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# Familial Predictors of Long-Term Outcome Following Inpatient Treatment for Eating Disorders

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FAMILIAL PREDICTORS OF LONG-TERM OUTCOME  
FOLLOWING INPATIENT TREATMENT FOR EATING DISORDERS

by

Anna Mae Jorgensen

A dissertation submitted to the faculty of

Brigham Young University

in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

Department of Counseling Psychology

Brigham Young University

June 2009

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BRIGHAM YOUNG UNIVERSITY

GRADUATE COMMITTEE APPROVAL

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This dissertation has been read by each member of the following graduate committee and by majority vote has been found to be satisfactory.

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As chair of the candidate's graduate committee, I have read the dissertation of Anna Mae Jorgensen in its final form and have found that (1) its format, citations, and bibliographical style are consistent and acceptable and fulfill university and department style requirements; (2) its illustrative materials including figures, tables, and charts are in place; and (3) the final manuscript is satisfactory to the graduate committee and is ready for submission to the university library.

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## ABSTRACT

### FAMILIAL PREDICTORS OF LONG-TERM OUTCOME FOLLOWING INPATIENT TREATMENT FOR EATING DISORDERS

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Department of Counseling Psychology

Doctor of Philosophy

The present investigation examined characteristic, symptomatic, and familial predictors of long-term symptom severity of eating disorders. The purpose of the study was to determine if, after accounting for a number of known predictors of outcome, familial variables explained a significant amount of additional variance in disordered eating and general well-being scores measured at post-treatment follow-up. The sample included 398 women, ages 13 to 56, who had completed eating disorder treatment at an inpatient facility. Hierarchical multiple regression analysis demonstrated that familial predictors at admission to treatment did significantly predict long-term outcomes, while changes from admission to treatment in symptoms and perceptions of parents did not predict recovery. Patients' relationships with their fathers significantly contributed to the regression model. Recommendations for future investigations are discussed.

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## Introduction

Eating disorders are one of the most common psychological disorders faced by women today and present a significant challenge to clinicians in assessment and treatment. Estimates of occurrence vary widely since many cases go unreported; however, it is predicted that currently as many as ten million females and one million males are struggling with an eating disorder such as Anorexia Nervosa (anorexia) or Bulimia Nervosa (bulimia) at any given time (National Eating Disorder Association, 2006).

Not only are eating disorders common, they are also costly. In the United States alone, expenses for the medical treatment of eating disorders surpass five billion dollars per year (National Eating Disorder Association, 2006). For those who receive intensive treatment in residential facilities, costs average nearly one thousand dollars per day while amounts for females treated at in-patient hospitals exceed \$2,000 daily. Time is another price to be paid, with patients in residential centers typically staying an average of 83 days and those hospitalized remaining in-patient for 20.74 days. Hence, for an average length of stay at a residential center, the approximate cost is \$79,348 and the cost of an average length of stay at an in-patient facility is approximately \$47,400 (Crow & Nyman, 2004; Frisch, Herzog, & Franko, 2006; Striegel-Moore, Leslie, Petrelli, Garvin, & Rosenheck, 2000).

Taking into consideration the massive amounts of time and money spent on treatment, creating efficient treatment options appears essential. Characterized by both chronicity and relapse, the health consequences of these disorders are severe, making appropriate assessment and treatment even more critical. Failure to receive help can result in starvation leading to kidney and cardiac disturbance, electrolyte imbalances, stunted growth, osteoporosis, loss of reproductive functions, heart attacks, and even death (Brambilla & Monteleone, 2003; Keel,

2005; National Eating Disorders, 2006; Pomeroy, 2001). The serious consequences of these disorders makes it imperative that practitioners recognize their core pathological features, understand potential explanatory factors, and maintain an awareness of treatment options.

So, what separates those who improve from those who do not? Clinicians, patients, and loved ones alike seek this answer hoping to discern predictors of recovery and chances for positive outcomes. Although long-term studies are sparse, researchers have begun to distinguish indicators of success. Several studies denote age of onset, duration of illness, severity of symptoms, and comorbidity as predictive of prognosis (Keel & Mitchell, 1997; Richards, et al., 2000; Steinhausen, 2002). Unfortunately, however, these common factors are largely determined prior to treatment and are out of the control of those people who have a direct impact on recovery. This presents a dilemma, leaving patients and their families, as well as clinicians, who impart substantial temporal and financial resources on treatment, devoid of any sense of personal power regarding hope for healing. Without an understanding of how recovery occurs, opportunities to focus on those changeable variables which influence outcome are unavailable and treatment remains inefficient. This aspiration of streamlining treatment prompts the search for critical predictors of outcome.

One approach to improving prospects for recovery is to refine treatment to specifically target the primary etiological factors contributing to the disorder (Keel, 2005). In attempting to pinpoint exactly what causes eating disorders, however, professionals face a significant challenge. Due to their complexity and the inability to conduct true experiments when studying eating pathology, explanatory theories are numerous and vary widely (Polivy & Herman, 2002). Nevertheless, since the initial recognition of eating pathology, one principal influence on the

development of the disorders has been highlighted: the family environment (Gull, 1874; Lasègue, 1873).

As has also occurred within other mental health fields, the family has continued to be the focus of blame since early detection of eating disorders. In fact, the “psychosomatic families” of anorexics were some of the first cases from which family therapy originated (Minuchin, Rosman, & Baker, 1978; Selvini-Palazzoli, 1974). Research abounds connecting family dysfunction with the acquisition of eating disorders (Crowther, Kichler, Sherwood, & Kuhnert, 2002; Keel, 2005; McGrane & Carr, 2002; Vidovic, Juresa, Begova, Mahnik, & Tocilj, 2005; Wisotsky, et al., 2006). In early models, families of anorexics were classified as consisting of overprotective, insecure mothers and emotionally-distant, passive fathers (Kog & Vandereycken, 1989). Indeed, 19<sup>th</sup> century theorists believed in order for the patient to recover she had to be separated from her family, especially her parents (Chiple, 1860; Gull, 1874).

In some ways, this idea still holds true today, with many girls leaving their homes and being separated from their families in order to receive residential or in-patient treatment (Frisch, et al., 2006). On the other hand, parents are presently more involved in their daughter’s restoration to health than they ever have been in years past. Nowadays, numerous treatment centers involve parents and siblings in family therapy, either through conference calls when families are separated by distance, or through “family weeks” in which family members are invited to visit the patient for a week at a time to participate in rehabilitation collectively (Frisch, et al., 2006). Given the emphasis that has been placed on the family in both etiology and treatment it seems important to explore whether families deserve the responsibility they have been consigned. Additionally, one may wonder whether family members’ contributions of both time and money on treatment are worth it



When questioned themselves, more than 30% of anorexic patients described their family dysfunction as the chief contributor to the development of their disorder (Tozzi, Sullivan, Fear, McKenzie, & Bulik, 2003). While research has focused on determining how family dynamics influence the development of disordered eating, few studies have attempted to demonstrate how family dynamics impact recovery (Herzog, Kronmuller, Hartmann, Bergmann, & Kroger, 2000). Novel research suggests that women who improve during treatment have families that improve in functioning as well. One study illustrated that patients who recovered had families that were less enmeshed at follow-up and demonstrated a healthier pattern of interaction (Wallin & Kronvall, 2002).

Yet the question remains whether this improvement in interaction was a source or a product of the change in symptomology. Early studies have demonstrated that poor outcomes for eating disorders are associated with lower parental care (Bulik, Sullivan, Fear, & Pickering, 2000), more control and overprotection in childrearing (Castro, Toro, & Cruz, 2000), depression in mothers and substance abuse in fathers (Arikian, et al., 2008), family disruption (Strober, Bowen, & Preble, 1985), and paternal rejection (Castro, et al., 2000), while a return to health has been associated with quality family relationships (Wewetzer, Deimel, Herpertz-Dahlmann, & Mattejat, 1996) and maternal warmth (Krener, Abramowitz, & Walker, 1986). These conclusions demonstrate the importance of family interactions in recovery from eating disorders and highlight the need for further research in this field.

The research at hand intends to expand upon previous investigations on predictors of diagnosis and response to treatment by exploring how familial relationships impact long-term outcomes. Smith (2006) found that perception of one's relationship with her parents predicts symptom severity at intake for inpatient treatment. Patients' perceptions of the parenting they

received from their mother, as measured by the Mother Acceptance-Rejection Questionnaire, predicted their eating behaviors as measured on the Eating Attitudes Test, at admission to treatment. Additionally, one's relationship with her father, as measured on the Attitude Toward Father Scale, predicted individuals' concerns with body shape and size. An additional study on the same sample determined that not only do familial factors predict symptom severity at admission to treatment, but they additionally predict eating pathology at the termination of treatment. Using the same measures of attitude towards parents and parental acceptance-rejection, Tobler (2008) found that relationships with parents had a significant relationship with symptoms at both intake and discharge. Furthermore, Tobler discovered that when other possible predictors were taken into consideration, remembrance of mother's acceptance-rejection had the most statistically significant impact on change in symptom severity from intake to termination. The current study attempts to extend this research to post-treatment outcomes in an attempt to distinguish how perceptions of familial relationships influence long-term recovery.

#### *Rationale for Current Investigation*

Since early conceptualizations of eating disorders, the family has been a primary focus. However, there is a dearth of research regarding implications for familial predictors of recovery and the family has been all but ignored in the literature on post-treatment outcomes. The current study is designed to expand upon previous research with the same population which has found significant familial predictors of disordered eating at admission and termination. An examination of familial influences on long-term outcomes will hopefully provide a more in depth understanding of how successful treatment functions, what distinguishes those who recover from those who do not, as well as offer families of individuals with eating disorders some hope regarding their ability to impact outcomes.

### *Statement of Problem*

A considerable gap exists between theories of eating disorder etiology and findings on recovery, specifically in regards to familial influences. Additionally, long-term outcomes studies have been sparse, and fraught with limitations including small sample sizes, unclear diagnostic criteria, and poorly defined outcome measures.

### *Statement of Purpose*

The purpose of the current investigation is to address these previous limitations and issues by examining several potential predictors of long-term treatment outcomes. Perceptions of patients' familial relationships will be investigated for ability to predict long-term outcomes above and beyond previously established characteristic and symptomatic predictors.

### *Research Questions*

The study is designed to answer the following research questions:

1.) Which factors, including characteristic variables (age at admission, age of onset, history of sexual abuse, eating disorder diagnosis, length of follow-up), symptomatic variables (Outcome Questionnaire-45.2 and Global Assessment of Functioning), and familial variables (Mother Acceptance-Rejection Questionnaire, Father Acceptance-Rejection Questionnaire, Attitude Towards Mother, Attitude Towards Father) are significant predictors of post-treatment eating disorder symptoms (Eating Attitudes Test) and general wellbeing (Phone Survey)?

2.) Are changes in symptom severity (Outcome Questionnaire-45.2 and Global Assessment of Functioning) and perceptions of parental relationships (Attitude Towards Mother, Attitude Towards Father) from admission to termination of treatment significant predictors of post-treatment eating disorder symptoms (Eating Attitudes Test) and general wellbeing (Phone Survey)?

*Importance of the Study*

The hope of this research is to explore the connection between theories of etiology and evidence of long-term outcomes in order to provide new directions for treatment. Current literature connecting family dynamics to eating disorder chronicity and recovery has been sparse, although parents have been the focus of blame for decades. Though previous predictors of post-treatment outcomes have been identified, those recognized are typically unchangeable characteristics (i.e. age) that clinicians, patients, families, and friends may not find promising. In order to provide encouragement towards recovery, establishing predictors that may be changeable is deemed appropriate. Hence, a natural first step in this direction is the field of familial relationships.

## Review of Literature

### *Diagnostic Features and Core Characteristics*

Historical evidence of eating disorders dates back to the first millennium when Roman emperors self-induced vomiting to allow for continued consumption (Crichton, 1996). The first known eating disorder was recorded in the late fourth century in which a 20-year old woman died from self-starvation (Bemporad, 1996). Additional cases were noted among religious ascetics from the 12<sup>th</sup> to 17<sup>th</sup> century (Bell, 1985). Since gaining prominence in the second half of the 20<sup>th</sup> century, there have been specifically two primary eating disorders identified for which diagnostic criteria has been outlined in broadly accepted diagnostic manuals including the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* (American Psychiatric Association, 2000) and the *International Classification of Diseases (ICD)* (World Health Organization, 1998). The two major eating disorders recognized in research and clinical fields are Anorexia Nervosa (anorexia) and Bulimia Nervosa (bulimia) (American Psychiatric Association, 2000; Keel, 2005).

#### *Anorexia Nervosa*

The term “anorexia” is derived from the Greek and refers to an absence of appetite, portraying the refusal to eat typified by Anorexia Nervosa. According to the DSM-IV-TR (American Psychiatric Association, 2000) the diagnostic criteria for Anorexia Nervosa are met when an individual maintains a body weight below a minimally normal level, typically less than 85% of the expected weight range for their age and height. This refusal to maintain a normal body weight is also associated with an intense fear of gaining weight or becoming fat and a significant disturbance in the perception of the size or shape of the body. Additionally,

postmenarcheal females exhibit amenorrhea (the absence of at least three consecutive menstrual cycles).

Weight loss or failure to make expected weight gains is most commonly achieved by limiting food intake. Individuals frequently end up with an extremely restricted diet that consists solely of low calorie foods. Even after weight loss is achieved, fear of becoming fat is not alleviated, and concerns about weight gain are even likely to increase. Individuals with anorexia are characteristically unable to accurately judge their body size and shape, typically assessing themselves as globally obese despite thinness. As a result of this distorted observation, individuals with anorexia often employ a variety of methods to estimate body size, including excessive weighing, measuring of body parts, and mirror checking. Self-esteem is highly dependent on the success of achieving thinness and weight gain is viewed as a failure of self-control. Although the physiological consequences of their starvation are extremely devastating, anorexia remains an ego-syntonic disorder and struggling individuals deny they are harming themselves, lacking insight into the seriousness of their problem.

