecutive menstrual cycles as a diagnostic criterion proves problematic in children and adolescents. Specifically, many young adolescent girls have not yet experienced puberty and therefore have never had a menstrual period. In addition, menstrual cycles are often unpredictable for some time after menarche begins. Finally, anorexia may delay the onset of puberty and menarche in young adolescent girls (Robin, Gilroy, & Dennis, 1998).

Furthermore, children do not possess the cognitive aptitude to use abstract reasoning and therefore may fail to meet the criterion of an intense fear of weight gain and disturbances in body weight perception (Robin, Gilroy, & Dennis, 1998). In a study of eating disordered children ranging in age from 5 to 11 years, Jaeffe and Singer (1989), found that none of the children expressed a fear of being fat.

In addition, medical consequences may be permanent in severe cases of anorexia nervosa in children and adolescents that have not yet gone through puberty (Fisher et al., 1995). For example, growth retardation may result in short stature if deprivation of food is not brought to an end before the closure of the

epiphyses. Additionally, sterility and secondary sex characteristics that are not fully developed may be a product of the delay of puberty caused by anorexia nervosa. Furthermore, impaired gains in peak bone mass during childhood and adolescence may lead to osteoporosis in adulthood (Robin, Gilroy, & Dennis, 1998).

On a more positive note, research suggests that the majority of medical consequences of disordered eating improve with nutritional rehabilitation.

Additionally, intense treatment of early onset anorexia nervosa can avert many of the more severe consequences (Robin, Gilroy, & Dennis, 1998). As such, clinicians must be familiar with the signs of disordered eating in children and adolescents and promptly begin treatment, often before they meet full DSM-IV diagnostic criteria (Lask & Bryant-Waugh, 1993).

Further research in this field is imperative as a result of the numerous complications in the eating disorder population of children and adolescents. There is a lack of research specifically in the area of children and adolescents with eating disorders, relapse rates are high, and the likelihood of medical problems, as well as mortality, is significant. Therefore, it is critical to clarify the relationship between many of the variables that may result in such negative outcomes.

Anorexia Nervosa and Family Characteristics

Research into the etiology of eating disorders has emphasized a multifactorial model and many studies have attempted to determine factors that contribute to eating disorders. Some of the most prominent factors associated with eating disorders include: sociocultural, familial, and individual factors such as personality, cognition, and physiology (Polivy & Herman, 2002). The family system has been given a prominent role in developmental theories of eating disorders and many researchers have examined a number of family variables that may be associated with families with an eating disordered child.

Dysfunctional family interaction patterns have been well documented in patients with anorexia nervosa (Selvini-Palazzoli, 1974). Early family systems research by Minuchin and colleagues (1978), described the family of the anorexic patient as enmeshed, rigid, overprotective, and conflict avoidant. Other studies have also found that families with a child with anorexia nervosa displayed rigidity and dependency (Steinberg & Phares, 2001). Family factors are also believed to maintain eating disorders through inconsistent parenting, poorly resolved marital conflict, overly involved relationships, problems with communication, and a struggle for control between child and parents (Fosson, Knibs, & Bryant-Waugh, 1987; Lask & Bryant-Waugh, 1993).

Parenting

The parents of eating disordered patients have been described in the literature as conflict avoidant, overly involved, lacking boundaries, and as having poor conflict resolution skills (van Furth et al., 1996). According to Calam and colleagues (1990), overprotection implies excessive involvement on the part of the parents in the affairs of their children. As a result, children are prevented from developing autonomy, and a lack of perceived control over their lives ensues.

Researchers view this lack of perceived control as an important factor in the development of eating disorders (Slade, 1982). In support of this theory, researchers using the Parental Bonding Instrument (PBI) have found that perceived overprotection and low parental warmth are associated with eating disorders (Calam, Waller, Slade, & Newton, 1990).

Other studies have supported models in which early family experiences result in the development of unhealthy core beliefs, which may lead to the development of eating disorders (Cooper, 1997; Leung et al., 2000). Leung, Thomas, and Waller (2000), have reported a strong association between unhealthy parental bonding behaviors and dysfunctional core beliefs in anorexic women. For example, low perceived maternal care is associated with anorexic women developing the belief that they are defective, and they, in turn, set unrealistically high standards for themselves. Adolescents who perceive low family

communication, low parental caring, and low parental expectations are at increased risk for developing an eating disorder (Haudek et al., 1999). On the other hand, perceived parental encouragement of autonomy is related to a reduced amount of dieting behavior and may serve as a protective role against eating disorders (Strong & Huon, 1998).

Enmeshment

Boundary dissolution and enmeshment, characterized by a lack of differentiation and individuation among family members, is cited in the literature as an important family variable with regard to the development and maintenance of anorexia nervosa (Minuchin, Rosman, & Baker, 1978). Wallin and Hansson (1999) found anorexic families to be rated by observers as more enmeshed than non-clinical families. Empirical studies have produced mixed results, with some studies providing support for the presence of boundary related problems in anorexic families (Kog & Vandereycken, 1989; Kog, Vertommen, & Vandereycken, 1987), while other research has found no evidence of such boundary problems in these families (Dare et al., 1987; Harding & Lachenmeyer, 1991; Waller et al., 1990). Additionally, researchers have found anorexic women to be less individuated and more deferential to their parents than control subjects (Smolak & Levine, 1993; Humphrey, 1987) and have reported that parents tend to

watch and manage their anorexic daughters more so than control parents (Humphrey, 1989).

Although boundary dissolution has been implicated in the development and maintenance of anorexia nervosa, it does not appear to be specific to this disorder. Researchers have reported boundary problems in a number of psychiatric and childhood adjustment problems including: depression, anxiety, and identity disturbances (Fish et al., 1991; Fullinwider-Bush & Jacobvitz, 1993; Jacobvitz & Bush, 1996). Thus, research suggests that boundary problems may increase a child's risk for some form of psychopathology, one such form being an eating disorder, but that boundary problems are likely not a risk factor specific to eating disorders (Rowa et al., 2001).

Rowa, Kerig, and Gellar (2001) examined parent-child boundary dissolution in 30 anorexic women and compared them to a control group of 65 undergraduate women. The study utilized the Parent-Child Boundaries Scale (PBS; Kerig & Brown, 1996) in order to assess intergenerational boundary problems. Results indicated that anorexic women reported more boundary problems with mothers and fathers than did the control group of non-eating disordered women. Rowa, Kerig, and Gellar (2001) concluded that boundary problems do not seem to be specific to anorexia, but may be related to anorexia through the depressive affect that often accompanies the disorder.

Communication and Conflict Avoidance

A significant relationship between dysfunctional patterns of familial communication and eating disorders has been reported in the literature. For example, women who report high levels of eating disordered attitudes and behaviors have also reported their families to be inadequate in their ability to accurately express thoughts and emotions, provide empathy, resolve conflict, and take responsibility for their actions (Reeves & Johnson, 1992). Studies which specifically examined patterns of familial communication among eating disordered and non-eating disordered groups have found anorexic women to perceive their parents as more blaming, rejecting, and neglectful toward them compared to non-eating disordered women (Humphrey, 1986, 1988).

Research examining perceptions of family interactions among eating disordered patients have reported inconsistent results. For instance, studies have reported anorexic families to perceive members as less supportive, with limited reinforcement for the expression of feelings compared to control subjects (Stern et al., 1989). Conversely, other researchers have reported that despite the presence of boundary problems, instability, and conflict avoidance, anorexic women perceived their family as stable and cohesive (Kog & Vandereycken, 1989).

Moreno and colleagues (2000) investigated family dynamics and communication patterns among eating disordered women and non-eating

disordered women who served as controls. The study utilized the Parent-Adolescent Communication Scale (PACS; Barnes & Olson, 1982) to assess perceptions of intra family and intergenerational communication. Results revealed a lack of significant differences between the anorexic and control groups, which is consistent with some research findings (Humphrey, 1988) but not others (Stern et al., 1989). Moreno and colleagues (2000), noted that one explanation for their findings is that anorexics may fail to report problems in familial communication because of psychological traits associated with the disorder (e.g., denial). Additionally, the subjects were inpatients who typically suffer from more severe cases of anorexia, and subsequently may exhibit greater denial of family communication patterns. Thus, anorexics in the study may have, paradoxically, appeared more normal in their family communication patterns due to their more severe degree of pathology (Moreno, Selby, Aved, & Besse, 2000).

Researchers who have examined conflict within eating disordered families have consistently found the avoidance of overt expression of conflict by anorexics (Dare, Le Grange, Eisler, & Rutherford, 1994; Shugar & Krueger, 1995).

Avoiders tend to disclose little and focus on getting away from conflict, rather than getting actively involved (Botta & Dulmano, 2002). Despite the fact that all family members may reside in the same communication environment, their perceptions of family communication and conflict pattern may differ. In fact, research suggests that interpretations about the family communication can vary

according to which parent is involved (Austin, 1993). While one parent may encourage communication, the other may handle conflicts differently, resulting in mixed messages (Botta & Dulmano, 2002). As a result, it is important to examine family communication within dyads.

Botta and Dumalo (2002) surveyed 210 undergraduate women in an attempt to examine the likelihood that father-daughter communication and conflict resolution are related to eating disordered behaviors. They found that a lack of verbal assertiveness, through communication and conflict resolution with their father, adds to the probability that young women will develop anorexic behaviors. Additionally, their results indicated that skilled conflict resolution and open communication between fathers and daughters might offset the potential for eating disorders. Lattimore, Wagner, and Gowers (2000) observed adolescents with anorexia nervosa during a series of family problem solving tasks with their mothers. Their results indicated that anorexic dyads demonstrated more destructive communication than comparison dyads. For example, anorexic dyads showed more frequent disagreement, blame, mindreading, and negative affect. Results from this study support previous findings that deficiencies in communication skills exist in anorexic families, but do not, however, corroborate previous findings that describe anorexic families as conflict avoidant.

Perceived Family Functioning

Researchers (Kog & Vandereyecken, 1989; Waller, Calam, & Slade, 1989) and clinicians (Bruch, 1973; Palazzoli, 1974) have observed an association between abnormal patterns of perceived family functioning and eating disorders. Additionally, researchers have found that perception of family functioning may differ between members (Guttman & Laporte, 2002). For instance, Waller and colleagues (1990) used a questionnaire to compare eating disordered women's perception of family functioning with their parents', in an attempt to identify whose perception of family interaction would be the best predictor of the existence of eating disorders. Results indicated that the mothers', fathers', and daughters' perceptions of family interaction varied greatly, as did their abilities to predict the presence or absence of eating disorders in the daughter. The daughters' ratings of family functioning were found to be the best predictors of the presence of an eating disorder. Mothers' ratings were the next best in their predictive ability, while fathers' ratings had virtually no predictive power. Other studies have produced similar results in support of the notion that anorexic family members' perceptions of family functioning are frequently not in agreement. For example, Gowers and North (1999) found that adolescent patients and clinicians rated family functioning more severely than parents, suggesting that patients are more perceptive with regard to family functioning.