Diagnostic criteria include subtypes of the disorder which specify the method used to achieve weight loss. Although restricting is the primary method of attaining thinness, individuals with anorexia also may engage in bingeing and purging. The majority of those who binge-eat purge their food through self-induced vomiting or abuse of laxatives. In many cases of anorexia, individuals do not binge eat but are inclined to purge any food consumed, even diminutive amounts (American Psychiatric Association, 2000).

*Bulimia Nervosa*

The term “bulimia” originates from the Latin and Greek words for ravenous hunger which describes the insatiable consumption Bulimia Nervosa retains. The DSM-IV-TR (American Psychiatric Association, 2000) criteria for bulimia nervosa is composed of recurrent episodes of both binge eating and inappropriate compensatory behaviors (purging) that occur on average at least twice a week for three months. A binge is identified by rapid consumption in a discrete period of time (less than two hours) of amounts of food greater than what most individuals would eat in a similar situation. Such consumption typically continues until the individual is uncomfortably or even painfully full. Binges are characteristically accompanied by feeling a loss of control and can be triggered by mood, stress, or hunger. Although food consumed may vary, binges regularly include sweet or salty, high-calorie foods such as ice cream, cookies, cake, or chips. Binges are typically followed by compensatory behaviors. Diagnostic criteria for bulimia are divided into subtypes based on type of compensatory behavior, identifying those that compensate after a binge through purging (including engaging in self-induced vomiting, as well as misuse of laxatives, diuretics, or enemas) and those that compensate using other methods such as fasting or excessive exercise (American Psychiatric Association, 2000). Such compensatory mechanisms are employed as a method to prevent weight gain.

Similar to individuals with anorexia, those with bulimia typically experience a distorted sense of body shape and size, and base evaluations of self-worth on their physique. Those with an eating disorder characteristically place an excessive emphasis on body shape and weight and normally exhibit high levels of dissatisfaction with their appearance (American Psychiatric Association, 2000).

Debate rages concerning some aspects of symptomology, including the presence of weight phobia amid diagnostic criterion as well as the distinctiveness of the two disorders. Currently, inclusion of body image concern has been an essential indicator of diagnosis. However, global accounts of self-starvation and binge-purge cycles have not encompassed body dissatisfaction as a motivating factor for food refusal (Keel & Klump, 2003; Lee, Ho, & Hsu, 1993). This difference calls into question the significance of this diagnostic requisite. Additionally, the classification of eating disorders into separate categories has also been challenged. Some researchers argue eating disorders cannot be separated into distinct types, but rather exist on a continuum from healthy attitudes and behaviors to full-fledged syndromes (Keel, 2005).

### *Epidemiology*

Currently it is estimated that 3–10% of at-risk females (those in the age range of 15–29 years) have an eating disorder, with bulimia outnumbering anorexia two to one (Polivy & Herman, 2002). Prevalence of eating disorders has been speculated to have increased in recent decades. Anorexia has increased from a reported incidence around .24 in the first half of the twentieth century to nearly 4.2 in the 1990's (Keel, 2005). Since being first identified as a distinct disorder in 1979 incidence of bulimia rates have risen from .7–3.0 in 1980 to 12.2 in 1991 (Keel, 2005). Concerns regarding consistent identification of the disorders calls into question some reported rates (Keel, 2005), but researchers have surmised that frequency does in fact appear to have significantly climbed over recent decades (American Psychiatric Association, 2000; Hoek, van Hoeken, & Katzman, 2003; Keel & Klump, 2003).

Although today eating disorders are documented across numerous cultures and among several countries, the highest rates of occurrence exist in industrialized, western societies.



Additionally, while eating disorders are recognized among both men and women of a variety of ages, historically these disorders have been predominately identified in females between the ages of 15 to 29, and for the purpose of this text diagnosed individuals will be referred to in the feminine vernacular.

### *Etiology*

Aside from the commonly recognized risk factors of gender, age, ethnicity, and social system, there are numerous additional features associated with the development of disordered eating. However, the complexity of eating pathology and the inability to conduct true experiments in the field presents professionals with a significant challenge in attempting to pinpoint exactly what causes eating disorders, resulting in etiological theories which vary widely and sometimes offer conflicting guidance (Kinoy, 2001; Polivy & Herman, 2002). Currently, the most widely accepted approach to eating disorders is the “biopsychosocial” model (Polivy & Herman, 2002). This model attempts to cover all aspects that may influence development of disordered eating, identifying risk factors ranging from internal organic causes to external cultural expectations, giving credence to both nature and nurture as potential origins. This theory of causality recognizes the numerous factors that contribute to the onset of bulimia and anorexia. Influences uncontrolled by the victim of an eating disorder such as the occurrence of traumatic events, evidence of genetic transmission, and neuroendocrine dysfunction are incorporated, as well as personality traits such as perfectionism and impulsivity that may predispose someone to develop eating symptomology. Additionally, the model encompasses individual risk factors to explain the prevalence of anorexic and bulimic behaviors, such as low self-esteem, body dissatisfaction, and poor affect regulation. Sociocultural contributors such as internalization of the thin ideal propagated by the media are also highlighted. Researchers tend to agree that a

multidimensional model of this breadth is needed to explain such complicated, multifaceted disorders.

Although each of the factors incorporated in the biopsychosocial model are essential, one topic that has received particular attention in theories about eating disorder etiology is family functioning. When eating disorders were first gaining recognition, Chipley (1860) theorized that self-starvation was an attempt to gain attention from family members. Salvador Minuchin (1978) was one of the first therapists to develop an explanation of the disorder based on familial influences. He theorized that families were composed of smaller groups called subsystems and that eating pathology was a manifestation of undifferentiated boundaries between these groups. Because family subsystems were either enmeshed or disengaged, conflicts became somaticized and expressed through one individual's diagnosis (Minuchin, et al., 1978). Family dynamics continue to be a focus of study under the biopsychosocial model and are examined for the potential to perpetuate as well as prevent onset of eating disorders.

#### *The Family and Eating Disorder Pathogenesis*

Why have families been implicated as a major cause of eating disorders? One possible explanation may be that eating disorders often develop during adolescence, a time in a child's life in which the family remains the primary social context (Keel, 2005). Eating disorders tend to occur during stages of development in which the child is beginning to individuate and separate herself from the family unit (Latzer, Hochdorf, Bachar, & Canetti, 2002). Hence, early clinicians attributed etiology to those families from which the patients came (Keel, 2005). Another rationale for the focus on the family may be the assumption of responsibility of feeding ascribed to the parents (Brusch, 1978; Keel, 2005). Whatever the reason, families, especially parents, have been blamed for the onset of their child's eating disorder for decades. Today, there are

perhaps as many theories of family influence as there are authors, and the number continues to grow, making it a challenge to sift through extraneous features (Kog & Vandereycken, 1989a). There are, however, some defined patterns of family interaction that appear consistent among theories and across research.

### *Stereotypical Family Types*

*The household.* Families plagued by eating disorders notoriously belong to a higher social class than the general population (Kog & Vandereycken, 1989a). Additionally, families with eating disorders are also more likely to have other family members with both physiological and psychological illnesses (Kog & Vandereycken, 1989a). Depression and alcohol abuse are among the most common psychiatric disorders in these families (Arikian, et al., 2008; Kog & Vandereycken, 1989a). Marital discord is also typical within eating disorder families, with parents frequently demonstrating contradictory child-rearing practices (Boskind-Lodahl, 1976). The family unit as a whole also appears to be more focused on appearance and food when compared to families without eating disorders (Hill & Franklin, 1998; Keel, 2005). Resembling the patient's own denial, families of eating disorders are often characterized by failure to recognize the severity of the patient's symptoms. Parents often minimize their daughters' state and appear oblivious to indicators of her distress and ill health (Andersen, 1985; Vandereycken & Meermann, 1984).

*The mother.* As one's mother is naturally the first source of food, maternal influences on eating disorders have been emphasized in the literature (Kog & Vandereycken, 1989a). In early family theories, the patient was even identified as a manifestation of the mother's pathology (Edhouse, 1985). Mothers of eating disorder patients have been portrayed in a variety of ways, including being described as perfectionistic, rigid, obsessive-compulsive, powerless, dependent,

and affection-seeking (Kog & Vandereycken, 1989a). Some literature suggests upon first feeding, mothers of those with eating disorders demonstrate a lack of awareness of the child's needs and respond inappropriately to cues of hunger (such as holding a hungry baby rather than feeding her). Theorists insinuate that such ineffective responses may have impaired the child's ability to recognize her own internal states later, ultimately leading to an eating disorder (Brusch, 1978).

*The father.* Although the father has been indicted less often than the mother, assumptions regarding his influence on the eating disorder abound as well. The father has been predominantly characterized as emotionally distant, intelligent but unreachable, angry, violent, ambivalent, and demanding (Kog & Vandereycken, 1989a). He has additionally been described as both weak and dominant, and in some cases the deterioration of the father-daughter relationship during puberty has been noted (Sights & Richards, 1984).

#### *Theories of Parents as Perpetrators*

Considering the descriptions outlined above, it seems natural to conclude that the parents of those with anorexia and bulimia were the primary cause of the disorder itself. Although current research has attempted to absolve parents of guilt (Whitney & Eilser, 2005), the family has remained the primary source of blame and numerous theories have attempted to explain how family dynamics produce eating disorders.

*Psychoanalytic paradigms.* Some of the first theories to explain the development of anorexia were psychoanalytic, emphasizing an oedipal conflict as the root of the disorder (Keel, 2005). From the psychoanalytic perspective, anorexia is an attempt to avoid sexual maturity. In families where mothers failed to model appropriate femininity and fathers were overly flirtatious with their daughters, girls feared becoming women like their mothers (Frazier, Faubion, Giffin,

& Johnson, 1955). This fear was then symbolized by oral impregnation leading the daughter to endeavor to offset puberty through self-starvation (Szyrynski, 1973).

*Psychodynamic views.* A psychodynamic position theorized that parents' mismatch of providing for their daughter's needs led the daughter to adjust by accommodating her needs to the environment, accordingly becoming the ideal child. This perspective appears consistent with parental reports of their daughters as cooperative and well behaved. As daughters neared adolescence and were unprepared for autonomy, however, rebellion replaced compliance as they attempted to assert themselves. Ultimately, daughters resorted to rejection of the primary symbol of maternal nurturance, food, in order to reinstate control over their own lives (Brusch, 1978).

*Social learning theories.* Since eating disorders run in families, some assert that disordered eating is another learned behavior. These theorists suggest that the emergence of disordered eating may occur because of an over-emphasis on eating in the family. The perception persists that daughters will more likely to fall into disordered eating if they come from families where parents use food to reward or punish their children and where appearance is emphasized. Research has demonstrated findings consistent with this theory, including revealing that mothers who dieted more were more likely to have daughters with an eating disorder (Gershon, et al., 1983), as well as discovering that daughter's body dissatisfaction was predicted by parental comments on weight (Smolak, Levine, & Schermer, 1999).

*Attachment models.* Attachment theory proposes that the bonds between infant and caregiver are essential in guiding the development of personal and social identity (Bowlby, 1982). Various theorists have argued that disruptions in attachment have played a considerable role in the development of eating pathology (Sugarman & Kurash, 1982). In particular, parental over-control and rewarding of dependence may have prevented a daughter from developing

appropriate self-reliance. Research appears consistent with this view, demonstrating that women with eating disorders display insecure attachment and separation distress (Armstrong & Roth, 1989), compulsive care-seeking and compulsive self-reliance (Ward, Ramsay, Turnbull, Benedettini, & Treasure, 2000), and childhood interactions that represent insecure and avoidant attachment styles (Dallos, 2004).

*Family systems conceptualizations.* As relayed previously, attempts to treat anorexia led many clinicians to utilize a systems perspective in order to understand the disorder, ultimately prompting the development of family systems approaches and family therapy. Family systems has a lot to say about eating disorders, with Minuchin leading the way, as described previously, in developing conceptualizations of the pathology. Minuchin et al. (1978) posited that families in which anorexia appeared were characterized by enmeshed boundaries between family members. These families exhibited parental alliances with the daughter, entering her into the spousal subsystem and triangulating her in the middle of the marital relationship. These undifferentiated boundaries between family members were exacerbated by the disengaged and particularly strong boundaries separating the family from other external relationships. Hence, the family would continue to maintain a façade of closeness to the outside world, while suppressing any opportunity for conflict expression within the system. As a result, the daughter somaticized the family problems through her eating disorder, representing within her own person the intrafamilial strife (Minuchin, et al., 1978).

Other authors of family systems approaches have established similar conclusions. Doerr-Zegers, Petrasic, & Morales (1985) claimed that an eating disorder family was characterized by an attitude of self-sacrifice in which ultimately the patient resolved to maintain the vulnerable homeostasis of the family through her illness. White (1983) additionally suggested that shame

was induced among family members to promote conformity. Selvini-Palazzoli (1974), on the other hand, asserted that guilt in the family remained unspoken as parents engaged in secret coalitions with their daughter and refused to directly communicate their needs. Thus, each family member in the triad was symbolically married to every other member, and the daughter was forced to attempt to divide her time equitably. Selvini-Palazzoli theorized that while family members' vocal messages were those of affection, unspoken non-verbal encounters sent the message of rejection.

#### *Research on Parents as Martyrs*

A new perspective has taken effect in recent years, as recognition of the deleterious consequences of blaming parents for their child's disorder has gained prominence. Instead of exacerbating the unrelenting guilt such blame has caused, attempts are being made to acknowledge how eating disorders hurt the family (Whitney & Eisler, 2005). Studies have found that the burden of caring for an individual with an eating disorder is severe, often resulting in mental health problems for the caregiver (Santonastaso, Saccon, & Favaro, 1997; Treasure, Murphy, Todd, Gavan, Joyce, & Szmukler, 2001). Monteleone et al. (2005) recognized the disadvantage of blaming families for the eating disorder, especially when research is primarily correlational and no specific causal direction can be assumed. These authors emphasize that the burden of tending to someone with an eating disorder may lead to feelings of confusion, anger, and desperation, which may in turn create the very dysfunctional system so many believe is at the root of the disorder.

### *Empirically Supported Themes*

Notwithstanding the copious theories and sometimes divergent views on the dynamics within an eating disorder family, some uniform conclusions do exist. Overall, eating disorder families do appear more dysfunctional than controls (Keel, 2005; McGrane & Carr, 2002), and within the population of those diagnosed, increased dysfunction appears to coincide with elevated symptom levels (Wistosky, et al., 2006). Current findings on these families indicate that such interactions correspond with a couple identifiable themes.

*Communication.* Families of those with eating disorders tend to demonstrate less effective communication styles (Casper & Troiani, 2001). Lack of empathy, increased conflict, and less nurturance have been regularly associated with these families (Humphrey, 1986, 1988, 1989; Humphrey, Apple, & Kirschenbaum, 1986). Communication among family members is frequently portrayed as conflicting, with contradictory messages of nurturance and neglect being expressed (Humphrey, 1989). One study highlighted patients' report of impaired communication with their mothers (Vidovic, et al., 2005). These families often also make direct accusations, placing blame on other family members (Humphrey, 1986, 1988, 1989; Humphrey, et al., 1986) as well as emphasizing physical appearance verbally and even criticizing their daughter's body (Crowther, et al., 2002; Smolak, et al., 1999). Additionally, emotional expression is deemphasized in families of those diagnosed (Casper & Troiani, 2001; Latzer, et al., 2002).

*Boundaries.* Families of individuals with an eating disorder often demonstrate rigid and even confusing boundaries. Kog and Vandereycken (1989b) explain that a pattern of both control and dependence is prevalent. Relationships in these families are inundated with conflicting messages regarding personal boundaries, often with both detachment and reliance being expressed (Humphrey, 1989; Ward, et al., 2000). Evidence indicates that intrusiveness, neglect,



rejection, isolation, and overprotection are all exhibited (Humphrey, 1986, 1988, 1989; Humphrey, et al., 1986; Rorty, Yager, & Rossotto, 2000; Shoebridge & Gowers, 2000).

Although evidence appears to support the notion that individuals with eating disorders have more dysfunctional families, findings do not institute the direction of the relationship or suggest that families have any impact on outcomes. Moreover, although this research concludes that families in distress demonstrate worse symptomology, findings do not offer guidance for change or implication for treatment. The family has clearly been associated with both development and maintenance of the disorder, but are family relationships related to recovery? The influence of family dynamics on healing remains unknown. Given the difficulty of treating eating disorders, this area warrants further investigation.

#### *Prognosis*

Despite many etiological theories of eating disorders and numerous modalities of treatment, prognosis for recovery from these disorders still appears relatively bleak. Death is a very real outcome of these diagnoses with anorexia currently holding the highest risk of premature death of all psychiatric disorders (Harris & Barraclough, 1998). Across outcomes studies, approximately 5% of patients with anorexia and .3% of patients with bulimia do not survive (Keel & Mitchell, 1997; Steinhausen, 2002). Although recovery is possible, many patients struggle throughout their lifetime. Reviews of research suggest approximately 30–40% of women with anorexia recover, while 30–35% improve, and 20–25% remain ill (Richards, et al., 2000; Steinhausen, 2002). Rates for women with bulimia appear more hopeful, with the literature suggesting that approximately 50% of individuals recover, 30% improve, and 20% remain in chronic condition (Keel & Mitchell, 1997; Hsu 1995). Relapse, however, is quite common among both disorders. Approximately one-third of women previously asymptomatic

return to anorexic behaviors (Keel, 2005) while about 30% of women with bulimia relapse after recovery (Keel & Mitchell, 1997).

### *Predicting Outcomes*

Researchers have attempted to gain an understanding of what distinguishes patients who recover from those who do not by examining predictors of outcome.

#### *Characteristic Factors*

*Age of onset.* There has been some debate regarding predictability of outcome based on age. In a meta-analysis on outcomes for anorexia, 13 studies suggested that an older age of onset was associated with a worse prognosis, while 14 studies found no such correlation, and two studies demonstrated that a younger age at onset predicted more favorable outcomes (Keel, 2005; Steinhausen, 2002). For bulimia, fewer studies have highlighted age as a prognostic factor (Keel, 2005); however, Yager (1989) found results consistent with research on anorexia demonstrating that a younger age of onset predicted recovery. Furthermore, researchers have suggested that measurement of age has been contradictory across studies with some utilizing age when the disordered eating began and others using age at clinical presentation, potentially years following onset of the disorder (Swift, 1982).

*Duration of illness.* Researchers appear to consistently report findings that chronicity of eating disorders leads to less favorable outcomes, for both anorexic and bulimic diagnoses. Hence, the longer an individual has an eating disorder, the less likely she is to recover (Keel, 2005; Richards, et al., 2000; Steinhausen, 2002) and the shorter the interval between symptom onset and treatment, the better the prognosis (Richards, et al., 2000).

*Duration of follow-up.* Recovery rates for patients with anorexia increase as duration of follow-up increases, suggesting that individuals with anorexia continue to recover over time

(Herzog, et al., 1999; Keel, 2005). However, for bulimia, a short duration of follow-up was related to more positive outcomes, while a longer duration did not predict outcomes in either direction (Keel & Mitchell, 1997).

### *Symptomatic Factors*

*Severity.* Across studies and disorders, findings remain consistent that the severity of symptoms indicates prognosis, with more severe symptomology predicting less favorable outcomes (Hsu, 1995; Richards, et al., 2000). Rate of purging in particular appears to be an especially clear indicator of outcome, with numerous studies identifying both bulimics and anorexics who engage in compensatory behaviors as less likely to recover (Baell & Wertheim, 1992; Deter & Herzog, 1994; Deter, Schellberg, & Koop, 2005; Herzog, Schellberg, & Deter, 1997; Steinhausen, 2002; Steinhausen, Rauss-Mason, Siedel, 1991). In anorexia, weight at referral also predicted poor outcomes (Bizeul, Sadowsky, & Rigaud, 2001; Finfgeld, 2002; Herzog, et al., 1997).

*Comorbidity.* Psychological characteristics forecast treatment outcomes as well with comorbid diagnoses presenting a limitation to recovery (Fichter & Quadflieg, 2004). Those diagnosed as having personality disorders, such as borderline and obsessive-compulsive personality disorder, are less likely to display a reduction of symptoms (Steiger & Stotland, 1996; Steinhausen, 2002), while those who maintain traits such as impulsivity (Keel & Mitchell, 1997), low self-esteem (Bell, 2002; Baell & Wertheim, 1992; Fairburn, Kirk, O'Conner, Anastasiades, & Cooper, 1987), ineffectiveness (Baell & Wertheim, 1992; Bizeul, Sadowsky, & Rigaud, 2001), and a history of substance abuse are also at odds for recovery (Keel, 2005).

*Familial Factors*

Unlike theories of causality, few studies of long-term outcome have attempted to identify social or relational prognostic factors. Long-term prediction has remained in the realm of characteristic variables, giving some credence to symptom severity and psychological predictors, while leaving social influences almost entirely unexamined. Despite a rich literature of family influences on the development of eating disorders, parents have been virtually left out of the realm of research on recovery. In a review of the outcome literature, Steinhausen et al. (1991) concluded that conflict-free, parent-child relationships for individuals with anorexia were associated with good outcome; however, few studies appear to examine these variables.

*Evidence of Family Impact on Outcomes*

Only a handful of studies have attempted to research how relationships impact outcome. Despite the lack of research on familial predictors of prognosis, some studies associate family functioning with outcomes. Strober, Freeman, and Morrell (1997) asked participants and their families to respond to interview questions regarding sibling and interfamilial relationships, including marital functioning, areas of conflict, methods to employ conflict resolution, parental attitudes regarding discipline, autonomy, intimacy, and expression of emotion. They were then reassessed at the patients discharge from treatment to measure differences from intake to termination. The study concluded that time to recovery was lengthened for participants with disturbances in family relationships. Although this study helps establish a relationship between family problems and length of treatment, these findings do not explain whether the familial influences persisted after termination.

Wallin and Kronvall (2002) studied twenty-six families of patients with anorexia to identify prognostic factors affecting the outcome of treatment. Together families completed

several self-rating questionnaires, participated in a semi-structured interview, and then completed three short tasks that were videotaped for later rating. Families had to arrive at a consensus on four questions from the Family Relation Scale, discuss the circumstances that initiated their call to the clinic for treatment, and finally complete a jigsaw puzzle together. These interactive activities were videotaped and rated by observers following the tasks based on cohesion, adaptability, hierarchal organization, family competence, and family style. At a two year follow-up these procedures were performed a second time. Supporting the idea that recovery is founded in improvement in familial interactions, it was found that for fully recovered patients, there was a significant improvement in nearly all measured dimensions, while partially recovered and chronically ill courses of outcome were not associated with this same magnitude of change. The recovered group appeared more competent, more expressive, more cohesive and less enmeshed, and more adaptable, with clearer hierarchal boundaries. These findings support the notion that family improvement elicits recovery; however, this particular study only examined the families of those with anorexia.

Another study completed by Bulik et al. (2000) contacted 70 previously terminated anorexic patients at a 12 year follow-up and had them fill out measures of their current symptomology and functioning. Based on their responses to the Eating Disorders Inventory and additional self-report of symptoms, participants were divided into fully recovered, partially recovered, and chronically ill groups. It was discovered that the chronically ill group reported significantly lower maternal care than both the fully and partially recovered groups and significantly lower paternal care than the fully recovered group. One limitation of this study was that patients were located following termination and assessed only at long-term follow-up, hence

results could not conclude whether perception of care had changed over time or if it had remained the same from intake to follow-up.

Castro et al. (2000) examined participants' self-reported memories of parental rearing practices to detect predictors of outcome. One hundred and fifty eight participants diagnosed with anorexia responded to the My Memories of Upbringing questionnaire, providing an assessment of adults' memories of their parents' rearing behavior to compare with a control group of non-diagnosable participants. It was found that patients with unfavorable outcomes reported greater perception of rejection and overprotection from both parents, with the highest predictive variable being rejection from father. Overall perceptions of parents; however, did not differ from that of controls. Unfortunately, yet again the sample only included those with anorexia and did not explore the perceptions of individuals with bulimia.

Herzog et al. (2000) used a systemic approach when analyzing patients and their families to identify predictors of outcome. Thirty eight women who were diagnosed with an eating disorder and their family were assessed using System for Multiple Level Observation of Groups (SYMLOG), an approach to family assessment based on interaction process analysis. The SYMLOG method allowed for analysis of individual personalities, dyadic relationships, and the comprehensive unit all simultaneously. The model of behavior included three primary components: dominance, friendliness, and goal orientation. Researchers found that family perception patterns were prognostic of outcome, with discrepancies in members' reported friendliness indicating a poorer outcome. The study was limited, however, by a small sample size, and a relatively simple outcome criterion (diagnosis).

The previous studies indicate that family dynamics may impact outcome, yet conclusions are tentative and limited. The current study attempts to build upon these findings in order to

establish the impact of family relationships on outcome above and beyond classical prognostic factors.

### *Hypothesis One*

The regression model of characteristic, symptomatic, and familial variables will significantly predict post-treatment symptoms on both the EAT and the Phone Survey. Additionally, the familial variables will significantly add to the predictive ability of the model, taking into account the previously included variables, demonstrated by a significant change in the F-value and an increase in the percent of variance explained when the familial variables are added to the equation.

### *Hypothesis Two*

The regression model of characteristic, symptomatic change, and familial change variables will significantly predict post-treatment symptoms on both the EAT and the Phone Survey. Additionally, the familial change variables will significantly add to the predictive ability of the model, taking into account the previously included variables, demonstrated by a significant change in the F-value and an increase in the percent of variance explained when the familial variables are added to the equation.

## Method

### *Setting*

Data was collected from patients at the eating disorder treatment clinic, Center for Change, in Orem, Utah. Center for Change offers multi-dimensional, comprehensive treatment for women including acute inpatient care, long-term residential care, transitional follow-up, and outpatient services, to treat women with anorexia, bulimia, binge eating, and additional comorbid diagnoses. Center for Change opened in 1996 and is currently run by a team of nearly 50 staff from medical, psychological, and nutritional fields. Patients participate in an individualized approach to healing, including individual, group, and family therapy, dietary counseling, experiential interventions, recreational treatment, and life skills education.

Treatment at Center for Change is guided by current research and established guidelines for eating disorder management. The center's treatment approach is founded on the principles that eating disorders are learned behaviors which can be changed and everyone has the capacity for recovery. The healing process at Center for Change is guided by the belief that a nurturing setting, high quality interventions, and appropriate aftercare are imperative for transformation to occur. Family therapy is an integral part of treatment at Center for Change. Families of adolescents are involved in weekly therapy sessions, and families are encouraged to attend Family Week in person to spend quality time improving their relationships. Treatment intensity is personalized for each patient based on their initial assessment and ongoing evaluations, and is gradually tapered as progress is made (Center for Change, 2007).