Conversely, other researchers have found that eating disorder patients and their parents do not differ in their reports of family functioning. For instance, Guttman and Laporte (2002) found that members of families whose daughters were restricting anorexics were in agreement regarding perceptions of family functioning. Similarly, North, Gowers, and Byram (1995) reported that anorexic adolescents and their mothers rated their family functioning as normal, while objective interview ratings suggested the families were dysfunctional. North and colleagues (1995) suggested that this result might have been obtained as a result of the commonly noted tendency for families with an anorexic member to unconsciously deny difficulties within the family (Crisp, 1980). Alternately, Guttman and Laporte (2002), have suggested that the agreement between parent and child perception of family functioning may reflect the cohesion and enmeshment that has been described in the literature as characteristic of anorexic families (Minuchin, 1974; Kog & Vanderecken, 1985).

Maternal Factors

Mother-Daughter Interactions and Eating Disorders

Research has often focused on mother-daughter relationships as an explanation for the occurrence of weight concern and eating disorders among

young women. It has been suggested that mothers may convey their own weight concerns to their daughters (Ogden & Steward, 2000). Hall and Brown (1982) reported that mothers whose daughters were anorexic showed greater body dissatisfaction than mothers of non-eating disordered daughters. Similarly, Steiger, Stotland, Ghandirian, and Whitehead (1994) reported a direct association between mothers' and daughters' levels of weight concerns.

Numerous aspects of the mother-daughter relationship have been studied in families with eating disordered children, in an attempt to identify aspects of the relationship that may relate to the development of weight concerns in the eating disordered daughter. For example, Smith, Mullis, and Hill (1995) suggested that an overly close relationship between mother and daughter might lead to an enmeshed relationship and subsequently, problems with separation in adolescence. Bruch (1973) postulated that anorexia might be the result of the daughter's struggle to develop her own self-identity within a mother-daughter relationship that limits her autonomy. Additionally, Ogden and Steward (2000) found that daughters reported higher levels of body dissatisfaction when their mothers reported lower beliefs in their own and their daughter's autonomy. Furthermore, when the mother-daughter relationship was enmeshed, with unclear boundaries between the two, the daughters also reported higher levels of body dissatisfaction. This research suggests that the complex interactions of the

mother-daughter relationship may facilitate weight concerns in the daughter (Ogden & Steward, 2000).

Maternal Characteristics

Mothers may play an integral role in the transmission of cultural values regarding weight, shape, and appearance to their daughters and subsequently may influence their daughter's pathology (Hill & Franklin, 1998). Mothers with an eating disordered daughter believed that their daughters should lose more weight and described them as less attractive than comparison mothers did (Pike & Rodin, 1991). Other researchers have investigated the contributions of mothers' direct comments about their child's weight and the modeling of weight concerns on children's weight concerns and weight loss efforts. Smolak, Levine, and Schermeer (1999) found direct maternal comment on a child's weight to be a significant predictor of weight loss attempts and fat concerns in elementary school children. Although direct comments were found to be more influential than modeling, a daughter's concern about being or becoming too fat was found to be related to the mother's complaints about her own weight as well.

In addition, mothers of patients with an eating disorder may be more disgruntled with the general functioning of the family system, and are more eating disordered themselves, when compared with mothers who do not have a child or

adolescent with an eating disorder (Hill & Franklin, 1998; Pike & Rodin, 1991). Other researchers have supported this finding, and have found that mothers who have daughters with eating disorders have demonstrated higher levels of disordered eating on the Eating Disorder Inventory, an earlier onset of dieting behavior, and less reported satisfaction with family cohesion than controls (Garner & Olmsted, 1983; Gowers and Shore, 2001; Pike & Rodin, 1991). Researchers argue that these findings are consistent with the theory that maternal attitudes play an important role in the development of psychopathology in the child (Gowers & Shore, 2001). However, a clear and consistent relationship has not yet emerged.

Maternal Eating Disorders

Research has reported that mothers who have an eating disorder themselves have a negative influence on their children's attitudes and behaviors, and tend to feed their children erratically, using food for nonnutritive purposes. Additionally, these mothers express concern about the weight of their daughters as early as two years of age. By the time a child whose mother has an eating disorder is five years old, he or she will already exhibit greater negative affect than control children and face a serious risk for the development of an eating disorder later on (Agras, Hammer, & McNicholas, 1999). Stein, Murray, Cooper,

and Fairburn (1996) found that the children of eating disordered mothers weighed less for their height than the children of control mothers. In a controlled study of 1-year old infants of eating disordered mothers observed during eating and play situations, researchers found that eating disordered mothers were more intrusive during eating and play, and expressed more critical and derogatory remarks during eating than control mothers (Stein, Woolley, Cooper, & Fairburn, 1994). These studies suggest that eating disordered mothers may influence their children's development as early as infancy, and possibly set the stage for the emergence of an eating disorder during adolescence.

Approximately 50% of children whose mothers have an eating disorder have psychiatric disorders (Hodes et al., 1997). Strober et al. (1990) found that adolescent daughters of mothers with anorexia nervosa were five times more likely to develop eating disorders themselves than daughters of mothers in a non-clinical control group.

Maternal Depression

Parental depression is a risk factor for several childhood psychiatric disorders, and specifically for depression. Children of depressed parents face a two to three fold increased risk for developing depression than children of non-depressed parents (Weissman, Pilowsky, Wickramarante, Talati, Wisniewski, et

al., 2006). Children of depressed parents often have disorders that begin prior to puberty, persist into adolescence and adulthood, and are associated with impairments in social and occupational functioning (Weissman, Wickramaratne, Nomura, et al., 2005).

Weissman and colleagues (2006) studied 151 mother-child pairs in an attempt to determine the presence of an association between effective treatment of women diagnosed with depression and a reduction of symptoms and diagnoses in their children. After 3 months of medication treatment, there was a significant relationship between the remission of maternal depression and decreases in the children's diagnoses and symptoms. Overall, children whose mothers' depression remitted displayed an 11% decrease in their rates of diagnoses, while children whose mothers' remained depressed displayed an 8% increase in their rates of diagnoses. The results from this study suggest that the treatment of maternal depression is additionally associated with positive outcomes in their children. These results have implications for the eating disordered population in which the depressed mother of an anorexic patient may be influencing her daughter's psychopathology.

Cognitions and Anorexia Nervosa

Abnormal beliefs about weight and shape are thought to play a causal role in the maintenance of eating disordered behavior. These beliefs are dysfunctional because of their rigidity and extremeness, and they hold immense personal meaning to the individual (Garner & Bemis, 1982). The automatic assumptions and underlying thoughts held by an eating disorder patient include information-processing errors and systematic distortions in the processing and interpretation of events. Often these distortions and processing errors include dichotomous or all or nothing reasoning (Cooper, Todd, & Wells, 1998).

According to Cooper, Todd, and Wells (1998), underlying assumptions about weight, shape, and eating, as well as negative self-beliefs, must be present for the development of an eating disorder. In one study, patients with anorexia nervosa scored higher on a measure of assumptions and beliefs characteristic of eating disorders than both 'normal' dieters, and female controls. These results suggest that the presence of assumptions about eating and negative self-beliefs distinguish patients with anorexia nervosa from 'normal' dieters (Cooper & Turner, 2000).

Personal identity structures and self-schemata are beliefs and rules an individual operates around, and they form the basis for how the individual organizes their lives. They both appear to be important in determining

perceptions, thoughts, affect, and behavior (Vitousek & Hollon, 1990). Guidano and Liotti (1983) have found that individuals with anorexia nervosa have a personality identity characterized by beliefs of general ineffectiveness and failure, and draw attention to early experiences within the family resulting in the formation of these unhealthy beliefs.

Other studies have focused on more general dysfunctional beliefs held by individuals with eating disorders. According to Marshall et al. (1993), anorexic women hold more abnormal beliefs concerning guilt, self-esteem, and self-evaluation than non-eating disordered control women. In addition, Clark et al. (1989) demonstrated that anorexic women hold more general depressive beliefs about the self and the future than control women.

Anorexia Nervosa and Comorbid Depression

In addition to anorectic symptoms, other psychological symptoms are reported in a large percentage of patients with anorexia nervosa. Research has shown that children and adolescents diagnosed with an eating disorder have an increased risk for comorbid psychiatric disorders and disturbances when compared to non-eating disordered peers. Rates of comorbid depression in eating disordered individuals have been reported to range from 15% to 50% depending on the study (Laessle, et al., 1987). A study by Fosson and colleagues (1987)

found that 56% of 15-year olds with anorexia nervosa were also diagnosed with depression. Additionally, McDermott and colleagues (2006) reported that approximately 40% of children and adolescents diagnosed with anorexia nervosa would also meet criteria for depression at first assessment.

Additionally, significant associations between depressive psychopathology and eating disordered psychopathology have been reported in eating disordered patients (Laessle, Kittl, Fichter, Wittchen, & Pirike, 1987). For instance, McDermott and colleagues (2006) found that children and adolescents with anorexia nervosa and comorbid depression reported higher eating disordered symptom scores on the Eating Disorders Examination (EDE) (Cooper & Fairburn, 1987), more gastrointestinal symptoms, and a longer duration of illness at assessment than those without comorbidity. The researchers concluded that depressive symptoms significantly influence eating disordered psychopathology and that comorbidity has some applicability as an indicator of the severity of eating disordered psychopathology.

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

Over the past few decades, hundreds of reports have been published on the course and outcome of anorexia nervosa. The underlying aim of these studies has

been an attempt to provide a more comprehensive and long-term description of anorexia nervosa (Pike, 1998). Studies on the long-term course of eating disorders vary greatly with regard to sample characteristics and size, follow-up interval, assessment procedures, statistical analyses, diagnostic criteria, and conceptualization of the stages of remission, recovery, and relapse. Despite the previously mentioned variance among studies, they are all in agreement that eating disorders are multifaceted disorders that are often associated with a chronic course. As a result, generalizations across studies are extremely difficult because of the previously mentioned methodological issues (Richard, Bauer, Kordy, COST Action B6, 2005). In addition to the methodological issues, significant heterogeneity of illness course has been observed in individuals with anorexia nervosa. Consequently, attempts to identify the specific factors that determine the course and outcome of anorexia nervosa are unclear and the picture that emerges is that of a disorder that refuses to conform to a distinct and predictable course (Pike, 1998).