### *Procedures*

Upon admission into treatment, a battery of assessment instruments was routinely administered to women during their first week at Center for Change. Additionally, a diagnostic evaluation was completed by an intake therapist. Patients were notified during the evaluation that written and vocal responses would be utilized for assessment and research purposes and that their participation in research was voluntary and would remain confidential.

At termination of treatment, participants completed an additional collection of measures, filling out many of the same inventories they did at admission in conjunction with the primary therapist's concluding evaluation. After a period of time, ranging from 2 to 62 months post-treatment, participants were contacted again to participate in a follow-up study. They were contacted by both phone and mail. Over the phone, participants responded to an oral survey, developed by the Center for Change research team, regarding current eating behaviors, general distress, and everyday functioning. Through the mail they were sent another battery of measures to complete and mail back to the center. Both the Institutional Review Board at Center for Change and Brigham Young University approved use of the data collected for research, and once permission was granted by patients to utilize their responses, data was entered into the Center for Change electronic database.

### *Measures*

In order to examine the influence of familial predictors on outcome, a variety of instruments were utilized. Patients were evaluated a number of ways, including assessment of general characteristics, symptom severity, familial relationships, and post-treatment outcomes.

### *Characteristic Variables*

*Demographics.* At admission to treatment, patients completed the Diagnostic Survey of Eating Disorders, a written intake questionnaire which provides demographic information as well as historical and current accounts of disordered eating and body image. Responses regarding demographic measures including ethnicity, religious affiliation, marital status, and education, were assembled for purposes of describing the population sample. Additional reports of age at admission to treatment and age of disordered eating onset were utilized as predictors of outcome in the statistical analysis.

*Diagnosis.* Patient diagnosis of Anorexia Nervosa, Bulimia Nervosa, or Eating Disorder Not Otherwise Specified, as determined at admission through the intake interview, was included as a potential predictor of outcome in the analysis.

*Childhood sexual abuse.* Patient confirmation of childhood sexual abuse on the Life Events Survey taken at admission as well as verification of a sexual abuse history during the intake evaluation was included in the analysis as a predictor variable. Indication of childhood sexual abuse was recorded as either “yes” or “no” without any attempt to measure the extent or severity of the experience.

### *Symptomatic Variables*

*Outcome Questionnaire 45.2 (OQ-45.2).* The outcome questionnaire is a symptom and distress inventory utilized in therapy to assess patient functioning (Lambert, et al., 1996). The measure consists of 45 items each on a 5 point Likert scale and was administered to women at the center upon admission, at termination, and at follow-up. The questionnaire can be divided into three subscales: Subjective Discomfort (SD), Interpersonal Relationships (IR), and Social Role Performance (SR). Psychometric evaluations of the scale have demonstrated internal

consistency levels of .93 and test-retest reliability of .84 (Kadera, Lambert, & Andrews, 1996; Umphress, Lambert, Smart, Barlow, & Clouse, 1997). Summing the three subscales derives a total score which provides a global estimate of disturbance. The total score may range from 0 to 180, with higher scores indicating more symptom distress. Total scores above 70 are viewed as clinically significant (Lambert, et al., 1996). The OQ-45.2 is regularly utilized to assess patient progress in therapy and Lambert and colleagues have demonstrated reliable change indexes that represent significant clinical reduction of symptoms (Lambert, et al., 1996). A reduction of 14 or more points on a patient's total score indicates a significant decline in overall distress. Patient scores on the OQ-45.2 at both admission and termination were included as predictors of outcome in the statistical investigation.

*Global Assessment of Functioning (GAF).* The Global Assessment of Functioning is a subjective rating ranging from 0 to 100 assigned to patients by mental health professionals. Lower scores indicate poorer psychological, social, and occupational functioning. Throughout treatment at Center for Change the patient's GAF score is regularly documented based on criteria established in the DSM-IV-TR (American Psychiatric Association, 2000). The GAF scores reported at admission and termination were utilized in the analysis. The GAF recorded at admission was used as a predictor variable and a GAF change score was created to represent the difference of between each participant's GAF score at admission and termination. This GAF change score was also utilized as a predictor in the analysis.

#### *Familial Variables*

*Parent Acceptance-Rejection Questionnaire (MARQ & FARQ).* The Mother (MARQ) and Father (FARQ) Acceptance-Rejection Questionnaires indicate individuals' reported perception of the treatment they received from their parents while they were children. These

surveys were given to patients at admission to the center. Each measure is composed of 60 items, with a possible range of scores from 60 to 240. Higher scores suggest more experienced rejection, indifference, and hostility. Scores above 150 indicate the respondent views the parent as cold, indifferent, rejecting, neglectful, and hostile, with higher scores signifying more intense perception of rejection. Scores below 120 suggest a perception of the parent as warm, affectionate, and accepting, with lower scores corresponding with stronger perceptions of acceptance. With a sample of 147 college students both reliability and validity of the measure was found to be adequate. Internal consistency ranged from .86 to .95 and construct, concurrent, convergent, and discriminate validities of the scale were deemed sufficient (Rohner, Saavedra, & Granum, 1978). Patient scores at admission were included as predictors in the analysis.

*Attitudes Toward Parents Scale (AFS & AMS).* At both admission and termination, patients took the Attitudes Toward Father Scale (AFS) and Attitudes Toward Mother Scale (AMS) to assess the individual's reported extent, degree, and severity of problems with her parents. The scales measure degree of contentment or contention that exists with one's parents, ranging from 0 to 100, with higher scores representing more anger, resentment, distrust, and dislike. Scores above 30 are considered clinically significant and indicate the present of a problem with one's parents. The instrument has demonstrated reliability with a mean alpha of .95 and test-retest correlations at .95 as well (Hudson, 1982). Scores at both admission and termination were utilized for purpose of predicting outcome. Admission scores were entered as predictors in the analysis as well as change scores computed by the difference between scores from admission to termination.

*Post-Treatment Outcome Measures*

*Eating Attitudes Test (EAT)*. The EAT is a 40 item survey that assesses symptoms of anorexia and bulimia. Respondents endorse eating behaviors and attitudes on a 6 point Likert-scale ranging from “always” to “never”. For clinical purposes, the EAT is typically scored from 0 to 3 (Garner & Garfinkel, 1979), with the three bottom-most selections receiving the same numerical value (zero). However, with the intent of maintaining variability of responses for research purposes, each selection was allocated a separate score, ranging from 0 points for the response “never” to 5 points for the answer “always”. Hence, the possible span of total scores ranges from 0 to 200, with higher scores indicating more eating pathology. The EAT has demonstrated that it is a reliable and valid measure of eating problems, with an alpha coefficient ranging from .79 to .94 and a test-retest reliability coefficient of .84 (Fisher, Pastore, Schneider, Pegler, & Napolitano, 1994; Garner & Garfinkel, 1979; Hesse-Biber & Marino, 1991). The EAT has demonstrated significant correlation with diagnostic criterion ( $r = .87$ ) and sensitivity to clinical remission (Garner & Garfinkel, 1979). This measure was completed by patients at admission, termination, and at various post-treatment follow-up intervals. Scores on the post-treatment administration of the EAT represented long-term outcomes in the analysis.

*Phone Survey*. The Center for Change research team developed a measure of outcome that could be conducted by phone (see Appendix). This survey was administered at long-term follow-up intervals by a staff member at Center for Change who contacted former patients by phone and asked them questions about their perceptions of their recovery and general wellbeing. The survey consisted of 12 questions which included current eating patterns and frequency of bulimic or anorexic behaviors in the past month, ratings of recent relationship quality, satisfaction with life, emotional functioning, and ability to fulfill daily social roles. A total long-

term outcome score was computed based on patients' responses to the Phone Survey with higher scores indicating more disturbances. Since the Phone Survey was a newly created measure, psychometric tests were run to determine its properties and appropriateness for the study. Internal consistency was analyzed, resulting in a Cronbach alpha of .83, above the typical .80 cutoff expected for a respectable reliability for research purposes. Additionally a factor analysis was run which demonstrated that the measure is composed of three components. Examining the questions that loaded onto each factor gave reason to conclude that the three factors could be labeled (1) outcome distress symptoms (depression, anxiety, social role conflict, and relationship conflict), (2) eating disorder symptoms (binging, purging, restricting, and laxative use), and (3) life satisfaction. When these three factors were scored as subscales, none of the Cronbach Alphas on these subscales exceeded .80, providing support for the decision to compute an overall total score based on all of the items. Finally, an analysis of variance was computed to see if patients with different eating disorder diagnoses differed on their total Phone Survey scores. They did not significantly differ from each other, providing supportive evidence that the Phone Survey is a valid long-term outcome measure for eating disorder patients, regardless of eating disorder diagnosis. In regards to convergent validity data, the Phone Survey correlated with both the EAT ( $r = .58$ ) and the OQ-45.2 ( $r = .62$ ) at long-term follow-up providing evidence that the phone survey correlates well with established standardized measures of eating disorder and psychological symptoms at long-term follow-up.

### *Sample*

In order to discover relational predictors of long-term treatment outcomes, data was gathered from 398 women who received inpatient treatment for an eating disorder. As a result of some patients' partial responses to the surveys, the actual number of participants in the study

varied based on the measure examined. The average age of the participants was 22.03 and ranged from 13 to 56 years. The majority of the participants were single (80.2%), while 17.2 percent were married, 2.3 percent were divorced, and .3 percent of the participants were separated. The majority of the participants indicated a Caucasian ethnicity at 96.5 percent, while .6 percent identified as black, .6 percent as Asian, and .3 as Hispanic. Seventy-two percent of the participants reported a religious affiliation with the Church of Jesus Christ of Latter-day Saints (LDS) while 8.3 percent of participants indicated no membership in an organized religion. The percentage of participants that designated themselves as Catholic was 5.3 percent, while 4.7 designated themselves as Protestant, and .9 percent reported being Jewish. The remaining participants reported an affiliation with numerous “other” select religions. The average age of onset of the eating disorder was 15.43. The data suggests the majority of the patients entered treatment in the middle of college, with 44.1 percent of the participants indicating their highest level of education as “some college”. A total of 21.4 percent of participants designated their highest level of education as “some high school,” while 18.8 percent graduated high school and 10.4 percent graduated college. A small percentage of participants (3.5%) had not yet completed junior high when admitted for treatment.

### *Analysis*

In order to examine the relationship between post-treatment outcomes and predictor variables multiple regression analyses were conducted. Specifically, it was determined that a sequential multiple regression would be most appropriate for the purpose of the analysis. A sequential, or hierarchical, multiple regression allows the researcher to specify the order in which the predictor variables are entered into the analysis (Mertler & Vannatta, 2005). Sequential regressions are utilized when substantive knowledge leads the researcher to believe that specific

variables in a set of predictors may be more influential than others, and those variables are entered into the analysis first (Mertler & Vannatta, 2005). Succeeding variables are then added into the analysis to establish the magnitude of variance they can account for above and beyond what has been explained by preceding variables (Aron & Aron, 1999). As identified previously, prior research on predictors of eating disorder outcomes has already established the predictive ability of certain characteristic and symptomatic variables, hence for the purpose of this study, these variables were entered in the analysis first. In order to determine if familial variables explain additional variance above and beyond those known predictors, familial measures were entered into the analysis last. Thus, predictor variables were entered into the model in groups, in a pre-established order. The first block of predictors entered into the model included characteristic variables (age at admission, age of disordered eating onset, diagnosis, history of childhood sexual abuse, and length of post-treatment follow-up). The second block entered included symptomatic variables represented by scores on the Outcome Questionnaire and the Global Assessment of Functioning. The third and final block entered into the model incorporated the familial variables of scores on the Parent Acceptance-Rejection Questionnaire and the Attitude Toward Parents Scales. Four sequential regressions were computed in accordance with the studies' research questions (two regressions per question). In response to the first question, predictor variables were included in block two and three that were based on measures taken at admission to treatment. For the second question, predictor variables based on patients' change in scores from admission to termination were included in the analysis. Separate regressions were run on two different measures of long-term outcomes: scores on the EAT and the Phone Survey. Variables were entered using pairwise deletion to utilize all of the gathered data and handle missing information.



## Results

### *Descriptive Statistics*

Participants in the study completed many instruments and for the planning of inferential analyses descriptive statistics were initially explored including running measures of central tendency (see Table 1) and bivariate correlations (see Table 2). Additionally, paired sample t-tests were run for purposes of describing the change participants experienced from admission to termination of treatment.

### *Characteristic Variables*

A total of 398 patients were included as participants in the analysis, and a diagnosis was recorded for 348 of the patients. A total of 135 patients were diagnosed with anorexia, while 103 were diagnosed with bulimia, and 110 were diagnosed with an eating disorder not otherwise specified. The length of follow-up ranged from 2 to 62 months ( $N = 278$ ,  $M = 12.81$ ,  $SD = 9.22$ ). The average Body Mass Index (BMI), a statistical measure that compares an individual's weight and height, for those diagnosed with anorexia averaged 15.49 at intake and 18.56 at termination. Participants who were diagnosed with bulimia averaged a BMI of 22.02 upon admission and a BMI of 20.83 at discharge. A BMI of between 18.5 and 25 is considered within a normal healthy range. These reports seem typical of an inpatient eating disorder population, especially considering the distinction in weight range criteria for the separate diagnoses. The average age of admission to treatment was 22.03 ( $N = 349$ ,  $SD = 7.53$ ) and the average age of onset of the disorder was 15.43 ( $N = 347$ ,  $SD = 4.85$ ). Again, these ages appear representative of an eating disorder population, given that most eating disorders develop from mid to late adolescence and most women do not receive treatment at the outset of the disorder. Additionally, 109 of the participants reported experiencing childhood sexual abuse ( $N = 358$ ) (see Table 1).