General Outcome

Despite the aforementioned difficulties in the study of the outcome of anorexia nervosa, follow-up studies have provided valuable information regarding the long-term course of the illness. The most common outcome domains assessed in anorexia nervosa include: mortality, weight, eating behavior, and psychological functioning (Herzog, Keller, Lavori, 1988).

Many studies employ the Morgan and Russell (1975) Criteria or the modified Morgan-Russell Criteria (1991) to assess general outcome of anorexia nervosa. General outcome is based on measures of weight and menstruation, and includes three basic outcome groups: good, intermediate, and poor (Pike, 1998). Despite the widespread use of the Morgan-Russell Criteria, it is not without problems. According to Pike (1998), it fails to take into consideration some behavioral and psychological components of anorexia nervosa, and consequently may result in an inaccurate classification of outcome.

As a result, the outcome studies of anorexia nervosa report a wide range in terms of overall outcome. In a review of the current long-term studies of anorexia nervosa, Pike (1998), reported that the majority of studies found that approximately 50 to 70% of individuals diagnosed with anorexia nervosa achieved a good to intermediate outcome. Conversely, approximately 15 to 25% of anorexia nervosa patients were reported as having chronic symptoms. Herpetz-Dahlmann and colleagues (1995) followed-up 34 adolescents diagnosed with anorexia nervosa 3 and 7 years after discharge from an inpatient facility. Results at 7-year follow-up revealed that 58% of the adolescents had a good outcome, 21% were rated as having intermediate outcomes, and 21% were rated as chronically ill (poor outcome). The general outcome of this study was comparable

to that of other studies of general outcome of anorexia nervosa (Herpetz-Dahlmann et al., 1995).

Predictors of Outcome

Studies within the eating disorder literature have found several variables to be predictive of outcomes in anorexia nervosa. According to Herzog and colleagues (1993), the type of eating disorder appears to be a significant predictor of outcome. More specifically, a poorer outcome has been associated with a diagnosis of anorexia nervosa than a diagnosis of bulimia nervosa. For example, Herzog, Dorer, Keel, Selwyn, Ekelad, Flores, et al. (1999) conducted a prospective, naturalistic, longitudinal study of a group of anorexia nervosa and bulimia nervosa patients. During the 7.5-year study, only 33.7% of the anorexia nervosa patients achieved full recovery as opposed to 73.8% of the bulimia nervosa patients. Results also revealed that the strongest predictor of outcome in this sample was a diagnosis of anorexia nervosa at intake.

In addition, low body weight has been identified as having a significant predictive value for anorexia nervosa (Hebrebrand et al., 1997). For instance, Herzog, Deter, Fiehn, and Petzold (1997) followed 84 anorexia nervosa patients over a 12-year period in an attempt to identify predictors of long-term physical

outcome. Results indicated that low weight (<60% of average body weight) at initial examination was predictive of a lethal course of anorexia nervosa.

Duration of illness has also been found to be a predictor of outcome in studies of anorexia nervosa (Herzog, et al., 1993; Ratnasuriya, Eisler, Szmulker, & Russell, 1991). Ratnasuriya and colleagues (1991) followed 41 anorexia nervosa patients over a 20-year period following hospital admission. A poorer outcome was found to be associated with a longer duration of illness. More specifically, results revealed that although individual patients could still recover after a 15-year period, the rate of recovery drastically decreased after an illness duration of 12-15 years.

Additionally, Ratnasuriya and colleagues (1991) found a later age of anorexia nervosa onset to be predictive of a worse outcome. Patients in the good and intermediate outcome groups had a mean age of illness onset of 14.9 years, while the patients in the poor outcome group had a mean age of illness onset of 21.5 years. Similarly, Steinhausen, Rauss-Mason, and Seidel (1991) reported that an earlier onset of anorexia nervosa is associated with a more favorable prognosis.

Outcome and Family Functioning

Family therapy is an integral part of the treatment of anorexia nervosa in children and adolescents, yet there is insufficient knowledge regarding the nature

of change within the family system. As a result, Wallin and Kronvall (2002) followed 26 families who had an adolescent daughter diagnosed with anorexia nervosa and had been admitted for treatment on an inpatient or outpatient basis. The purpose of the study was to investigate how family functioning changes after 2 years of family therapy. At follow up, the patients were evaluated according to the Global Clinical Rating Scale and further classified as recovered or not recovered. Patients could not meet any DSM-IV diagnoses, including anorexia nervosa, to be included in the recovered group. Improvements in family functioning were found in both groups on all observer-rated dimensions including: cohesion, enmeshment, rigidity, adaptability, and competency. At follow-up, the families in the recovered group had better family competence overall and were less enmeshed. Additionally, the recovered group reported an increase in expressiveness at follow-up. Results from this study support the hypothesis that clinical recovery from anorexia nervosa is associated with the normalization of dysfunctional family functioning.

Other researchers have also investigated changes in the functioning of a family with a child or adolescent diagnosed with anorexia nervosa subsequent treatment. For instance, Robin, Siegel, and Moye (1995) assessed family functioning using both self-report ratings and observer ratings of familial conflict in 22 anorexic female adolescents. At 1-year follow-up, observer ratings revealed a decrease in negative communication and an in increase in positive

communication. Additionally, self-report ratings using the Parent Adolescent Relationship Questionnaire revealed a reduction in conflict related to eating.

Conversely, other researchers have not found improvements in family functioning over the course of treatment. For instance, Gowers and North (1999) investigated the relationship between illness severity in adolescents diagnosed with anorexia nervosa and associations in perception of family functioning.

Results revealed that improvement in anorexic patients were not associated with improvement in family functioning over a one-year period. Additionally, the researchers did not find an association between problems in family functioning and the severity of anorexia.

Outcome and Comorbid Depression

Longitudinal studies of eating disorders report a prevalence of depressive symptoms at follow-up in 20% to 40% of patients (Morgan & Russel, 1975; Hsu, Crisp, & Harding, 1979). Rosenvinge and Mouland (1990) suggested that a diagnosis of a depressive disorder at follow-up is associated with a poor outcome for the eating disorder. The researchers found that of the 41 anorexic patients in the study, none with comorbid depression had a good outcome. Similarly, a study by Herpertz-Dahlmann and Remschmidt (1993) investigated the relationship between depressive symtomatology and eating disordered psychopathology in 34

adolescent patients diagnosed with anorexia nervosa at 3-year follow-up. Results revealed a significant relationship at follow-up between severity of eating disorder and severity of depression. The researchers concluded that adolescents who suffer from depression at follow-up are more likely to continue to meet criteria for anorexia nervosa at follow-up than adolescents without depression. Conversely, Halmi et al. (1991) found no significant difference at 10-year follow up in the lifetime prevalence of affective disorders in the patients that maintained an eating disorder compared to those who had recovered. However, patients with a diagnosable eating disorder had more current depressive symptoms than those without an eating disorder at follow-up. In conclusion, research within the pediatric and adolescent populations suggest that comorbid depression may be a negative treatment indicator.

CHAPTER III

RATIONALE, AIMS, AND HYPOTHESES

Rationale and Aims

Maternal factors have been found to be important to the psychiatric status of children (Weissman, et al., 2006). However, there is a lack of research in the eating disorder literature pertaining to the impact of maternal psychopathology (i.e. depression and eating disordered cognitions) on a child or adolescent with anorexia nervosa. This study investigated the relationship between maternal psychopathology and eating disorders. Specifically, this study examined the relationship between maternal cognitions regarding food weight, and shape and their children's cognitions regarding the same constructs. In addition, the relationship between maternal depressive symptoms and the severity of the child or adolescent's comorbid depression and eating disordered attitudes and cognitions was analyzed. Furthermore, the impact of maternal eating disordered cognitions on the child or adolescent's treatment outcome was also assessed.

As a result of the lack of consistent research concerning the relationship between maternal psychopathology and pediatric anorexia nervosa, further study is needed. It is expected that data from this study will add to the existing literature regarding children and adolescents with anorexia nervosa. More

specifically, this study aimed to identify maternal characteristics that put children and adolescents with anorexia nervosa at risk for poor treatment outcomes.

Questions and Hypotheses

The following questions and hypotheses were examined in this study:

Question One

Is there a positive relationship between mothers and children on measures of eating disordered psychopathology (e.g. cognitions) in a population of children and adolescents with anorexia nervosa?

Hypothesis One:

A positive correlation will exist between the mothers' eating disordered cognitions and the child or adolescent's degree of eating disordered psychopathology (as measured by total scores of mother's and child's MAC-R) in a sample of youth with anorexia nervosa.

Question Two

Is there a relationship between severity of maternal depression and severity of child or adolescent's depression and severity of child or adolescent's eating disordered attitudes and cognitions at baseline?

Hypothesis Two:

A positive correlation will exist between maternal depression (as measured by total score of BDI-II) and severity of patient's a.) depression (as measured by total score of CDRS-R) and b.) eating disordered attitudes and cognitions (as measured by total score of EAT-26 and MAC-R).

Question Three

Do children and adolescents of mothers who have less eating disordered cognitions exhibit greater improvement at discharge from an acute psychiatric treatment facility than the youth whose mothers have more eating disordered cognitions?

Hypothesis Three:

Mothers who score lower on measures of eating disordered cognitions (total score of MAC-R) will have children who demonstrate greater improvement at discharge (as measured by three indicators of outcome: 1) Discharge BMI; 2) Improvement of BMI at discharge of at least 1 standard deviation from baseline; 3) the Clinical

Global Impressions Scale global improvement at discharge) than those children whose mothers score higher on measures of eating disordered cognitions.

CHAPTER IV

METHOD

Subjects

All subjects were obtained 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 subjects enrolled in the study. All subjects were admitted to the inpatient or partial hospitalization programs at Children's Medical Center Dallas for treatment of anorexia nervosa or eating disorder not otherwise specified.

Inclusion Criteria

- Children and adolescents between eight and eighteen years of age at hospital admission.
- 2. All subjects must have been living with at least one parent or legal guardian who had been the child's primary caregiver for at least one year and must have had at least one primary caregiver who was willing to participate in the study with his or her child.
- 3. All subjects must have had a primary DSM-IV diagnosis of anorexia nervosa or eating disorder not otherwise specified (EDNOS) with

below healthy weight (i.e. body mass index [BMI] below 18.5) at admission to the unit. Patients who also met DSM-IV criteria for other Axis I disorders were not excluded from the study as long as the principal reason for admission to the unit was an eating disorder.

Exclusion Criteria

- Patients who had a diagnosis of a psychotic disorder were excluded from this study.
- 2. Patients were excluded from this study if they had below normal intellectual functioning (IQ<80), as determined by history given by caregiver, medical chart review, and direct observation. If there was any concern about intelligence, a clinician could administer the Weschler Intelligence Scale for Children (WISC-IV).

Setting and Procedure

All procedures were conducted at Children's Medical Center Dallas on the psychiatry unit. A research associate recruited potential subjects into the study by direct solicitation at the child or adolescent's admission to inpatient or partial hospitalization at the psychiatry unit of Children's Medical Center. At the child's intake for admission to the unit, a research associate gave the parent or legal

guardian a consent form that included a written explanation concerning the purpose, procedures, possible risks and benefits, and confidentiality associated to the study. The consent form specified that participation in the study was voluntary and that the child or adolescent's treatment would not be affected by their decision to participate. The research associate explained the study to both the parent and the child, and used developmentally appropriate terms with the child. Both the child and caregiver were given the opportunity to ask questions prior to consenting for participation in the study.

After all aspects of the study had been explained and completely understood by the participant and their caregiver, they were both asked to sign the informed consent form (See Appendix A). Participants and their caregivers were also asked to sign a Health Insurance Portability and Accountability Act (HIPAA) authorization form, which specified instances in which the subjects' protected health information (PHI) might be disclosed. Copies of these forms were provided to the primary caregiver and placed in the child's medical chart after signing. Informed consent from participants and their primary caregivers was obtained prior to the collection of any data. In addition, the following demographic and illness variables were obtained from the child or adolescent's medical chart: date of admission, age, date of birth, and ethnicity. Height and weight information was also collected from the medical chart and was used to calculate the patient's body mass index (BMI).

The mother or female caregiver completed the Mizes Anorectic

Cognitions Questionnaire-Revised (MAC-R) and the Beck Depression InventoryII (BDI-II). The patient completed the Mizes Anorectic Cognitions QuestionnaireRevised (MAC-R) and the Eating Attitudes Test (EAT-26). The primary caregiver and patient completed these measures within one week of admission to the unit.

Also within one week of admission to the unit, a trained research associate met with the subject and parent/legal guardian separately to obtain valid psychiatric diagnoses using a semi-structured DSM-IV based interview. The semi-structured interview administered was the Schedule for Affective Disorders and Schizophrenia for School Aged Children, Present and Lifetime (K-SADS-PL) (Klein, 1993). The trained research associate also completed the Childhood Depression Rating Scale—Revised (CDRS-R) (Poznanski et al., 1984) and two expert clinicians' (clinical psychologist and psychiatrist) completed the Clinical Global Impressions-Severity (CGI-S).

All research subjects participated in the standard treatment program offered by the psychiatry medical staff at Children's Medical Center, which is the treatment utilized regardless of the patient's involvement with this research project. All patients were placed on an individualized meal plan in order to restore their physical health and weight. The patient's family members participated in meal education sessions in order to learn skills to effectively support the patient during mealtime at the hospital and upon discharge. In addition to individual and

family therapy twice a week, all patients participated in a daily support group, coping skills group, and nutrition group. They also attended an eating disorder process group twice a week. Inpatients slept at the hospital, while partial hospitalization patients remained at the hospital from 7 am until 7 pm and did not spend the night at the hospital.

Upon the child or adolescent's discharge, height and weight were obtained for a second time from the medical chart. The Clinical Global Impressions-Improvement (CGI-I) was also completed at discharge by the same two expert clinicians'. All acquired data (including self-report measures and clinician-rated measures) was kept in a locked filing cabinet, in a locked room at The University of Texas Southwestern Research Center for Pediatric Psychiatry. All data that was entered into the confidential database was double-checked to ensure accuracy prior to analyses.

Measures

Clinician Rated Measures:

The Schedule for Affective Disorders and Schizophrenia for Schoolaged Children- Present and Lifetime Versions (KSADS-PL; Kaufman et al., 1997)

The KSADS-PL 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 82symptom 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 with the Beck Depression Rating Scale and .89 with the Children's Depression Rating Scale (Ambrosini, 2000). Test-retest reliability has been established as ranging from .63 for attention deficit hyperactivity disorder to .90 for major depression (Ambrosini, 2000). Additionally, interrater reliability has been established as .8 (Ambrosini, 2000).

Self-Report Measures of Eating Disordered Cognitions and Attitudes:

Mizes Anorectic Cognitions Questionnaire-Revised (MAC-R) (Mizes, 1994)

The MAC-R, an updated version of the Mizes Anorectic Cognitions questionnaire (MAC), is a time efficient, self-report instrument that assesses eating disordered cognitions associated with anorexia and bulimia nervosa (Mizes et al., 2000). The MAC-R contains 24 items, and examines cognitive processes related to eating behavior using three subscales: rigid weight regulation and fear of weight gain, weight and approval from others, and self-control and self-esteem. Each item on the MAC-R is rated on a 5-point scale, with 1 being strongly disagree and 5 being strongly agree. Ten of the 24 MAC-R items are reversed scored to reduce response bias (Osman, Chiros, Guiterrez, Kopper, & Barrios, 2001). The MAC-R is written at a sixth grade level and is suitable for persons in middle school through adulthood. The internal consistency for the total score of the MAC-R was high with a Chronbach's alpha of .90 (Osman, et al., 2001). Internal consistency of the MAC-R subscales is also satisfactory, with a Chronbach's alpha of .89 for the self-control and self-esteem scale, .75 for the rigid weight regulation and fear of weight gain, and .72 for the weight and approval from others scale (Osman, et al., 2001). Concurrent validity has been demonstrated as well. The MAC-R was 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), the MAC-R self-control scale (r=.70, p=.00), approval (r=.43, p=.00), and fear of weight gain (r=.40, p=.00) (Mizes et al., 2000).

Eating Attitudes Test (EAT-26) (Garner, Olmsted, Bohr, & Garfinkel, 1982)

The EAT-26 is a standardized measure of symptoms and concerns characteristic of eating disorders (Garner, Olmstead, & Polivy, 1983). It is a 26-item self-report screening measure that is widely used as a result of its sensitivity in identifying eating disorders and partial syndrome eating disorders.

Furthermore, its use has been previously validated in the adolescent population (Wood, Waller, Miller, & Slade, 1992). Individuals report their agreement with statements concerning attitudes of weight and 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 and is made up of three subscales. The dieting subscale assesses avoidance of high calorie foods and concerns with being thinner. 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-26 has been reported to range from .79 to .94 (Allison, 1995). 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).

Measures of Psychopathology Specifically Related to Depression:

The Beck Depression Inventory-II (BDI-II) (Beck, Steer, & Brown, 1996)

The BDI-II is a revision of the amended Beck Depression Inventory (BDI-1A) (Beck & Steer, 1993), a widely utilized self-report measure of depression.

The BDI-II is a 21-item self-report instrument designed to measure the severity of depressive symptomatology in adolescents and adults, and is based on DSM-IV diagnostic criteria for the major depressive disorders. Each item is rated on 4-point scale (0 to 3), with higher scores indicating more severe symptoms.

Summing the ratings for all 21 items yields a total score with a possible range of 0-63 designed to indicate severity of depression (Osman, Kopper, Gutierrez, Bagge, & Barrios, 2004). Clinical interpretation of scores utilizes the following interpretive ranges: 0-13 - minimal depression; 14-19 - mild depression; 20-28 - moderate depression; and 29-63 - severe depression. Validity between the BDI-II and its predecessor has been well established with correlations of .93 and .84 respectively. Additionally, adequate convergent validity has been demonstrated

between the BDI-II and two other measures of depression: the Revised Hamilton Psychiatric Rating Scale for Depression (Hamilton, 1960) and the Beck Hopelessness Scale (Beck & Steer, 1988), with correlations of .68 and .71 respectively. Internal consistency of the BDI-II is high, with reported alpha values greater than or equal to .90 in adult clinical and nonclinical samples. Furthermore, the reliability of the BDI-II has been well established, with Cronbach alpha coefficients ranging from .91-.93 (Beck, Steer, & Brown, 1996).

The Child Depression Rating Scale—Revised (CDRS-R) (Poznanski, Grossman, Buchsbaum, Freeman, & Gibbons 1984)

The CDRS-R is a 17-item clinician-rated instrument that is used to measure the presence and severity of depressive symptomatology in children and adolescents. The CDRS-R, a modified version of the CDRS (Poznanski, Cook, & Carroll, 1979), is a semi-structured interview appropriate for children (ages 6 to 12 years), adolescents, their parents, teachers, case workers, or other reliable informants. The measure takes approximately 30 minutes to administer, and includes seventeen (17) symptom areas. The last 3 areas are evaluations of the child/adolescent's nonverbal characteristics. Each item is rated on a 1 to 5 or 1 to 7 point scale, with a 1 describing absence of the given symptom. For the purposes of this study, all subjects were given a standard score of 5 on question 5. This question pertains to appetite disturbance and is rated from 1 to 5 (1= no problems

or changes in eating patterns; 3= Mild but notable change from usual eating habits; 5= Avoids eating and/or is not hungry most of the time or describes a noteworthy increase in appetite and/or excessive food intake). All subjects were given the maximum score on the appetite disturbance item due to inconsistent scoring of this particular item, and the fact that all the subjects were hospitalized for an eating disorder. Additionally, the intention of the CDRS-R for this study was to look at depression. The CDRS-R yields a total score ranging from 17 to 113, with a score of 40 or greater being considered compatible with a diagnosis of depression. The CDRS-R has been used successfully in the psychopharmacology studies for some time and allows for ready comparison to be made across studies. The CDRS-R has demonstrated good interrater reliability with an intra class correlation of .95; it also correlated highly with global ratings of improvement (Chambers et al., 1985).

Outcome Measures:

The Clinical Global Impressions Scale (CGI) (National Institute of Mental Health, 1970)

The CGI is used as a clinician assessment of overall symptom severity and improvement, each with a seven-point scale, with lower values being more favorable. A psychiatrist and a psychologist complete the CGI. At intake, only

severity can be rated. However, both severity and improvement can be rated in subsequent assessments. This is a standard scale for outcomes treatment research, and a CGI improvement of 1 (very much) or 2 (much) improved is considered to be an acceptable response to acute treatment, as is a clinical severity rating of less than or equal to 3. The CGI was developed during the PRB collaborative schizophrenia studies (National Institute of Mental Health, 1970). The items on the CGI are considered universal, and are therefore appropriate for use in pediatric as well as adult populations. The intra class correlation for CGI improvement as a continuous variable in the above study was 0.93, and if used as a categorical variable, was k = .95 (National Institute of Mental Health, 1970).