Table 1

*Central Tendency of Predictor and Outcome Variables*

Measure	N	M	SD
Age at Admission	349	22.03	7.53
Age of Onset	347	15.43	4.85
Length of Follow-up	278	12.81	9.22
Phone Survey	278	30.76	7.63
OQ 45.2 Admission	357	53.62	21.67
OQ 45.2 Discharge	320	53.62	21.67
GAF Admission	336	39.68	7.82
GAF Discharge	329	56.19	11.38
MARQ	345	92.46	40.45
FARQ	339	97.62	40.16
AMS Admission	354	29.96	27.46
AFS Admission	338	29.39	25.89
AMS Discharge	309	25.32	22.91
AFS Discharge	296	25.30	23.17
EAT-40	214	84.79	38.07
Phone Survey	278	30.76	7.63

Table 2

*Intercorrelations Between Predictor and Outcome Variables*

Variables	1	2	3	4	5	6
1. Age at Admission	–					
2. Age of Onset	.50**	–				
3. Anorexia	.06	.05	–			
4. Bulimia	-.18*	-.05	-.42**	–		
5. Childhood Sex Abuse	.20**	-.01	-.03	-.11*	–	
6. Length of Follow-up	-.06	-.10	-.01	-.01	-.07	–
7. OQ at Admission	.07	.06	-.05	-.02	.21**	-.17*
8. OQ Change	.08	.07	-.05	.07	.10	-.22
9. GAF at Admission	.10	.06	-.13*	.02	-.16**	.04
10. GAF Change	.05	.04	.04	.03	-.01	-.02
11. MARQ	.18	-.04	.03	-.04	.23**	-.06
12. FARQ	.14*	.02	-.12*	-.10	.25**	-.07
13. AMS	.12*	-.07	-.01	-.04	.18**	-.12
14. AMS Change	.02	-.03	-.04	.02	-.03	-.08
15. AFS	.09	-.03	-.10	-.07	.23**	-.12
16. AFS Change	.05	.03	-.08	.11	.06	-.04
17. EAT-40	.15*	.04	-.04	-.08	-.12	.08
18. Phone Survey	.21**	.10	-.01	-.02	.02	-.03

*Note.* \*\* $p < .01$ , two-tailed. \* $p < .05$ , two-tailed.

Table 2 continued

*Intercorrelations Between Predictor and Outcome Variables*

Variables	7	8	9	10	11	12
1. Age at Admission						
2. Age of Onset						
3. Anorexia						
4. Bulimia						
5. Childhood Sex Abuse						
6. Length of Follow-up						
7. OQ at Admission	–					
8. OQ Change	.57**	–				
9. GAF at Admission	-.18**	.03	–			
10. GAF Change	.08	.08	-.47**	–		
11. MARQ	.32**	.12*	-.19**	.04	–	
12. FARQ	.39**	.15*	-.10	.03	.52**	–
13. AMS	.33**	.09	-.16**	.05	.78**	.39**
14. AMS Change	.23**	.31**	.01	.03	.35**	.20**
15. AFS	.36**	.16**	-.08	.00	.38**	.75**
16. AFS Change	.20**	.28**	-.01	.00	.14**	.30**
17. EAT-40	.15*	-.07	-.11	-.11	.01	.10
18. Phone Survey	.24**	.03	-.06	.02	.29**	.21**

*Note.* \*\* $p < .01$ , two-tailed. \* $p < .05$ , two-tailed.

Table 2 continued

*Intercorrelations Between Predictor and Outcome Variables*

Variables	13	14	15	16	17	18
1. Age at Admission						
2. Age of Onset						
3. Anorexia						
4. Bulimia						
5. Childhood Sex Abuse						
6. Length of Follow-up						
7. OQ at Admission						
8. OQ Change						
9. GAF at Admission						
10. GAF Change						
11. MARQ						
12. FARQ						
13. AMS	–					
14. AMS Change	.52**	–				
15. AFS	.53**	.29**	–			
16. AFS Change	.27**	.53**	.47**	–		
17. EAT-40	-.03	.02	-.08	.02	–	
18. Phone Survey	.11	.09	.05	-.04	.33**	–

*Note.* \*\* $p < .01$ , two-tailed. \* $p < .05$ , two-tailed.

*Symptomatic and Familial Predictors*

Scores on numerous measures were explored as potential predictors of outcome. Symptom severity measures included totals on the OQ-45.2 and patients' GAF scores (see Table 1). Participant averages on the OQ-45.2 indicate symptoms above clinical cut-offs at admission, while scores at termination reduced on average more than 34 points, indicating a significant decline in overall distress ( $t = 25.62$ ,  $df = 307$ ,  $p < .01$ ). Average scores on the GAF at admission indicated major impairment in several areas of functioning while averages at termination demonstrate improved psychological, social, and occupational functioning with symptomology reduced to moderate levels. This improvement from admission to termination was statistically significant ( $t = -22.95$ ,  $df = 325$ ,  $p < .01$ ). Familial predictors were gathered from scores on the MARQ, FARQ, AMS, and AFS questionnaires. Scores on both the MARQ and FARQ, taken only at admission, showed averages which indicated participants' generally felt accepted by their parents. On the AMS and AFS at admission averaged near the clinical cut-off of 30 indicating that participants had a great degree of contention with their parents. At termination, these scores dropped approximately 4 points on average, a significant difference, indicating an improved perception of both one's father ( $t = 3.38$ ,  $df = 272$ ,  $p < .01$ ) and one's mother ( $t = 4.57$ ,  $df = 293$ ,  $p < .01$ ).

*Post-Treatment Outcome Measures*

The EAT was used as a measure of disordered eating at post-treatment. Of the patients surveyed, 214 individuals responded to EAT questionnaire. Of this sample, the mean for the EAT was 84.79 (SD = 38.07) demonstrating that some moderate disordered eating symptoms still persisted even at follow-up. Responses to the phone survey developed by Center for Change

research team was also used as an additional measure of long-term outcome. A total of 278 patients completed this measure, averaging a mean score of 30.76 (SD = 7.63) (see Table 1).

### *Inferential Analyses*

#### *Research Question One*

Which factors, including characteristic variables (age at admission, age of onset, eating disorder diagnosis, history of sexual abuse, length of follow-up), symptomatic variables (OQ-45.2 & GAF), and familial variables (MARQ, FARQ, AMS, AFS) are significant predictors of post-treatment eating disorder symptoms (EAT) and general wellbeing (Phone Survey)?

In order to determine which variables significantly predict post-treatment outcomes for eating disorders, two separate sequential regressions were run. As detailed previously, scores on measures completed at admission were entered as predictor variables, while the EAT was utilized as the dependent variable of the first regression, and the second regression employed scores on the Phone Survey as the post-treatment outcome measure.

Results on the first regression (see Table 3) revealed that entering the first block of characteristic predictors into the regression model did not significantly predict post-treatment outcome measured on the EAT ( $F = 1.96$ ,  $df = 153$ ,  $p = .08$ ). However, upon entering the second block of symptomatic predictors, the regression model was able to significantly predict post-treatment disordered eating outcomes ( $F = 2.55$ ,  $df = 151$ ,  $p = .01$ ). And, finally, when the third block of familial predictors was included ability to predict eating symptoms at follow-up increased further ( $F = 2.74$ ,  $df = 147$ ,  $p < .01$ ). With all three blocks of predictors included in the analysis, the model was able to explain 18% of the variance of participants' follow-up EAT scores. In the equation, the change in the F-value from inputting the first block to adding the

second was significant ( $p = .02$ ). Adding the third block also increased the F-value a significant amount ( $p = .03$ ).

Table 3

*First Regression: Ability of Models to Predict Outcome on the EAT*

Model	R <sup>2</sup>	F	Sig.	R <sup>2</sup> Change	F Change	Sig. Change
1	.07	1.96	.08	.07	1.96	.08
2	.12	2.55	.01**	.05	4.09	.02*
3	.18	2.74	<.01**	.06	2.87	.03*

Note. \*\* $p < .01$ , \* $p < .05$ .

In examining the Beta standardized coefficients (see Tables 4, 5, & 6), the analysis revealed five measures in particular that had significant predictive relationships with the long-term outcome scores on the EAT: age at admission (Beta = .22,  $t = 2.33$ ,  $p = .02$ ), childhood sexual abuse (Beta = -.21,  $t = -2.62$ ,  $p = .01$ ), the OQ-45.2 at admission (Beta = .20,  $t = 2.30$ ,  $p = .02$ ), the FARQ (Beta = .40,  $t = 2.82$ ,  $p = .01$ ), and the AFS (Beta = -.42,  $t = -3.01$ ,  $p < .01$ ).

Table 4

*First Regression Model 1: Predictors of Outcome Block 1 on the EAT*

Block	Predictor Variables	Beta	t	Sig.
1 Characteristic Predictors	Age at Admission	.22	2.33	.02*
	Age at Onset	-.06	-.68	.50
	Anorexia Diagnosis	-.10	-1.16	.25
	Bulimia Diagnosis	-.12	-1.33	.19
	Childhood Sexual Abuse	-.18	-2.18	.03*
	Length of Follow-Up	.08	.97	.33

Note. \*\* $p < .01$ , \* $p < .05$ .



Table 5

*First Regression Model 2: Predictors of Outcome Blocks 1 & 2 on the EAT*

Block	Predictor Variables	Beta	t	Sig.
1 Characteristic Predictors	Age at Admission	.21	2.33	.02*
	Age at Onset	-.08	-.86	.39
	Anorexia Diagnosis	-.11	-1.27	.21
	Bulimia Diagnosis	-.12	-1.40	.16
	Childhood Sexual Abuse	-.23	-2.82	<.01**
	Length of Follow-Up	.11	1.34	.18
2 Symptomatic Predictors	OQ at Admission	.18	2.18	.03*
	GAF at Admission	-.12	-1.50	.14

Note. \*\* $p < .01$ , \* $p < .05$ .

Table 6

*First Regression Model 3: Predictors of Outcome Blocks 1, 2, & 3 on the EAT*

Block	Predictor Variables	Beta	t	Sig.
1 Characteristic Predictors	Age at Admission	.23	2.52	.01**
	Age at Onset	-.11	-1.20	.23
	Anorexia Diagnosis	-.08	-.98	.33
	Bulimia Diagnosis	-.10	-1.22	.23
	Childhood Sexual Abuse	-.21	-2.62	.01**
	Length of Follow-Up	.09	1.13	.26
2 Symptomatic Predictors	OQ at Admission	.20	2.30	.02*
	GAF at Admission	-.12	-1.57	.12
3 Familial Predictors	MARQ	-.23	-1.53	.13
	FARQ	.40	2.82	<.01**
	AMS at Admission	.14	.96	.34
	AFS at Admission	-.42	-3.01	<.01**

Note. \*\* $p < .01$ , \* $p < .05$ .

Results on the second sequential regression (see Table 7), in which the Phone Survey was used as the dependent variable, demonstrated that the first block of predictors did not result in a model that significantly predicted outcomes ( $F = 1.73$ ,  $df = 217$ ,  $p = .12$ ). However, entering the second block of predictors lead to a significant regression model ( $F = 3.05$ ,  $df = 215$ ,  $p < .01$ ) and adding the third block increased ability to predict eating symptoms at follow-up further ( $F = 3.11$ ,  $df = 211$ ,  $p < .01$ ). With all three blocks of predictors entered into the regression, the model predicted 15% of the variance of scores on the Phone Survey. Adding the third block of predictors increased the F-value significantly ( $p < .01$ ). The Beta coefficients reveal (see Tables 8, 9, & 10) three of the predictors in particular contributed significantly to the model's ability to predict the variance. Age at admission (Beta = .20,  $t = 2.56$ ,  $p = .01$ ), scores on the OQ-45.2 at admission (Beta = .21,  $t = 2.91$ ,  $p < .01$ ), and scores on the FARQ (Beta = .24,  $t = 1.98$ ,  $p = .05$ ) all significantly contributed to the regression model. These findings together on both regressions indicate that hypothesis one was confirmed.

Table 7

*Second Regression: Ability of Models to Predict Outcome on the Phone Survey*

Model	R <sup>2</sup>	F	Sig.	R <sup>2</sup> Change	F Change	Sig. Change
1	.05	1.73	.12	.05	1.73	.11
2	.10	3.05	<.01**	.06	6.73	<.01**
3	.15	3.11	<.01**	.05	3.00	.02*

Note. \*\* $p < .01$ , \* $p < .05$ .

Table 8

*Second Regression Model 1: Predictors of Outcome Block 1 on the Phone Survey*

Model	Predictor Variables	Beta	t	Sig.
Block 1 Characteristic Predictors	Age at Admission	.22	2.82	<.01**
	Age at Onset	-.02	-.21	.83
	Anorexia Diagnosis	-.02	-.32	.75
	Bulimia Diagnosis	-.01	-.05	.96
	Childhood Sexual Abuse	-.03	-.47	.63
	Length of Follow-Up	-.03	-.38	.70

Note. \*\* $p < .01$ , \* $p < .05$ .

Table 9

*Second Regression Model 2: Predictors of Outcome Blocks 1 & 2 on the Phone Survey*

Model	Predictor Variables	Beta	t	Sig.
Block 1 Characteristic Predictors	Age at Admission	.23	2.92	<.01**
	Age at Onset	-.03	-.42	.68
	Anorexia Diagnosis	-.01	-.15	.88
	Bulimia Diagnosis	<.01	.03	.98
	Childhood Sexual Abuse	-.08	-1.20	.23
	Length of Follow-Up	.01	.17	.87
Block 2 Symptomatic Predictors	OQ at Admission	.24	3.59	<.01**
	GAF at Admission	-.01	-.21	.83

Note. \*\* $p < .01$ , \* $p < .05$ .