Body Mass Index (BMI)

Body Mass index is an objective measure of body weight that is adjusted based on an individual's height. BMI is calculated by dividing an individual's weight in kilograms by their height in meters squared. BMI for children and adolescents is gender and age specific and is used to assess underweight, overweight, and risk for obesity (Centers for Disease Control and Prevention).

CHAPTER V

RESULTS

Interrater Reliability

Prior to all other analyses, interrater reliability was established between raters on the CGI-I. CGI-I scores were provided by two independent raters: the clinical psychologist (rater 1) and psychiatrist (rater 2) of the psychiatry unit at Children's Medical Center. As previously described, Improvement scores on the CGI are based upon a seven-point scale, with lower values indicating more favorable outcomes. CGI-I scores of 1 (very much improved) and 2 (much improved) are considered to be an acceptable response to acute treatment. CGI-I scores were converted to categorical data (i.e., scores of 1 and 2 denoting good treatment response/outcome, and scores from 3 to 7 denoting poor treatment response/outcome).

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). Therefore, 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.

Descriptive Statistics

Demographic and Illness Variables

The sample consisted of 43 patients (41 inpatients and 2 partial hospitalization patients) and their primary caregivers. 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 following 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 1. 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) than discharge (M=17.58, SD=1.27). The average length of hospitalization was 28.75 days (SD=13.92). Furthermore, BMI improved significantly over the course of hospitalization, t(42)=-10.06, p=.000.

Many of the demographic characteristics of this sample are similar to other studies that have examined a sample of eating disordered youth. Approximately eighty-eight percent of the sample was female (n= 38), while the rest were male (n=5). The majority of the sample was Caucasians, with only 2 African-Americans, 4 Latinas, and 1 Asian (Appendix C, Table 2). Additionally, the majority of the sample was made up of restricting anorexics (n=30), with only 5 purging anorexics and 8 individuals with ED NOS (Appendix C, Table 3). The sample was also characterized by a high rate of comorbid diagnoses, which is consistent with the current eating disorder literature. Specifically, 69.77% of the sample met criteria for a depressive disorder and 27.91% met criteria for an anxiety disorder (Appendix C, Table 4).

Sample Descriptive Statistics for Measures Utilized

A summary of descriptive statistics for all available maternal measures is provided in Appendix C, Table 5. The mean total score of maternal MAC-R, a measure of eating disordered cognitions, was 53.56 (SD= 12.05), with scores ranging from 36 to 95. On the BDI-II, a measure of the severity of depressive symptoms, the maternal total scores ranged from 0 to 36, with a mean total score of 13.38 (SD=9.33).

A summary of descriptive statistics for all available patient measures is available in Appendix C, Table 6. Total scores of the patient MAC-R ranged from 43-115, with a mean total score of 74.12 (SD=19.21). On the EAT-26, a measure of eating disordered attitudes, the patient mean total score was 32.62 (SD=18.97), with scores ranging from 0 to 67. Finally, on the CDRS-R, a measure of depressive symptomatology, the patient mean total score was 55.46 (SD=15.30), with a range of scores from 26 to 90.

Measures of Eating Disordered Cognitions and Attitudes:

Due to the absence of adolescent normative data on the MAC-R, the sample's total score was compared to a non-clinical sample of 290 undergraduates by a one sample t-test (Osman, Chiros, Gutierrez, Kopper, & Barrios, 2001). As seen in Appendix C, Table 7, the ED sample reported significantly more anorectic cognitions than the non-clinical sample (p<.000). Please refer to Appendix C, Table 6 for a summary of descriptive statistics for all available patient MAC-R data, and Appendix C, Table 5 for all available maternal MAC-R data.

The ED sample's mean total scores of the EAT-26 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 used to control for Type I error and specified that p<.0025 was needed for

significance at the .01 alpha level. As seen in Appendix C, Table 8, total scores of the EAT-26 were significantly higher in the ED sample than the non-clinical group, indicating that the ED group endorsed unhealthier eating attitudes. Please refer to Appendix C, Table 6 for descriptive statistics for all available patient EAT-26 data.

Statistical Analyses

Hypothesis One

A positive correlation will exist between the mothers' eating disordered cognitions and the child or adolescent's degree of eating disordered psychopathology (as measured by total scores of mother's and child's MAC-R) in a sample of youth with anorexia nervosa.

The bivariate relationship between maternal eating disordered cognitions (total score of mother's MAC-R) and the patient's degree of eating disordered cognitions (total score of patient's MAC-R) was assessed by Pearson Product Moment correlation coefficient. Total scores of maternal MAC-R were not significantly correlated with the total scores of patient's MAC-R, r = -.04, p > .05.

Hypothesis Two

A positive correlation will exist between maternal depression (as measured by total score of BDI-II) and severity of patient's a.) depression (as measured by total score of CDRS-R) and b.) eating disordered attitudes and cognitions (as measured by total score of EAT-26 and MAC-R).

The bivariate relationship between the total scores of maternal BDI-II and the total scores of patient's CDRS-R was assessed by Pearson Product Moment correlation coefficient. The bivariate relationship between total scores of maternal BDI-II and the total scores of patient's MAC-R, as well as the bivariate relationship between the total scores of maternal BDI-II and the total scores of patient's EAT-26, were also assessed by Pearson Product Moment correlation coefficients.

Total scores of maternal BDI-II total were not significantly correlated with total scores of patient's CDRS-R, r=.01, p>.05. Similarly, total scores of maternal BDI-II were not significantly correlated with total scores of patient's EAT-26, r=.04, p>.05. Total scores of maternal BDI-II were significantly correlated with total scores of patient's MAC-R, r=.38, p<.03.

Hypothesis Three

Mothers who score lower on measures of eating disordered cognitions (total score of MAC-R) will have children who demonstrate greater improvement at discharge (as measured by three indicators of outcome: 1) Discharge BMI; 2) Improvement of BMI at discharge of at least 1 standard deviation from baseline; 3) the Clinical Global Impressions Scale global improvement at discharge) than those children whose mothers score higher on measures of eating disordered cognitions.

The bivariate relationship between total scores of maternal MAC-R and discharge BMI was assessed by Pearson Product Moment correlation coefficient. Partial correlations were used to control for baseline BMI. The bivariate relationship between total scores of maternal MAC-R and the three categorical indicators of outcome (Improvement of BMI by at least 1 SD, CGI-I rater 1, and CGI-I rater 2) were assessed by independent *t*-tests.

The total scores of maternal MAC-R were not significantly correlated with discharge BMI, r = .02, p > .05. Similarly, total scores of maternal MAC-R 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 9-11).

CHAPTER VI

DISCUSSION

Overview of the Study

This study was designed to systematically evaluate the relationship between maternal psychopathology and severity of child psychopathology, specifically related to eating disordered cognitions and depressive symptoms, within a sample of children and adolescents hospitalized for anorexia nervosa. Additionally, the present study aimed to identify relationships between maternal cognitions regarding food, weight, and shape and acute treatment outcomes of children and adolescents diagnosed with anorexia nervosa. Research has indicated that mothers may play an important role in the transmission of cultural values regarding weight, shape, and appearance to their children. However, a clear and consistent relationship between the transmissions of these values from mother to child has not emerged. In addition, the majority of eating disorder literature focuses on the characteristics of mother's with anorexia nervosa, rather than the characteristics of the mother's of children who have anorexia nervosa, as this study aimed to do. Furthermore, although maternal depression has been found to be important to the psychiatric status of children (Weissman, et al., 2006), there is a lack of research in the pediatric eating disorder literature pertaining to the impact of maternal depression on a child or adolescent with anorexia nervosa.

Given the limited knowledge of this specific population, the present study aimed to identify aspects of maternal psychopathology that influences the child's psychopathology. Additionally, this study aimed to identify maternal characteristics that put children and adolescents with anorexia nervosa at risk for poor treatment outcomes. Cognitions regarding food, weight, and shape, as well as eating attitudes, were assessed by patient self-report (MAC-R and EAT-26). A clinician-rated instrument was used to measure the presence and severity of depressive symptoms in the patient (CDRS-R). Maternal eating disordered cognitions and depressive symptoms were evaluated by parent self-report (MAC-R and BDI-II). Outcome was defined by weight restoration (BMI) and clinician ratings of improvement by the unit's clinical psychologist and psychiatrist (CGI-I).

Discussion of Results

The first hypothesis posited that a positive relationship would exist between maternal eating disordered cognitions and the child or adolescent's degree of eating disordered psychopathology (i.e. cognitions). Although maternal psychopathology seems to play a role in the child's psychopathology, a clear and

consistent relationship between maternal psychopathology and child's psychopathology in an eating disordered population has not emerged. However, this study did not find a significant relationship between maternal eating disordered cognitions and the degree of eating disordered cognitions in the child diagnosed with anorexia nervosa.

The fact that there was not a direct relationship between the mother and child's cognitions regarding food, weight, and shape suggests that cognitions alone might not be significant enough to influence children's behavior. It is also possible that the child's eating disorder psychopathology may be influenced more by other factors, or a combination of factors. However, it is important to note that maternal eating disordered cognitions were assessed within one week of their child's admission for treatment of an eating disorder as an inpatient or partial hospitalization patient. This may have led to an underreporting of eating disordered cognitions by the mothers, due to a heightened awareness of eating disordered cognitions and the fact that the MAC-R is a fairly face valid measure.

The second hypothesis theorized that a positive relationship would exist between maternal depressive symptoms and severity of child's depression, as well as severity of child's eating disordered attitudes and cognitions. Studies have shown that parental depression is a risk factor for several childhood psychiatric disorders, and specifically for depression (Weissman, et al., 2005). Additionally, research has suggested that the treatment of maternal depression is associated

with a reduction in symptoms and psychiatric diagnoses in children (Weissman, et al., 2006). This research has implications for the eating disorder population, which is lacking in research pertaining to the influence of maternal depression on the child's eating disordered psychopathology.

The present study failed to find a significant relationship between maternal depressive symptoms and the child's severity of depression or eating attitudes. There was, however, a significant relationship found between maternal depressive symptoms and the child's degree of eating disordered cognitions. This suggests that maternal depressive symptomatology influences the severity of the child's eating disorder psychopathology, specifically related to their cognitions. It is possible that mothers' who are depressed are not emotionally available to support their children as well as mothers' who are not depressed. This lack of emotional support may contribute to the severity of the child's eating disorder psychopathology. However, because the present study was cross-sectional, it is not possible to precisely determine the direction of this relationship. Maternal depressive symptoms may be influencing the child's psychopathology, or conversely, the child's psychopathology may be influencing maternal depression.

The present study also hypothesized that the presence of less maternal eating disordered cognitions at intake would predict a better outcome in their children at discharge, as defined by different measures of weight restoration (BMI) and clinical judgment (CGI-I) by two independent raters. Although

maternal psychopathology appears to be important to the outcome of children with psychiatric disorders (Weissman, et al., 2006), maternal eating disordered cognitions have not been specifically studied with regard to treatment outcomes of children and adolescents with anorexia nervosa. Despite the expectation that the degree of maternal eating disordered cognitions at admission would predict the child's outcome over an acute period of treatment, this hypothesis was not supported. In fact, a trend emerged in which the children with good outcomes had mothers with slightly higher average MAC-R scores. The MAC-R is extremely face valid, and as a result, those with higher scores are acknowledging their eating disordered distortions. Consequently, it is possible that these mothers might be more in touch with their own psychopathology, and may be more supportive of their children. Conversely, it is possible that the children whose mothers have more eating disordered cognitions achieve good outcomes because they are hospitalized, and therefore removed from the influence their mothers disorders while they receive extensive treatment for their eating disorder.

Limitations

The present study has a number of limitations that are worth discussing.

For instance, the small sample size limited the power of the current study to detect significant relationships. In addition, the largely homogenous demographic

character of the sample did not allow analyses differentiating gender, ethnicity, and subtype of anorexia, which may have given way to information of interest to researchers and clinicians who work with the eating disorder population.

Although the demographic makeup of this sample is congruent with the demographic makeup of other studies within the eating disorder literature, the ability to generalize these results are limited for ethnic minorities, males, and anorexia nervosa purging type. A larger sample size would increase the power to detect significant relationships, allow for comparisons among the subgroups, and improve the external validity of the sample by having a more representative sample.

Another limitation of this study was the use of self-report measures to assess both mother and child's degree of eating disordered cognitions, as well as the mother's presence and severity of depressive symptoms. Self-report measures are susceptible to underreporting; consequently, levels of eating disordered cognitions and depressive symptoms in both mother and child may have been artificially lowered. Underreporting by the patients may be due to denial of the seriousness of their eating disorder or depressive symptomatology, while underreporting by mothers may be the result of a heightened awareness of eating disordered cognitions due to the hospitalization of their child or denial of their own psychopathology. It is also possible that the mothers' sole focus is on the health and well-being of their hospitalized child, and their own psychopathology

is therefore set aside for the time being. Additionally, the study of the relationship of maternal psychopathology to eating disorders was limited by the measures that were employed. The poor interrater reliability on the CGI-I is also a limitation of the present study. This methodological issue can be resolved in future studies by utilizing consensus training prior to data collection in order to ensure that raters base their assessments on common criteria, thereby increasing interrater reliability.

Clinical Implications and Areas for Future Research

Although the previously mentioned methodological issues limit the present study, it contributes important information to the current body of literature on pediatric anorexia nervosa. It is one of very few studies that have evaluated maternal psychopathology in relation to treatment outcome in a homogeneous group of severely ill patients requiring hospitalization. Additionally, it is one of few studies that have assessed the influence of maternal depression in an eating disordered population. The fact that maternal depressive symptoms may be related to more eating disordered cognitions in the child has implications for future treatment of children and adolescents with anorexia nervosa. Depression is a treatable psychiatric disorder, and treating maternal depression is likely to be associated with an improvement in the child's psychopathology. Therefore, the

treatment of maternal depression may be important in the treatment of children and adolescents with anorexia nervosa. It is also possible that maternal depressive symptoms could be a screening tool for severity of the child's eating disorder.

Future research should investigate the relationship between the treatment of maternal depression and treatment outcomes of children and adolescents diagnosed with anorexia nervosa. Further study of the mother child relationship might also employ a more longitudinal design, in order to investigate the relationship of maternal psychopathology over treatment course, including outpatient treatment following acute hospitalization.

Conclusion

In conclusion, the present study found a significant relationship between maternal depressive symptoms and severity of patient's eating disordered cognitions in a sample of children and adolescents hospitalized for anorexia nervosa. The fact that this study did not find a direct relationship between the mother and child's cognitions regarding food, weight, and shape suggests that cognitions alone might not be significant enough to influence children's behavior. In addition, results from this study suggest that maternal depressive symptoms play a more influential role in the child's eating disorder psychopathology than

maternal eating disordered cognitions. As a result, improved assessment of mother's with eating disordered children may be important, as it offers another possible target of intervention. It is likely that the treatment of maternal depression may also be associated with an improvement in the child's eating disorder psychopathology.

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.	Telephone No.	
	(regular office hours)	(other times)	
Betsy Kennard, Psy.D.	214.648.4403	214.648.4403	
Stephanie Setliff, M.D.	214.456.6471	214.456.6471	
Wells Housson, B.A.	214.648.4447	214.648.4447	

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 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 inpatient treatment program at Children's Medical Center in changing dysfunctional family interaction patterns.

PROCEDURES: This study will assess patients recently admitted to an inpatient 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 stay in the hospital. Most patients remain in the inpatient program anywhere from 4 to 8 weeks. However, some patients remain in the inpatient unit much longer. After discharge from the inpatient unit, two follow-ups will be conducted at 6 months

and 12 months. Depending upon inpatient stay, the study can last from 52 weeks up to 56 weeks. Again this could be longer, if the patient remains hospitalized 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

Information pertaining to your participation in this study that will be kept at UT Southwestern: You and your child have the right to privacy. All information obtained from this research that can be identified with you or your child will remain confidential within the limits of the law. You and your child will need to sign a consent form to participate in this study, which will contain both of your names. You and your child will also be videotaped during this study. The videotapes will be labeled by an identification number only. The videotapes and the consent forms will be maintained in separate locked files and only Dr. Kennard and her research assistant will have access to the videotapes. Data entered into the computer for this study will be coded by with a variable called a "study identifier". No match-up of the data to the subject will be able to be made. The "study identifier" will be included in the database for all subjects. The study identifying variable will be used in data analysis as a comparative variable to distinguish study outcomes. Separate "study identifiers" will be used in order to

compare the patients of the Eating Disorder study and the Major Depressive Disorder study.

Information available to other people: An Institutional Review Board (IRB) is a group of people who are responsible for assuring the community that the rights of participants in research are respected. Members and staff of the IRB at this medical center may review the records of your child's participation in this research. A representative of the Board may contact you or your child for information about both of your experiences with this research. If you or your child wish, either or both of you may refuse to answer any questions the representative of the Board may ask.

Research personnel at the Timberlawn Research Foundation (the sponsor of this study) may review your child's medical and research records kept at UT Southwestern to assure the quality of the information used in the research.

Publication of the results of the research: The results of this research may appear in scientific publications without identifying you or your child in any way.

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 responsible representative's name	-	
(printed) (if applicable)		

Legally responsible representative's	Date	
Signature (if applicable)		
Witness' name (printed)	_	
Witness' signature	Date	
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	Date	
years old)		

APPENDIX B

(HIPPA Notification)

The University of 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 Healthy Information for Research Purposes

- 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
- 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 tak	te part in the Research Project.		
The Researchers may still us and share the health is	nformation that they have already		
collected before you cancelled the authorization. T	o 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	led to you.		
Signature of Research Participant	Date		
For Legal Representatives of Research Participan	nts (if applicable):		
Printed Name of Legal Representative:			
Relationship to Research Participant:			

I certify that I have the legal authority un	der applicable law to make this
Authorization on behalf of the Research I	Participant identified above. The basis for
this legal authority is:	
(e.g. parent, legal guardian, person with le	egal power of attorney, etc.)
Signature of Legal Representative	 Date

APPENDIX C

(Tables)

Table 1

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 2

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 3

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 4

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 5
Sample Descriptive Statistics for Maternal Measures (for all available data)

Variable	n	Mean	SD	Range
Total Score MAC-R	36	53.56	12.05	36-95
Total Score BDI-II	36	13.38	9.33	0-36

Table 6
Sample Descriptive Statistics for Patient Measures (for all available data)

Variable	n	Mean	SD	Range
Total Score MAC-R	41	74.12	19.21	43-115
Total Score EAT-26	39	32.62	18.97	0-67
Total Score CDRS-R	35	55.46	15.30	26-90

Table 7

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

	ED Group	ED Group Normative Sample	
	<u>M</u> (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.

Table 8 Comparison of ED Sample at Intake (n=39) to Non-Clinical Sample (n=475) for *EAT-26*

	ED Group	Normative Sample	
	<u>M</u> (SD)	M (SD)	p
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 9

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

Measure	n=12 Poor Outcome <u>M</u> (SD)	n=24 Good Outcome <u>M</u> (SD)	df	t	p
Total Score	50.33 (10.27)	55.17 (12.74)	34	-1.14	> .05

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

Table 10

Independent t-test of Maternal MAC-R and CGI-I (rater 1) as an Indicator of Poor versus Good Outcome

Measure	n=11 Poor Outcome <u>M</u> (SD)	n=25 Good Outcome <u>M</u> (SD)	df	t	p
Total Score	51.09 (9.21)	54.64 (13.13)	34	.81	> .05

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

Table 11

Independent t-test of Maternal MAC-R and CGI-I (rater 2) as an Indicator of Poor versus Good Outcome

Measure	n=9 Poor Outcome <u>M</u> (SD)	n=27 Good Outcome <u>M</u> (SD)	df	t	p
Total Score	48.00 (11.22)	55.41 (11.93)	34	1.64	> .05

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

References

- Agras, S., Hammer, L, & McNichloas, F. (1999). A prospective study of the influence of eating-disordered mothers on their children. *International Journal of Eating Disorders*, 25, 253-262.
- Allsion, D. B. (1995). *Handbook of Assessment for Eating Behavior and Weight-Related Problems*. London: Sage Publications.
- Ambrosini, P. J. (2000). Historical development and present status of the schedule for affective disorders and schizophrenia for school-age children (K-SADS). *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(1), 49-58.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: APA.
- Austin, E. (1993). The importance of perspective in parent-child interpretations of family communication patterns. *Journalism Quarterly*, 70, 558-568.
- Barnes, H.L., & Olson, D.H. (1982). *Parent-Adolescent Communication Scale*. St. Paul, MN: Family Social Science, University of Minnesota.
- Beck, A.T., Steer, R.A. (1993). *Manual for the Revised Beck Depression Inventory*. San Antonio, TX: Psychological Corporation.
- Beck, A.T., Steer, R.A. (1988). *Manual for the Beck Hopelessness Scale*. San Antonio, TX: Psychological Corporation.
- Beck, A.T., Steer, R.A., Brown, G.K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.