Table 10

*Second Regression Model 3: Predictors of Outcome Blocks 1, 2, & 3 on the Phone Survey*

Model	Predictor Variables	Beta	t	Sig.
Block 1 Characteristic Predictors	Age at Admission	.20	2.56	.01**
	Age at Onset	-.03	-.35	.73
	Anorexia Diagnosis	-.01	-.07	.94
	Bulimia Diagnosis	.01	.12	.91
	Childhood Sexual Abuse	-.10	-1.43	.16
	Length of Follow-Up	-.01	-.12	.91
Block 2 Symptomatic Predictors	OQ at Admission	.21	-2.91	<.01**
	GAF at Admission	.00	.03	.98
	MARQ	.14	1.12	.27
Block 3 Familial Predictors	FARQ	.24	1.98	.05*
	AMS at Admission	-.05	-.42	.68
	AFS at Admission	-.22	-1.89	.06

Note. \*\* $p < .01$ , \* $p < .05$ .

*Research Question Two*

Are changes in symptom severity (OQ-45.2) and perceptions of parental relationships (AFS, AMS) from admission to termination of treatment significant predictors of post-treatment eating disorder symptoms (EAT) and general wellbeing (Phone Survey)?

In order to examine the significance of change predictors on eating disorder symptoms (see Table 11, 12, 13, 14) and general wellbeing (see Table 15, 16, 17, 18), two sequential regressions were conducted. Neither regression, however, revealed a model that significantly predicted outcome. None of the blocks of predictors on either follow-up measure significantly predicted outcome. Hence, hypothesis two was not supported.

Table 11

*Third Regression: Ability of Change Models to Predict Outcome on the EAT*

Model	R <sup>2</sup>	F	Sig.	R <sup>2</sup> Change	F Change	Sig. Change
1	.07	1.77	.11	.07	1.78	.11
2	.08	1.57	.14	.01	.95	.39
3	.09	1.27	.26	<.01	.13	.88

Note. \*\* $p < .01$ , \* $p < .05$ .

Table 12

*Third Regression Model 1: Change Predictors of Outcome Block 1 on the EAT*

Model	Predictor Variables	Beta	t	Sig.
Block 1 Characteristic Predictors	Age at Admission	.22	2.22	.03*
	Age at Onset	-.06	-.65	.52
	Anorexia Diagnosis	-.10	-1.10	.27
	Bulimia Diagnosis	-.12	-1.27	.21
	Childhood Sexual Abuse	-.18	-2.08	.04*
	Length of Follow-Up	.08	.92	.36

Note. \*\* $p < .01$ , \* $p < .05$ .

Table 13

*Third Regression Model 2: Change Predictors of Outcome Blocks 1 & 2 on the EAT*

Model	Predictor Variables	Beta	t	Sig.
Block 1 Characteristic Predictors	Age at Admission	.22	2.26	.03*
	Age at Onset	-.06	-.61	.54
	Anorexia Diagnosis	-.09	-1.04	.30
	Bulimia Diagnosis	-.11	-1.17	.24
	Childhood Sexual Abuse	-.18	-2.04	.04*
	Length of Follow-Up	.07	.86	.39
Block 2 Symptomatic Predictors	OQ Change	-.03	-.38	.71
	GAF Change	-.11	-1.29	.20

Note. \*\* $p < .01$ , \* $p < .05$ .

Table 14

*Third Regression Model 3: Change Predictors of Outcome Blocks 1, 2, & 3 on the EAT*

Model	Predictor Variables	Beta	t	Sig.
Block 1 Characteristic Predictors	Age at Admission	.22	2.21	.03*
	Age at Onset	-.06	-.59	.56
	Anorexia Diagnosis	-.09	-1.01	.31
	Bulimia Diagnosis	-.11	-1.19	.24
	Childhood Sexual Abuse	-.17	-2.01	.05*
	Length of Follow-Up	.07	.86	.39
Block 2 Symptomatic Predictors	OQ Change	-.05	-.52	.61
	GAF Change	-.11	-1.27	.21
Block 3 Familial Predictors	AMS Change	.02	.16	.87
	AFS Change	.03	.34	.73

*Note.* \*\* $p < .01$ , \* $p < .05$ .

Table 15

*Fourth Regression: Ability of Change Models to Predict Outcome on the Phone Survey*

Model	R <sup>2</sup>	F	Sig.	R <sup>2</sup> Change	F Change	Sig. Change
1	.05	1.70	.17	.05	1.53	.17
2	.05	1.15	.33	<.01	.07	.93
3	.07	1.34	.21	.02	2.04	.13

*Note.* \*\* $p < .01$ , \* $p < .05$ .

Table 16

*Fourth Regression Model 1: Change Predictors of Outcome Block 1 on the Phone Survey*

Model	Predictor Variables	Beta	t	Sig.
	Age at Admission	.22	2.65	<.01**
	Age at Onset	-.02	-.20	.84
Block 1	Anorexia Diagnosis	-.02	-.30	.76
Characteristic	Bulimia Diagnosis	-.01	-.05	.96
Predictors	Childhood Sexual Abuse	-.03	-.45	.65
	Length of Follow-Up	-.03	-.35	.72

*Note.* \*\* $p < .01$ , \* $p < .05$ .

Table 17

*Fourth Regression Model 2: Change Predictors of Outcome Blocks 1 & 2 on the Phone Survey*

Model	Predictor Variables	Beta	t	Sig.
	Age at Admission	.22	2.64	<.01**
	Age at Onset	-.02	-.23	.82
Block 1	Anorexia Diagnosis	-.02	-.30	.77
Characteristic	Bulimia Diagnosis	-.01	-.08	.94
Predictors	Childhood Sexual Abuse	-.04	-.48	.63
	Length of Follow-Up	-.02	-.31	.76
Block 2	OQ Change	.03	.34	.73
Symptomatic	GAF Change	.01	.12	.91
Predictors				

*Note.* \*\* $p < .01$ , \* $p < .05$ .

Table 18

*Fourth Regression Model 3: Change Predictors of Outcome Blocks 1, 2, & 3 on Phone Survey*

Model	Predictor Variables	Beta	t	Sig.
Block 1 Characteristic Predictors	Age at Admission	.22	2.63	.01**
	Age at Onset	-.01	-.07	.94
	Anorexia Diagnosis	-.02	-.28	.78
	Bulimia Diagnosis	.01	.13	.90
	Childhood Sexual Abuse	-.02	-.25	.80
	Length of Follow-Up	-.01	-.19	.85
Block 2 Symptomatic Predictors	OQ Change	.02	.20	.84
	GAF Change	.00	.06	.06
Block 3 Familial Predictors	AMS Change	-.14	1.82	.07
	AFS Change	.16	-1.66	.10

*Note.* \*\* $p < .01$ , \* $p < .05$ .



## Discussion

The purpose of the current study was to evaluate the relationship between eating disorder patients' perceptions of their parents and their long-term outcomes following treatment. The present investigation was intended to examine whether patient reports of their familial relationships was able to explain the variability of scores on post-treatment outcome measures, above and beyond known predictors. More specifically, the study aimed to determine if a patient's self-reported relationship with her parents as measured at intake predicted her post-treatment disordered eating and general well-being, having already taking into account the variance explained by known characteristic and symptomatic predictors of outcome.

Additionally, another goal of the study was to explore whether or not a change in a patient's attitude towards her parents also predicted outcomes, having previously taken into consideration known predictors. This study was important to the current knowledge base, given a previous dearth of research regarding the influence of familial relationships on outcomes. As familial explanations of the development of eating disorders are abundant in the literature, it was deemed appropriate to consider such relationships with the hope that they may also contribute to recovery. Consistent with the recommendation of Hsu (1995) as well as Wilson & Fairburn (1993) the current study focused on identifying such patient traits which may lead to recovery and attempted to uncover variables that predicted response to treatment.

This study was unique in several ways. First, it used a relatively large sample size ( $N = 398$ ) of women admitted to inpatient treatment, while much of the extant research has utilized small samples who received outpatient treatment. Second, the study incorporated patients with more than one eating disorder diagnosis (anorexia, bulimia, and Eating Disorder Not Otherwise Specified were all investigated) while past familial studies have primarily focused on anorexia as

the diagnosis of choice. Third, the study included measures taken at admission, discharge, and long-term follow-up. Fourth, the study utilized measures of outcome that were standardized instruments designed to assess degree of symptoms along a continuum. Previous studies have been limited by their design to explore differences between groups of recovered and chronic cases rather than explore predictability over a range of symptoms. Fifth, this study employed two measures of outcome: disordered eating and general well-being. Previous studies have focused solely on recovery from eating pathology. The final way in which the present investigation was unique is that a number of known predictors of outcome were taken into account in the analysis, so that any variance explained by familial variables would be above and beyond that explained by classical characteristic and symptomatic factors.

### *Findings*

#### *Hypothesis One*

Research question one explored how characteristic, symptomatic, and familial predictors measured at admission to treatment explained scores on both measures of disordered eating and general well-being. Hypothesis one was supported in the discovery that the model of characteristic, symptomatic, and familial predictors significantly predicted disordered eating and general well-being at follow-up. More specifically, adding the familial variables at the end of the analysis significantly increased the percentage of variance explained by the model. Hence, the conclusion can be drawn that the relationship these patients had with their parents predicts post-treatment outcomes, even after known characteristic and symptomatic predictors were taken into account.

Of special interest are the variables that contributed significantly to the model, including age at admission, childhood sexual abuse, the OQ-45.2, the FARQ, and the AFS for eating

symptomology outcomes, and age at admission, the OQ-45.2, and the FARQ for post-treatment well-being. Exploring the Beta coefficients for these factors explains how they relate to the outcome measure.

Looking at the age of admission variable, it appears that as age of admission increases, disordered eating at follow-up increases and post-treatment well-being is reduced. This finding appears consistent with the literature as it may suggest a longer duration of the disorder, and chronicity has repeatedly been associated with worse prognosis (Keel, 2005; Richards, et al., 2000; Steinhausen, 2002). The current study extends this finding beyond simple prediction of eating symptoms to overall well-being.

Scores on the OQ-45.2 also significantly contributed to the regression model, with more distress being associated with worse prognosis (i.e. a rise in disordered eating and a decline in general well-being). This finding is consistent with the idea that comorbidity of disorders and expression of other psychological symptoms decreases one's likelihood of recovery (Baell & Wertheim, 1992; Bell, 2002; Fairburn, et al., 1987; Fichter & Quadflieg, 2004; Keel, 2005; Steiger & Stotland, 1996; Steinhausen, 2002). Hence, as one reports more subjective discomfort, problems in interpersonal relationships, and difficulties with social role performance at admission to treatment, long-term symptoms are less likely to improve.

Finally, scores on the FARQ significantly contributed to both regression models in question one. Higher scores on the FARQ, a measure of reported levels of acceptance and rejection experienced from one's father, correlated with increased symptomology at post-treatment. Accordingly, the patient's perspective of her father as accepting or rejecting at admission to treatment significantly predicts her outcome at follow-up. Patients who view their fathers as more cold, indifferent, rejecting, neglectful, and hostile are less likely to have long-

term improved eating or well-being, while those who perceive their father as warm, affectionate, and accepting are more likely to have recovered from their eating disorder and report elevated well-being at follow-up. This result appears consistent with the discovery that chronically ill patients reported less paternal care than those who were fully recovered (Bulik, et al., 2000), as well as the finding that the highest predictor of unfavorable outcomes across parental variables was paternal rejection (Castro, et al., 2000). This discovery is consistent with hypothesis one in declaring that familial variables are important predictors of outcome and add to the predictability of the model. It is especially notable that perception of the paternal relationship has a greater impact on recovery than perception of the maternal relationship, which appears consistent with previous findings (Castro, et al., 2000). The influence of the paternal relationship corresponds with the focus of etiological literature on the father, describing him as more emotionally distant and unreachable than the mother (Kog & Vandereycken, 1989a).

Aside from the three variables that significantly contributed to both models, there were two factors in particular which also contributed significantly to the prediction of disordered eating, but did not contribute the model for general well-being. The first of these was childhood history of sexual abuse. Those women who indicated childhood experience(s) of sexual abuse were surprisingly more likely to recover from their eating symptomology than those who did not have such an experience. Although, traumatic events have been regularly associated with the theoretical explanations of the pathogenesis of eating disorders (Rodriguez, Perez, & Garcia, 2005), major meta-analyses assert that sexual abuse is not specifically associated with the development of an eating disorder (Wonderlich, Brewerton, Jolic, Dansky, & Abbott, 1997; Smolak & Murnen, 2002). Yet, in 2005, Rodriguez, Perez, & Garcia found that exposure to sexual trauma was associated with response to treatment, and that for those receiving outpatient

therapy the highest probability of poor outcome was associated with those who had experienced sexual abuse. The current finding flies in the face of the conclusion that childhood sexual abuse impedes recovery, indicating that those who reported childhood sexual abuse actually had better outcomes.

The second variable which predicted disordered eating but not general well-being is perhaps the most interesting and surprising predictor in the model. This predictor is the AFS, a measure of an individual's severity of problems with her father, which, contrary to expectations, displayed an inverse relationship with post-treatment symptomology. That is, as a patient's reported problems with her father increased, her disordered eating at follow-up decreased. Despite the significantly strong correlation between the AFS and the FARQ ( $r = .75$ ), the measures functioned very differently in the regression model. Perhaps this is due to their distinct instrumental value. While the FARQ assesses the patient's perspective on how her father treated her, the AFS explores her opinion of her father. Additionally, the measures diverge in temporal reference as well. Whereas the FARQ focuses on past events, the AFS assesses the patient's current perspective on her relationship with her father. Some conclusions can be drawn from this distinction. It may be that even as greater sense of paternal acceptance during childhood leads to more positive outcomes, a more negative evaluation of one's current relationship with her father at intake predicts a more favorable prognosis. Considering the literature on inconsistent family boundaries among those with eating disorders and a pervasive façade of closeness as well as denial of conflict despite relational struggles (Kog & Vandereycken, 1989a), this seemingly paradoxical finding may make sense theoretically. Perhaps it could be understood under the supposition that as patients are more candid about their current discontent in their relationship with their father, their likelihood of recovery increases. For those patients who uphold the

pretense of closeness at intake, disordered eating is retained at follow-up. Future research will be needed to further investigate this possibility.