- Beumont, P., Al-Alami, M., & Touyz, A. (1988). Relevance of a standard measurement of undernutrition to the diagnosis of anorexia nervosa: use of Quetelet's body mass index (BMI). *International Journal of Eating Disorders*, 7, 399-405.
- Botta, R. A., & Dumlao, R. (2002). How do conflict and communication patterns between fathers and daughters contribute to or offset eating disorders?

 **Journal of Health Communication, 14(2), 199-219.
- Bruch, H. (1973). Eating disorders: obesity, anorexia nervosa and the person within. New York: Basic Books.
- Calam, R. & Slade, P. (1987) Eating problems and sexual experience: some relationships. *British Review of Bulimia and Anorexia Nervosa*, 2, 37-43.
- Calam, R., Waller, G., Slade, P., & Newton, T. (1990). Eating disorders and perceived relationships with parents. *International Journal of Eating Disorders*, 9 (5), 479-485.
- Chambers, W. J., Puig-Antich, J., Hirsch, M., Paez, P., Ambrosini, P. J., Tabrizi, M. A., et al. (1985). The assessment of affective disorders in children and adolescents by semistructured interview. Test-retest reliability of the schedule for affective disorders and schizophrenia for school-age children, present episode version. *Archives of General Psychiatry*, 42(7), 696-702.
- Clark, D.A., Feldman, J., & Channon, S. (1989). Dysfunctional thinking in anorexia and bulimia nervosa. *Cognitive Therapy Res.*, 13, 377-387.

- Cooper, M. (1997). Cognitive theory in anorexia nervosa and bulimia nervosa: a review. *Behavioral and Cognitive Psychotherapy*, 25, 113-145.
- Cooper, M., & Turner, H. (2000) Brief report: underlying assumptions and core beliefs in anorexia nervosa and dieting. *British Journal of Clinical Psychology*, 39, 215-218.
- Cooper, M.J., Todd, G., & Wells, A. (1998). Content, origins, and consequences of dysfunctional beliefs in anorexia nervosa and bulimia nervosa. *Journal of Cognitive Psychotherapy*, 12(3), 213-230.
- Cooper, Z., & Fairburn, C. (1987). The eating disorder examination: a semistructured interview for the assessment of the specific psychopathology of eating disorders. *International Journal of Eating Disorders*, 6, 1-8.
- Crisp, A.H. (1980). Anorexia Nervosa: Let Me Be. London: Academic press.
- Crisp, A.H., Callendar, J.S., Halek, C., & Hsu, L.G. (1992). Long-term mortality in anorexia nervosa: a 20-year follow-up of the St. George's and Aberdeen cohorts. *British Journal of Psychiatry*, *161*, 104-107.
- Dare, C., Le Grange, D., Eisler, I., & Rutherford, J. (1994). Redefining the psychosomatic family: family process of 26 eating disordered families.

 *International Journal of Eating Disorders, 16, 211-226.
- Feldman, W., Feldman, E., & Goodman, J.T. (1998). Culture vs. biology: children's attitude toward thinness and fatness. *Pediatrics*, *81*, 190-194.
- Fish, M., Belsky, J., & Youngblade, L. (1991). Developmental antecedents and

- measurement of intergenerational boundary violation in a nonclinic sample. *Journal of Family Psychology*, *4*, 278-297.
- Fisher, M., Golden, N. H., Katzman, D. K., Kreipe, R. E., Rees, J., Schebendach, J., et al. (1995). Eating disorders in adolescents: a background paper.

 **Journal of Adolescent Health, 16(6), 420-437.
- Fosson, A., Knibbs, J., Bryant-Waugh, R., & Lask, B. (1987). Early onset anorexia nervosa. *Archives of Disease in Childhood*, 62, 114-118.
- Fullinwider-Bush, N., & Jacobvitz, D.B. (1993). The transition to young adulthood:generational boundary dissolution and female identity development. *Family Process*, *32*, 87-103.
- Garfinkel, P.E., Lin, E., Goering, P., Spegg, C., Goldbloom, D.S., Kennedy, S., Kaplan, A.S., & Woodside, D.B. (1995). Bulimia nervosa in a Canadian community sample: prevalence and comparison subgroups. *American Journal of Psychiatry*, 1052-1058.
- Garner, D. M. (1991). The Eating Disorder Inventory, Second Edition. Odessa, FL: Psychological Assessment Resources.
- Garner, D.M., & Bemis, K.M. (1982). A cognitive-behavioral approach to anorexia nervosa. *Cognitive Therapy and Research*, 6, 123-150.
- Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: psychometric features and clinical correlates. *Psychological Medicine*, 12(4), 871-878.

- Garner, D. M., Olmstead, M.P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia nervosag. *Int J Eat Disord*, 2, 15-34.
- Garner, D.M. & Olmstead, M.P. (1983). *Eating Disorders Inventory*. Lutz, FL: Psychological Assessment Resourses.
- Gowers, S., & North, C. (1999). Difficulties in family functioning and adolescent anorexia nervosa. *British Journal of Psychiatry*, *174*, 63-66.
- Gowers, S., & Shore, A. (2001). Development of weight and shape concerns in the etiology of eating disorders. *The British Journal of Psychiatry*, 179(3), 236-242.
- Guidano, V.F., & Liotti, G. (1983). Cognitive processes and emotional disorders:

 A structural approach to psychotherapy. NY: Guilford Press.
- Guttman, H. A., & Laporte, L. (2002). Family members' retrospective perceptions of intrafamilial relationships. *Contemporary Family Therapy*, 24(3), 505-521.
- Hall, A. & Brown, L.B. (1982). A comparison of the attitudes of young anorexia nervosa patients and non patients with those of their mothers. *British Journal of Eating Disorders*, 56, 39-48.
- Halmi, K., Eckert, E., Marchi, P., Sampugnaro, V., Apple, R., % Cohen, J.(1991). Comorbidity of psychiatric diagnoses in anorexia nervosa.Archives of General Psychiatry, 48, 712-718.

- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology and Neurosurgical Psychiatry*, 23, 56-62.
- Harding, T.P., & Lachenmeyer, J.R. (1986). Family interaction patterns and locus of control as predictors of the presence and severity of anorexia nervosa. *Journal of Clinical Psychology*, 42, 440-448.
 - Haudek, C., Rorty, M., & Henker, B. (1999). The role of ethnicity and parental bonding in the eating and weight concerns of Asian-American and Caucasian college women. *International Journal of Eating Disorders*, 25, 425-433.
- Hebrebrand, J., Himmelman, G.W., & Herzog, W. (1997). Prediction of low body weight at long-term follow-up in acute anorexia nervosa by low body weight referral. *American Journal of Psychiatry*, 154, 566-569.
- Herpetz-Dahlmann, B.M., & Remschmidt, H. (1993). Depression in anorexia nervosa at follow-up. *International Journal of Eating Disorders*, 14(2), 163-169.
- Herpetz-Dahlmann, B.M., Wewetzer, C., Schulz, E., & Remschmidt, H. (1995).

 Course and outcome in adolescent anorexia nervosa. *International Journal of Eating Disorders*, 19(4), 335-345.
- Herzog, W., Deter, H.C., Fiehn, W., & Petzold, E. (1997). Medical findings and

- predictors of long-term physical outcome in anorexia nervosa: a prospective, 12-year follow-up study. *Psychological Medicine*. 27, 269-279.
- Herzog, D. B., Dorer, D. J., Keel, P. K., Selwyn, S. E., Ekeblad, E. R., Flores, A.
 T., et al. (1999). Recovery and relapse in anorexia and bulimia nervosa: a
 7.5-year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38(7), 829-837.
- Herzog, D. B., Keller, M. B., & Lavori, P. W. (1988). Outcome in anorexia nervosa and bulimia nervosa. A review of the literature. *Journal of Nervous and Mental Disease*, 176(3), 131-143.
- Herzog, D. B., Sacks, N. R., Keller, M. B., Lavori, P. W., von Ranson, K. B., & Gray, H. M. (1993). Patterns and predictors of recovery in anorexia nervosa and bulimia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32(4), 835-842.
- Hill, A.J., & Franklin, J.A. (1998). Mothers, daughters and dieting: investigating the transmission of weight control. *BR. Journal of Clinical Psychology*, 37, 3-13.
- Hodes, M., Timimi, S., & Robinson, P. (1997). Children of mothers with eating disorders: a preliminary study. *Eur. Eat. Disord. Rev.* 5, 11-24.
- Hsu, L.G. (1983). The aetiology of anorexia nervosa. *Psychological Medicine*, *13*, 231-238.

- Hsu, L.G., Crisp, A.H., & Harding, B. (1979). Outcome of anorexia nervosa. *The Lancet*, 61-65.
- Humphrey, L.L. (1986). Structural analysis of parent-child relationships in eating disorders. *Journal of Abnormal Psychology*, 95, 395-402.
- Humphrey, L.L. (1987). Comparison of bulimic-anorexic and nondistressed families using structural analysis of social behavior. *Journal of the American Academy of Child and Adolescent Psychiatry*, 26, 248-255.
- Humphrey, L.L. (1988). Relationships within subtypes of anorexic, bulimic, and normal families. *American Academy of Child and Adolescent Psychiatry*, 27, 544-551.
- Humphrey, L.L. (1989). Observed family interactions among subtypes of eating disorders using structural analysis of social behavior. *Journal of Consulting and Clinical Psychology*, *57*, 206-214.
- Jacobvitz, D.B., & Bush, N.F. (1996). Reconstructions of family relationships: parent-child alliances, personal distress, and self-esteem. *Developmental Psychology*, *32*, 732-743.
- Jaeffe, A.C., & Singer, L.T. (1989). Atypical eating disorders in young children.

 International Journal of Eating Disorders, 8, 575-582.
- Kog, E., & Vandereycken, W. (1985). Family characteristics of anorexia nervosa and bulimia: A review of the research literature. *Clinical Psychology**Review, 5, 159-180.

- Kog, E., & Vandereycken, W. (1987). Minuchin's psychosomatic family model revised: a concept-validation study using a multitrait-multimethod approach. *Family Process*, 26, 235-253.
- Kog, E., & Vandereycken, W. (1989). Family interaction in eating disorder patients and normal controls. *International Journal of Eating Disorders*, 8, 11-23.
- Laessle, R.G., Kittl, S., Fichter, M.M., Wittchen, H., & Pirike, K.M. (1987) Major affective disorder in anorexia nervosa and bulimia: a descriptive diagnostic study. *British Journal of Psychiatry*, *151*, 785-789.
- Lask, B., & Bryant-Waugh, R. (Eds). (1993). *Childhood onset anorexia and related eating disorders*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Lattimore, P. J., Wagner, H. L., & Gowers, S. (2000). Conflict avoidance in anorexia nervosa: an observational study of mothers and daughters.

 European Eating Disorders Review, 8, 355-368.
- Leung, N., Thomas, G., & Waller, G. (2000) The relationship between parental bonding and core beliefs in anorexic and bulimic women. *British Journal of Clinical Psychology*, *39*, 205-213.
- Lewinsohn, P.M., Striegel-Moore, R.H., & Seeley, J.P. (2000) Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 1284-1292.

- Marshall, P.D., Palmer, R.L., & Stretch, D. (1983). The description and measurement of abnormal beliefs in anorexia nervosa: a controlled study. *Int. J. Methods Psychiatr. Res.*, 3, 193-200.
- McDermott, B., Forbes, D., Harris, C., McCormack, J., & Gibbon, P. (2006).

 Non-eating disorders psychopathology in children and adolescents with eating disorders: implications for malnutrition and symptom severity. *Journal of Psychosomatic Research*, 60, 257-261.
- Minuchin, S. (1974). *Families and Family Therapy*. Cambridge, MA: Harvard University Press.
- Minuchin, S., Rosman, B.L., & Baker, L. (1978). *Psychosomatic Families: Anorexia Nervosa in Context.* Cambrige, MA: Harvard University Press.
- Mizes, S.J. (1998). Neglected topics in eating disorders: guidelines for clinicians and researchers. *Clinical Psychology Review*, 18 (4), 387-390.
- Mizes, J. S., Christiano, B., Madison, J., Post, G., Seime, R., & Varnado, P. (2000). Development of the mizes anorectic cognitions questionnaire-revised: psychometric properties and factor structure in a large sample of eating disorder patients. *International Journal of Eating Disorders*, 28(4), 415-421.
- Moreno, J., Selby, M., Aved, K., & Besse, C. (2000). Differences in family dynamics among anorexic, bulimic, and normal women. *Journal of Psychotherapy in Independent Practice*, 1(1), 75-87.

- Morgan, H. G., & Russell, G. F. (1975). Value of family background and clinical features as predictors of long-term outcome in anorexia nervosa: four-year follow-up study of 41 patients. *Psychological Medicine*, *5*(4), 355-371.
- National Institute of Mental Health (1970). CGI: Clinical Global Impressions. In:

 Guy W., Bonato RR, eds. Manual for the ECDEU Assessment Battery.2.

 Rev ed. Chevy Chase, Md: National Institute of Mental Health.
- North, C., Gowers, S., & Byram, V. (1995). Family functioning in adolescent anorexia nervosa. *British Journal of Psychiatry*, *167*, 673-678.
- Ogden, J. & Steward, J. (2000). The role of the mother-daughter relationship in explaining weight concern. *International Journal of Eating Disorders*, 28, 78-83.
- Osman, A., Chiros, C.E., Gutierrez, P.M., Kopper, B.A., & Barrios, F.X. (2001).

 Factor structure and psychometric properties of the brief mizes anorectic cognitions questionnaire. *Journal of Clinical Psychology*, *57*(6), 785-799.
- Osman, A., Kopper, B., Gutierrez, P., Bagge, C., & Barrios, F. (2004). Reliability and validity of the beck depression inventory-II with adolescent and psychiatric inpatients. *Psychological Assessment*, 16(2), 120-132.
- Pike, K. M. (1998). Long-term course of anorexia nervosa: response, relapse, remission, and recovery. *Clinical Psychology Review*, 18(4), 447-475.
- Pike, K.M., & Rodin, J. (1991). Mothers, daughters, and disordered eating. *Journal of Abnormal Psychology*, 100, 198-204.

- Poznanski, E. O., Cook, S. C., & Carroll, B. J. (1979). A depression rating scale for children. *Pediatrics*, 64(4), 442-450.
- Poznanski, E. O., Grossman, J. A., Buchsbaum, Y., Banegas, M., Freeman, L., & Gibbons, R. (1984). Preliminary studies of the reliability and validity of the children's depression rating scale. *J Am Acad Child Psychiatry*, 23(2), 191-197.
- Polivy, J., & Herman, C. P. (2002). Causes of eating disorders. *Annual Review of Psychology*, *53*, 187-213.
- Ratnasuriya, R.H., Eisler, I., Szmukler, G.I., & Russell, G.F.M. (1991). Anorexia nervosa: Outcome and prognostic factors after 20 years. *British Journal of Psychiatry*, *158*, 495-502.
- Reeves, P.C., & Johnson, M.E. (1992). Relationship between family-of-origin functioning and self perceived correlates of eating disorders among female college students. *Journal of College Student Development*, *33*, 44-49.
- Richard, M., Bauer, S., Kordy, H., & COST Action B6. (2005). Relapse in anorexia nervosa- a 2.5 year follow-up study. *European Eating Disorders Review*, 13, 180-190.
- Robin, A. L., Gilroy, M., & Dennis, A. B. (1998). Treatment of eating disorders in children and adolescents. *Clinical Psychology Review*, 18(4), 421-446.
- Robin, A.P., Siegel, P., & Moye, A. (1995). Family versus individual therapy for anorexia: Impact on family conflict. *International Journal of Eating*

- Disorders, 17, 313-322.
- Rosenvinge, J.H., & Mouland, S.O. (1990). Outcome and prognosis of anorexia nervosa- a retrospective study of 41 subjects. *The British Journal of Psychiatry*, 156, 92-98.
- Rowa, K., Kerig, P., & Geller, J. (2001). The family and anorexia nervosa: examining parent-child boundary problems. *European Eating Disorders Review*, 9, 97-114.
- Santonastaso, P., Pantaro, M., Panarotto, L., & Silverstri, A. (1991). A followup study on anorexia nervosa: clinical features and diagnostic outcome. *European Psychiatry*, 6, 177-185.
- Selvini-Palazzoli, M. (1974). Self Starvation. London: Chaucer
- Shugar, G., & Krueger, S. (1995). Aggressive family communication, weight gain, and improved eating attitudes during systematic family therapy for anorexia nervosa. *International Journal of Eating Disorders*, 17, 23-31.
- Slade, P.D. (1982). Towards a functional analysis of anorexia nervosa and bulimia nervosa. *British Journal of Clinical Psychology*, 21, 167-179.
- Smith, L.M., Mullis, R.L., & Hill, W.E. (1995). Identity strivings within the mother daughter relationship. *Psychological Reports*, 76, 495-503.
- Smolak, L., Levine, M.P., and Schermer, F. (1999). Parental input and eright concerns among elementary school children. *International Journal of Eating Disorders*, 25, 263-271.

- Smolak, L., & Levine, M.P. (1993). Seperation-individuation difficulties and the distinction between bulimia nervosa and anorexia nervosa in college women.
- Steiger, H., Stotland, S., Ghadirian, A.M., & Whitehead, V. (1994). Controlled study of eating concerns and psychopathological traits in relatives of eating disorders probands: do familial traits exist? *International Journal of Eating Disorders*, 18, 107-118.
- Steinberg, A.B., & Phares, V. (2001). Family Functioning, Body Image, and
 Eating Disturbances. In J. K. Thompson, Smolak, L. (Ed.), *Body Image, Eating Disorders, and Obesity in Youth*: American Psychological
 Association.
- Steinhausen, H.-CH., Rauss-Mason, C., & Seidel, R. (1991). Follow-up studies of anorexia nervosa: a review of four decades of outcome research.

 Psychological Medicine, 21, 447-454.
- Stern, S.L., Dixon, K.N., Jones, D., Lake, M., Nemzer, E., & Sansone, R. (1989).

 Family environment in anorexia nervosa and bulimia. *International Journal of Eating Disorders*, 8, 25-31.
- Stice, E., Akutagawa, D., Gaggar, A., & Agras, W. S. (2000). Negative affect moderates the relation between dieting and binge eating. *International Journal of Eating Disorders*, 27(2), 218-229.
- Strober, M., Lampert, C., Morrell, W., Burroughs, J., & Jacobs, C. (1990). A

- controlled family study of anorexia nervosa: evidence of familial aggression and lack of shared transmission with affective disorders. *International Journal of Eating Disorders*, 9, 239-253.
- Strong, K.G., & Huon, G.F. (1998). An evaluation of a structural model for studies of the initiation of dieting among adolescent girls. *J. Psychosom. Res.*, *44*, 315-326.
- van Furth, E. F., van Strien, D. C., Martina, L. M., van Son, M. J., Hendrickx, J. J., & van England, H. (1996). Expressed emotion and the prediction of outcome in adolescent eating disorders. *International Journal of Eating Disorders*, 20(1), 19-31.
- Vitousek, K., B., & Hollon, S.D. (1990). The investigation of schematic content and processing in the eating disorders. *Cognitive therapy and Research*, *14*, 191-214.
- Waller, G., Slade, P., & Calam, R. (1989). Eating disorders and family interaction. *British Journal of Clinical Psychology*, 28, 285-286.
- Waller, G., Slade, P., & Calam, R. (1990). Family adaptability and cohesion: relation to eating attitudes and disorders. *International Journal of Eating Disorders*, 9, 225-228.
- Waller, G., Slade, P., & Calam, R. (1990). Who knows best? Family interaction and eating disorders. *British Journal of Psychiatry*, *156*, 546-550.

- Wallin, U., & Kronvall, P. (2002). Anorexia nervosa in teenagers: change in family function after family therapy at 2-year follow-up. *Nordic Journal of Psychiatry*, 56(5), 363-369.
- Ward, A., Ramsay, R., & Treasure, J.L. (2000). Attachment research in eating disorders. *Br. J. Med. Psychol.*, 73, 35-51.
- Weissman, M.M., Pilowsky, D.J., Wickramarante, P.J., Talati, A., Wisniewski, S.R., et al. (2006). Remissions in maternal depression and child psychopathology: a star*d-child report. *Journal of the American Medical Association*, 295, 1389-1398.
- Weissman M.M., Wickramaratne P., Nomura Y., et al. (2005). Families at high and low risk for depression: a 3-generation study. *Archives of General Psychiatry*, 62, 29-36.
- Wood, A., Waller, G., Miller, J., & Slade, P. (1992). The development of eating attitude test scores in adolescence. *International Journal of Eating Disorders*, 11(3), 279-282.
- Young, J.E. (1994). Cognitive therapy for personality disorders: a schemafocused approach. Sarasota, FL: Professional Resource Press.

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