### *Hypothesis Two*

The second research question emphasized the predictability of change variables on disordered eating and general well-being. Hypothesis two, surprisingly, was not supported, as neither regression was significant. Although change in symptom severity from admission to discharge was significant for both the OQ-45.2 ( $t = 25.62$ ,  $df = 307$ ,  $p = .00$ ) and the GAF ( $t = -22.95$ ,  $df = 325$ ,  $p = .00$ ), and patients' attitudes significantly changed towards their fathers ( $t = 3.38$ ,  $df = 272$ ,  $p = .00$ ) and mothers ( $t = 4.57$ ,  $df = 293$ ,  $p = .00$ ) from intake to termination, these changes did not predict post-treatment outcomes. Consequently, the lack of significance of the model suggests that while characteristic, symptomatic, and familial variables collected at intake predict long-term outcomes, patients' changes in symptoms and perception of parents may not.

### *Limitations*

There are some limitations to consider when interpreting the results of this study. Concerns regarding the sample utilized and some statistical issues are important to acknowledge.

### *Sample Issues*

Given that the sample consists of inpatient participants, findings may not be generalizable to a less severe, outpatient population. Additionally, the majority of the participants in the study were Caucasian, and all patients were women, limiting the application of the findings to men and ethnically diverse groups. More importantly, however, is the fact that because the long-term measures were gathered following termination of treatment and participants' responses were voluntary, measures were not collected from all the patients who received treatment at Center for

Change. Hence, participants self-selected into the study and it is possible that those who choose not to be included may have responded differently.

### *Statistical Concerns*

Because data was missing from a number of patients and different participants had taken different measures, pairwise deletion was utilized in the regression model. Although statistically this approach is acceptable, the product can resemble incomplete correlations that may not fully represent the data in its entirety.

Additionally, a few of the predictor variables included in the regression analysis had very low variance in scores, especially age at onset and length of follow-up, leading to more skewed and leptokurtic distributions for these factors. Although conceptually this small variability is consistent with expectancies for this population, it brings into question whether the data appropriately fulfilled assumption criteria for a regression analysis. Data transformations were considered; however, none proved useful in creating a more normal distribution of the data. As with all studies, caution should be exercised in interpreting results.

### *Implications*

#### *Research*

Research examining familial influences of post-treatment outcomes is currently in its earliest stages. There is still much to be done as findings are preliminary and not yet confirmed across studies. Current findings indicate that a significant discrepancy exists between patients' perception of their fathers and mothers, and that these differences may lead to predictable outcomes (Castro, et al., 2000). These discrepancies warrant further investigation. Additionally, further exploration into change variables should be conducted to determine if changes in other relational perceptions have any predictability over outcomes. The incongruence of the daughter's

perception of acceptance by her father with her discontent in the paternal relationship should continue to be examined. Finally, other measures of outcome should be included to distinguish between different types of responses to treatment.

### *Treatment*

The current findings suggest that a familial focus in treatment should continue to be encouraged, for patients across all eating disorder diagnoses. Rather than emphasize the probability that families contributed to the development of the disorder, it may be more beneficial to the population to emphasize how patients' perceptions of their parents may play a part in recovery.

### *Conclusions*

This study contributes to the current literature connecting familial relationships with eating disorder symptomology. The present investigation demonstrates that parental relationships predict recovery, even after taking into account classical predictors of outcome. The daughter's relationship with the father is especially relevant, as her perceived paternal childhood acceptance and her candor regarding current dissatisfaction with her father as reported at intake, are associated with improved outcomes. On the other hand, although symptoms and perception of parents may significantly change from admission to discharge, these changes do not seem to predict post-treatment symptoms. Rather, it is only the perceptions at admission that influence healing. This second finding is an unfortunate one; however, for it may reaffirm the notion that outcome is determined less by treatment variables and more by the patients' presentation at intake. This outlook again leaves clinicians, families, friends, and patients with little hope over having an impact on recovery. Ultimately, a connection between the familial relationships and



outcomes has been confirmed; however, further investigation is necessitated to verify more specifically just how perceptions of the family influence recovery.

## References

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (4th edition)*. Washington, DC: American Psychiatric Association.
- Anderson, A. E. (1985). Anorexia nervosa: The role of divorce. *BASH Newsletter*, 4, 9–10.
- Armstrong, J., & Roth, D. M. (1989). Attachment and separation difficulties in eating disorders: A preliminary investigation. *International Journal of Eating Disorders*, 8, 141–155.
- Arikian, A., Keel, P. K., Miller, K. B., Thuras, P., Mitchell, J. E., & Crow, S. J. (2008). Parental psychopathology as a predictor of long-term outcome in bulimia nervosa patients. *Eating Disorders*, 16, 20–29.
- Aron, A., & Aron, E. N. (1999). *Statistics for psychology (2<sup>nd</sup> ed.)*. Upper Saddle River, NJ: Prentice Hall.
- Baell, W. K. & Wertheim, E. H. (1992). Predictors of outcome in the treatment of bulimia nervosa. *British Journal of Clinical Psychology*, 31, 330–332.
- Bell, R. M. (1985). *Holy anorexia*. Chicago: University of Chicago Press.
- Bell, L. (2002). Does concurrent psychopathology at presentation influence response to treatment for bulimia nervosa? *Eating and Weight Disorders*, 7, 168–181.
- Bemporad, J. R. (1996). Self-starvation through the ages: Reflections on the pre-history of anorexia nervosa. *International Journal of Eating Disorders*, 19, 217–237.
- Bizeul, C., Sadowsky, N., & Rigaud, D. (2001). The prognostic value of initial EDI scores in anorexia nervosa patients: A prospective follow-up study of 5-10 years. *European Psychiatry*, 16, 232–238.
- Boskind-Lodahl, M. (1976). Cinderella's stepsisters: A feminist perspective on anorexia nervosa and bulimia. *Signs*, 2, 342–256.

- Botta, R. A. & Dumlao, R. (2002). How do conflict and communication patterns between fathers and daughters contribute to or offset eating disorders? *Health Communication, 14*, 199–219.
- Bowlby, J. (1982). *Attachment and loss: Volume 1 Attachment*. New York: Basis Books.
- Brambilla, F., & Monteleone, P. (2003). Physical complications and physiological aberrations in eating disorders: A review. In M. Maj, K. Halmi, J. J. Lopez-Ibor, & N. Sartorius (Eds.), *Eating Disorders* (pp. 139–192). West Sussex, England: John Wiley & Sons Ltd.
- Brusch, H. (1978). *The golden cage: The enigma of anorexia nervosa*. Cambridge, MA: Harvard University Press.
- Bulik, C. M., Sullivan, P. F., Fear, J. L., & Pickering, A. (2000). Outcome of anorexia nervosa: Eating attitudes, personality, and parental bonding. *International Journal of Eating Disorders, 28*, 139–147.
- Casper, R. C., & Troiani, M. (2001). Family functioning in anorexia nervosa differs by subtype. *International Journal of Eating Disorders, 30*, 338–342.
- Castro, J., Toro, J., & Cruz, M. (2000). Quality of rearing practices as a predictor of short-term outcome in adolescent anorexia nervosa. *Psychological Medicine, 30*, 61–67.
- Center for Change. (2007). *Programs*. Retrieved September 18, 2007.  
<http://www.centerforchange.com/programs/specialized.php>
- Chipley, W. J. (1860). On sitomania. *Journal of Psychological Medicine and Mental Pathology, 13*, 266–270.
- Crichton, P. (1996). Were the Roman emperors Claudius and Vitellius bulimic? *International Journal of Eating Disorders, 19*, 203–207.

- Crow, S. J. & Nyman, J.A. (2004). Cost-effectiveness of anorexia nervosa treatment. *International Journal of Eating Disorders*, 35, 155–160.
- Crowther, J. H., Kichler, J. C., Shewood, N. E., & Kuhnert, M. E. (2002). The role of familial factors in bulimia nervosa. *The Journal of Treatment & Prevention*, 10, 141–151.
- Dallos, R. (2004). Attachment narrative therapy: Integrating ideas from narrative and attachment theory in systemic family therapy with eating disorders. *Journal of Family Therapy*, 26, 40–65.
- Deter, H. & Herzog, W. (1994). Anorexia nervosa in a long-term perspective: Results of the Heidelberg-Mannheim study. *Psychosomatic Medicine*, 56, 20–27.
- Deter, H. C., Schellberg, D., & Kopp, W. (2005). Predictability of a favorable outcome in anorexia nervosa. *European Psychiatry*, 20, 165–172.
- Doerr-Zegers, O., Petrasic, J., & Morales, E. (1985). The role of the family in the pathogenesis of anorexia nervosa. In P. Pichot, P. Berner, R. Wolf, & K. Thau (Eds.), *Psychiatry, the state of the art. Vol. 4. Psychotherapy and psychosomatic medicine* (pp. 459–465). New York: Plenum Press.
- Edhouse, H. (1985). The child as the presenting symptom of the mother. Anorexia in a nine and a half year old child. *Australian Family Physician*, 4, 1–6.
- Fairburn, C. G., Kirk, J., O'Connor, M., Anastasiades, P., & Cooper, Z. (1987). Prognostic factors in bulimia nervosa. *British Journal of Clinical Psychology*, 26, 223–224.
- Fichter, M. M. & Quadflieg, N. (2004). Twelve-year course and outcome of bulimia nervosa. *Psychological Medicine*, 34, 1395–1406.
- Finfgeld, D. L. (2002). Anorexia nervosa: Analysis of long-term outcomes and clinical implications. *Archives of Psychiatric Nursing*, 16, 176–186.

- Fisher, M., Pastore, D., Schneider, M., Pegler, C., & Napolitano, B. (1994). Eating attitudes in urban and suburban adolescents. *International Journal of Eating Disorders, 16*, 67–74.
- Fonseca, H., Ireland, M., & Resnick, M. D. (2002). Familial correlates of extreme weight control behaviors among adolescents. *International Journal of Eating Disorders, 32*, 441–448.
- Frazier, S. H., Faubion, M. H., Giffin, M. E., & Johnson, A. M. (1955). A specific factor in symptom choice. *Staff Meetings of the Mayo Clinic, 30*, 227–2423.
- Frisch, M. J., Herzog, D. B., & Franko, D. L. (2006). Residential treatment for eating disorders. *International Journal of Eating Disorders, 39*, 434–442.
- Garner D. M., & Garfinkel, P. E. (1979). The eating attitudes test: An index of the symptoms of anorexia nervosa. *Psychological Medicine, 9*, 273–279.
- Gershon, E. S., Schreiber, J. L., Hamont, J. R., Dibble, E. D., Kaye, W., Nurnberger, J. L., et al. (1983). Anorexia nervosa and major affective disorders associated in families: A preliminary report. In S. B. Guze, F. J. Earls, & J. E. Barret (Eds), *Childhood psychopathology and development* (pp. 270–284). New York: Raven Press.
- Gull, W. W. (1874). Anorexia nervosa (apepsia hysterical, anorexia hysteria). *Transactions of the Clinical Society of London, 7*, 22–28.
- Harris, E. C., & Barraclough, B. (1998). Excess mortality of mental disorder. *British Journal of Psychiatry, 173*, 11–53.
- 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*, 829–837.

- Herzog, W., Kronmuller, K., Hartmann, M., Bergmann, G., & Kroger, F. (2000). Family perception of interpersonal behavior as a predictor in eating disorders: A prospective, six-year follow-up study. *Family Process, 39*, 359–374.
- Herzog, W., Schellberg, D., & Deter, H. (1997). *Journal of Consulting and Clinical Psychology, 65*, 169–177.
- Hesse-Biber, S., & Marino, M. (1991). From high school to college: Changes in women's self-concept and its relationship to eating problems. *Journal of Psychology: Interdisciplinary and Applied, 125*, 199–216.
- Hill, A. J., & Franklin, J. A. (1998). Mothers and daughters and dieting: Investigating the transmission of weight control. *Journal of Clinical Psychology, 37*, 3–13.
- Hoek, H. W., van Hoeken, D., & Katzman, M. A. (2003). Epidemiology and cultural aspects of eating disorders: A review. In M. Maj, K. Halmi, J. J. Lopez-Ibor, & N. Sartorius (Eds.), *Eating disorders* (pp. 75–104). West Sussex, England: John Wiley & Sons Ltd.
- Hsu, L. K. G. (1995). Outcome of bulimia nervosa. In K. D. Brownell & C. G. Fairburn (Eds.), *Eating disorders and obesity: A comprehensive handbook* (pp. 336–368). New York: Guilford Press
- Hudson, W. W. (1982). *The clinical measurement package: A field manual*. Chicago: Dorsey Press.
- Humphrey, L. L. (1986). Family relations in bulimic-anorexic and nondistressed families. *International Journal of Eating Disorders, 5*, 223–232.
- Humphrey, L. L. (1988). Observed family interactions among subtypes of eating disorders using structural analysis of social behavior. *Journal of Consulting and Clinical Psychology, 57*, 206–214.

- Humphrey, L. L. (1989). Observed family interactions among subtypes of anorexic, bulimic, and normal families. *Journal of the American Academy of Child and Adolescent Psychiatry*, 27, 544–551.
- Humphrey, L. L., Apple, R. F., & Kirschenbaum, D. S. (1986). Differentiating bulimic-anorexic from normal families using interpersonal and behavioral observational systems. *Journal of Consulting and Clinical Psychology*, 54, 190–195.
- Kadera, S. W., Lambert, M. J., & Andrews, A. (1996). How much therapy is really enough? A session-by-session analysis of the psychotherapy dose-effect relationship. *Journal of Psychotherapy Practice & Research*, 5, 132–151.
- Keel, P. K. (2005). *Eating disorders*. Upper Saddle River, New Jersey: Pearson Education.
- Keel, P. K., & Klump, K. L. (2003). Are eating disorders culture-bound syndromes? Implications for conceptualizing their etiology. *Psychological Bulletin*, 129, 747–769.
- Keel, P. K., & Mitchell, J. E. (1997). Outcome in bulimia nervosa. *American Journal of Psychiatry*, 154(3), 313–321.
- Kinoy, B. P. (2001). *Eating disorders: New directions in treatment and recovery*, 2<sup>nd</sup> edition. New York, New York: Columbia University Press.
- Kog, E., & Vandereycken, W. (1989a). The speculations: An overview of theories about eating disorder families. In W. Vandereycken, E. Kog, & J. Vanderlinden (Eds.), *The family approach to eating disorders* (pp. 7–24). New York: PMA Publishing Corp.
- Kog, E., & Vandereycken, W. (1989b). The facts: A review of research data on eating disorder families. In W. Vandereycken, E. Kog, & J. Vanderlinden (Eds.), *The family approach to eating disorders* (pp. 25–67). New York: PMA Publishing Corp.

- Krener, P. G., Abramowitz, S. I., & Walker, P. B. (1986). Relation of family factor to treatment outcome for bulimic patients. *Psychotherapy and Psychosomatics*, *45*, 127–132.
- Lambert, M. J., Hansen, N. B., Umphress, V., Lunnen, K., Okiishi, J., Burlingame, G. M., Heufner, J. C., & Reisinger, C. R. (1996). Administration and scoring manual for the Outcome Questionnaire (OQ-45.2). Wilmington, DE: American Professional Credentialing Services LLC.
- Lasègue, E. C. (1873). De l'anorexie hystérique. *Archives Générales de Médecine*, *21*, 385–403.
- Latzer, Y., Hochdorf, Z., Bachar, E., & Canetti, L. (2002). Attachment style and family functioning as discriminating factors in eating disorders. *Contemporary Family Therapy: An International Journal*, *24*, 581–599.
- Lee, S., Ho, T. P., & Hsu, L. K. (1993). Fat phobic and non-fat phobic anorexia nervosa: A comparative study of 70 Chinese patients in Hong Kong. *Psychological Medicine*, *23*, 999–1017.
- McGrane, D. & Carr, A. (2002). Young women at risk for eating disorders: Perceived family dysfunction and parental psychological problems. *Contemporary Family Therapy: An International Journal*, *24*, 385–395.
- Mertler, C. A., & Vannatta, R. A. (2005). *Advanced and multivariate statistical method (3<sup>rd</sup> Ed.)*. Glendale, CA: Pyrczak Publishing.
- Minuchin, S., Rosman, B. L., & Baker, L. (1978). *Psychosomatic families: Anorexia nervosa in context*. Cambridge: Harvard University Press.
- Monteleone, P., Treasure, J., Santonastaso, P., Favaro, A., & Brambilla, F. (2005). Families of people with an eating disorder. In N. Sartorius (Ed.), *Families and mental disorders: From burden to empowerment* (pp. 113–125). New York, NY: John Wiley & Sons Ltd.



- National Eating Disorders Association. (2006). General eating disorders information. Retrieved September 18, 2007. <http://www.nationaleatingdisorders.org>
- Polivy, J. & Herman, C. P. (2002). Causes of eating disorders. *Annual Review of Psychology*, 53, 187–213.
- Pomeroy, C. (2001). Medical evaluation and medical management. In J. E. Mitchell (Ed.), *The outpatient treatment of eating disorders: A guide for therapists, dieticians and physicians* (pp. 306–318). Minneapolis: University of Minnesota Press.
- Richards, P. S., Baldwin, B. M., Frost, H. A., Clark-Sly, J. B., Berrett, M. E., & Hardman, R. K. (2000). What works for treatment eating disorders? Conclusions of 28 outcome reviews. *Eating Disorders* 8, 189–206.
- Rodriguez, M., Perez, V., & Garcia, Y. (2005). Impact of traumatic experiences and violent acts upon response to treatment of a sample of Colombian women with eating disorders. *International Journal of Eating Disorders*, 37, 299–306.
- Rohner, R., Saavedra, J., & Granum, E. (1978). Development and validation of the parental acceptance-rejection questionnaire: Test manual. JSAS: *Catalog of Selected Documents in Psychology* (No. 1635).
- Rorty, M., Yager, J., & Rossotto, E. (2000). Parental intrusiveness in adolescence recalled by women with a history of bulimia nervosa and comparison women. *International Journal of Eating Disorders*, 28, 202–208.
- Santonastaso, P., Saccon, D., & Favaro, A. (1997). Burden and psychiatric symptoms on key relatives of patients with eating disorders: A preliminary study. *Eating and Weight Disorders*, 1, 44–48.
- Selvini-Palazzoli, M. P. (1974). *Self-starvation*. London: Chaucer.

- Shoebridge, P. J., & Gowers, S. G. (2000). Parental high concern and adolescent-onset anorexia nervosa: A case-control study to investigate direction of causality. *British Journal of Psychiatry, 176*, 132–137.
- Sights, J. R., & Richards, H. C. (1984). Parents of bulimic women. *International Journal of Eating Disorders, 3*, 3–13.
- Smith, M. H. (2006). Perceptions of parents, self, and god as predictive of symptom severity among women beginning inpatient treatment for eating disorders (Doctoral dissertation, Brigham Young University). *Dissertation Abstracts International, 67*, 562–675.
- Smolak, L., Levin, M. P., & Schermer, F. (1999). Parental input and weight concerns among elementary school children. *International Journal of Eating Disorders, 25*, 263–271.
- Smolak, L., & Murnen, S. K. (2002). A meta-analytic examination of the relationship between child sexual abuse and eating disorders. *International Journal of Eating Disorders, 31*, 136–150.
- Steiger, H. & Stotland, S. (1996). Prospective study of outcome in bulimics as a function of Axis-II comorbidity: Long-term responses on eating and psychiatric symptoms. *International Journal of Eating Disorders, 20*, 149–161.
- Steinhausen, H. (2002). The outcome of anorexia nervosa in the 20<sup>th</sup> century. *American Journal of Psychiatry, 159*, 1284–1293.
- Steinhausen, H., Rauss-Mason, C., & Seidel, R. (1991). *Psychological Medicine, 21*, 447–454.
- Striegel-Moore, R. H., Leslie, D., Petrill, S. A., Garvin, V., Rosenheck, R. A. (2000). One-year use and cost of inpatient and outpatient services among female and male patients with an eating disorder: Evidence from a national database of health insurance claims. *International Journal of Eating Disorders, 27*, 381–389.

- Strober, M., Bowen, E., & Preble, J. (1985). Predictors of weight change in juvenile anorexia nervosa. *International Journal of Eating Disorders, 4*, 605–608.
- Strober, M., Freeman, R., Morrell, W. (1997). The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse, and outcome predictors over 10–15 years in a prospective study. *International Journal of Eating Disorders, 22*, 339–360.
- Sugarman, A., & Kurash, C. (1982). The body as a transitional object in bulimia. *International Journal of Eating Disorders, 1*, 57–67.
- Swift, W. J. (1982). The long-term outcome of early onset anorexia nervosa: A critical review. *Journal of American Academy of Child Psychiatry, 21*, 38–46.
- Szyrynski, V. (1973). Anorexia nervosa and psychotherapy. *American Journal of Psychotherapy, 27*, 492–505.
- Tobler, S. B. (2008). Perceptions of parents, peers, romantic partner and God as predictive of symptom severity among women in treatment for eating disorders (Doctoral Dissertation, Brigham Young University). *Dissertation Abstracts International, 68*, 8417–8485.
- Tozzi, F., Sullivan, P. F., Fear, J. L., McKenzie, J., & Bulik, C. M. (2003). Causes and recovery in anorexia nervosa: The patient's perspective. *International Journal of Eating Disorders, 33*, 143–154.
- Treasure, J., Murphy, T., Todd, G., Gavan, K., Joyce, J., & Szmukler, G. (2001). The experience of caregiving for severe mental illness: A comparison between anorexia nervosa and psychosis. *Social Psychiatry & Psychiatric Epidemiology, 36*, 343–347.
- Umphress, V. J., Lambert, M. J., Smart, D. W., Barlow, S. H., & Clouse, G. (1997). Concurrent and construct validity of the outcome questionnaire. *Journal of Psychoeducational Assessment, 15*, 40–55.

- Vandereycken, W., & Meerman, R. (1984). *Anorexia Nervosa: A clinician's guide to treatment*. Berlin, New York: Walter de Gruyter.
- Vidovic, V., Juresa, V., Begovac, I., Mahnik, M., & Tocilj, G. (2005). Perceived family cohesion, adaptability, and communication in eating disorders. *European Eating Disorders, 13*, 19–28.
- Wallin, U., & Kronvill, P. (2002). Anorexia nervosa in teenagers: Change in family function after family therapy, at 2-year follow-up. *Nordic Journal of Psychiatry, 56*, 363–369.
- Ward, A., Ramsay, R., Turnbull, S., Benedettini, M., Treasure, J. (2000). Attachment patterns in eating disorders: Past in the present. *International Journal of Eating Disorders, 28*, 370–276.
- Wewetzer, C., Deimel, W., Herpertz-Dahlmann, B., & Matthejat, F. (1996). Follow-up investigation of family relations in patients with anorexia nervosa. *European Child & Adolescent Psychiatry, 5*, 18–24.
- White, M. (1983). Anorexia nervosa: A transgenerational system perspective. *Family Process, 22*, 255–275.
- Whitney, J., & Eisler, I. Theoretical and empirical models around caring for someone with an eating disorder: The reorganization of family life and interpersonal maintenance factors. *Journal of Mental Health, 14*, 575–585.
- Wilson, G. T., & Fairburn, C. G. (1993). Cognitive treatments for eating disorders. *Journal of Consulting and Clinical Psychology, 61*, 261–269.

- Wisotsky, W., Dancyger, I., Fornari, V., Swencionis, C., Fisher, M., Schneider, M., & Wisotsky, W. (2006). Is perceived family dysfunction related to comorbid psychopathology? A study at an eating disorder day treatment program. *International Journal of Adolescent Medicine and Health, 18*, 235–244.
- Wonderlich, S. A., Brewerton, T. D., Jolic, Z., Dansky, B. S., & Abbott, D. W. (1997). Relationship of childhood sexual abuse and eating disorders. *Journal of the Academy for Child and Adolescent Psychiatry, 36*, 1107–1115.
- World Health Organization. (1998). International statistical classification of diseases and related health problems. (10<sup>th</sup> revision. Ed.). Geneva: World Health Organization.
- Yager, J. (1989). Psychological treatment for eating disorders. *Psychiatric Annals, 19*, 477–482.

## Appendix

*Center for Change Long-term Follow-up Phone Survey*

1. Now that you've been out of treatment for some time, how much do you feel like the treatment program at the Center for Change helped you overall?

5                      4                      3                      2                      1  
 very much      much                      some                      very little      none

2. To what extent would you say you've recovered from your eating disorder?

\_\_\_ completely recovered    \_\_\_ mostly recovered    \_\_\_ partly recovered    \_\_\_ not recovered at all

3. During the past month, how frequently have you binged?

\_\_\_ never    \_\_\_ rarely    \_\_\_ about once a week    \_\_\_ several times a week    \_\_\_ several times a day

4. During the past month, how frequently have you purged?

\_\_\_ never    \_\_\_ rarely    \_\_\_ about once a week    \_\_\_ several times a week    \_\_\_ several times a day

5. During the past month, how frequently have you skipped or restricted meals to lose or maintain weight?

\_\_\_ never    \_\_\_ rarely    \_\_\_ about once a week    \_\_\_ several times a week    \_\_\_ several times a day

6. During the past month, how frequently have you used laxatives to control your weight?

\_\_\_ never    \_\_\_ rarely    \_\_\_ about once a week    \_\_\_ several times a week    \_\_\_ several times a day

7. How much depression (i.e. feeling sad, discouraged, or blue) have you experienced the past month?

\_\_\_ very much                      \_\_\_ much                      \_\_\_ some                      \_\_\_ a little                      \_\_\_ none

8. How much anxiety (i.e. feeling worried, nervous, or frightened) have you experienced the past month?

\_\_\_ very much                      \_\_\_ much                      \_\_\_ some                      \_\_\_ a little                      \_\_\_ none

## Appendix continued

9. How much conflict and problems have you experienced in your relationships this past month?

\_\_\_ very much      \_\_\_ much      \_\_\_ some      \_\_\_ a little      \_\_\_ none

10. How much difficulty have you had fulfilling your social responsibilities (e.g., at work, home, church) the past month?

\_\_\_ very much      \_\_\_ much      \_\_\_ some      \_\_\_ a little      \_\_\_ none

11. How have you felt about your relationship with God or your Higher Power the past month?

\_\_\_ very much      \_\_\_ much      \_\_\_ some      \_\_\_ a little      \_\_\_ none

12. In general, how happy and satisfied with your life have you felt since leaving the Center for Change?

\_\_\_ very much      \_\_\_ much      \_\_\_ some      \_\_\_ a little      \_\_\_ none