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# Modeling Treatment Outcomes in Eating Disorders

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# Modeling Treatment Outcomes in Eating Disorders

by

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A Dissertation submitted in partial satisfaction of
the requirements for the degree of
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Each person whose signature appears below certifies that this dissertation in his/her opinion is adequate, in scope and quality, as a dissertation for the degree Doctor of Philosophy.

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CONTENT

FIGURES

Figures Page

TABLES

ABBREVIATIONS

| | |
|---|---|
| 5-HT | Serotonin (5-hydroxytryptamine) |
| 5-HT1A | Serotonin receptor |
| 5-HT1B | Serotonin receptor |
| 5-HT2A | Serotonin receptor |
| AN | Anorexia Nervosa |
| ANCOVA | Analysis of Covariance |
| ANOVA | Analysis of Variance |
| APA | American Psychiatric Association |
| BMI | Body Mass Index |
| BN | Bulimia Nervosa |
| CBT | Cognitive Behavioral Therapy |
| DA | Dopamine |
| D2 | Dopamine receptor |
| D3 | Dopamine receptor |
| DBT | Dialectical Behavioral Therapy |
| DSM-IV-TR | Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision |
| eCBT | Enhanced Cognitive Behavioral Therapy |
| EDA | Eating Disorders Anonymous |
| EDE | Eating Disorders Examination |
| EDI | Eating Disorder Inventory |
| EDI-2 | Eating Disorder Inventory-2 |
| EDI-3 | Eating Disorder Inventory-3 |

| | |
|---|---|
| EDO | Eating Disorder |
| FDA | U.S. Food and Drug Administration |
| fMRI | Functional Magnetic Resonance Imaging |
| GABA | Gamma-Aminobutyric Acid |
| GPMC | Global Psychological Maladjustment Composite |
| HLM | Hierarchical Linear Modeling |
| HPA axis | Hypothalamic-Pituitary-Adrenal axis |
| ICAT | Integrative Cognitive-Affective Therapy |
| IPT | Interpersonal Therapy |
| NICE | National Institute for Clinical Excellence |
| NOS | Not Otherwise Specified |
| OA | Overeaters Anonymous |
| OQ | Outcome Questionnaire |
| OQ-45 | Outcome Questionnaire-45 |
| OQ-A | Outcome Questionnaire Analyst |
| OCD | Obsessive-Compulsive Disorder |
| PET | Positron Emission Tomography |
| PHP | Partial Hospital Program |
| PTSD | Post-Traumatic Stress Disorder |
| SCT | Social Cognitive Theory |
| SD | Standard Deviation |
| SessNum | Session Number |
| SIPN | Social Information Processing Network |

| | |
|---|---|
| SPECT | Single Photon Emission Computed Tomography |
| SSRIs | Selective Serotonin Reuptake Inhibitors |
| THC | Tetrahydrocannabinol |

ABSTRACT OF THE DISSERTATION

Modeling Treatment Outcomes in Eating Disorders:
Does Therapist Feedback Support Individually Tailored Service Allocation?

by

Kathryn Grace Truitt

Doctor of Philosophy, Graduate Program in Clinical Psychology
Loma Linda University, March 2011
David A. Vermeersch, Chairperson

Eating disorders are notoriously difficult and costly to treat, with only 40% of individuals with an eating disorder making a full recovery. Individually Tailored Service Allocation provides a dynamic treatment model defined by empirically accepted theory and consistently informed by data provided by the patient. The use of patient feedback allows for the tailoring of individual treatment plans to meet the unique and varied needs of each patient. Hierarchical Linear Modeling was used to examine the effect of Individually Tailored Service Allocation on eating disorder treatment outcomes. A total of 51 adult women meeting diagnostic criteria for an eating disorder participated in this study. Participants were randomly assigned to treatment as usual or individually tailored treatment groups. Changes in psychological dysfunction and distress were measured bi-weekly throughout the course of treatment using the Outcome Questionnaire 45. The results of this study indicate variability in levels of global psychological dysfunction (both within and between subjects) throughout the course of treatment appear to be the norm, rather than an exception, and this variability is related to eating disorder treatment outcomes. The choice of treatment methodology and level of Individually Tailored Service Allocation has the ability to drastically shift treatment outcomes.

## Introduction

*"Eating disorders are one the most troubling behavioral disorders in our society. Eating disorders rob girls and young women of their future and, not uncommonly, their lives."* (Park, 2007)

The psychological, physical, and social costs of an eating disorder are extremely high (Levine & Smolak, 2005). Anorexia nervosa has a projected mortality rate of 5% (Birmingham, Su, Hlynsky, Goldner, & Gao, 2005), and lifetime prevalence for bulimia nervosa is estimated to be 5% for females (Lewinsohn, Striegel-Moore, & Seeley, 2000). Individuals suffering from an eating disorder have a suicide risk that is 50 times higher than that of the general population (Park, 2007). Treatment is often costly and slow, with estimates for length of recovery ranging from 57 to 79 months (APA Work Group on Eating Disorders, 2000). Only 40% of individuals diagnosed with an eating disorder, even with treatment, will recover, with 20% obtaining partial recovery, experiencing no recovery, or dying. Even more disconcerting, only 1 in 10 males or females suffering from an eating disorder will get treatment for it. Only 35% of those 1 in 10 individuals will receive treatment at a facility that specializes in treating eating disorders (Noordenbox, 2002).

Three primary eating disorders are recognized diagnostically: anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified (NOS). Of these three diagnoses, there are 10 subtypes of eating disorders, none of which are a completely separate diagnostic and treatable entity from the others (Wonderlich, Joiner, Keel,

Williamson, & Crosby, 2007). To further complicate the presentation of an individual with an eating disorder, such individuals typically present with both Axis I and Axis II psychiatric disorders, including anxiety, depression, body dysmorphic disorder, obsessive compulsive disorder (OCD), post-traumatic stress disorder (PTSD), and substance abuse disorders (Costin, 1999; Kaye, Bulik, Thorton, Barbarich, & Masters, 2004). Eating disorders are also typically accompanied by neurological and cardiovascular difficulties (Chavez & Insel, 2007). Despite hundreds of research investigations examining etiology, comorbidities, correlating factors, neurological presentations, treatment, and outcome results, there are still more questions than answers about eating disorders.

Eating disorders are particularly troubling due to their chronic state and nebulous psychological presentation. There are no commonly accepted and empirically proven psychotherapeutic interventions for the treatment of either anorexia or bulimia. There is a dearth of objective research studies examining the effectiveness of anorexia treatments. In the few studies that do try to examine anorexia treatments effectively, no single treatment paradigm has demonstrated a clear advantage over the others (Chavez & Insel, 2007). Research on anorexia faces many large hurdles impeding the development and implementation of evidence-based treatments.

The study of the treatment of bulimia has, fortunately, better results. Fluoxetine, a serotonin reuptake inhibitor, has shown promising results in reducing binge/purge behaviors, improving food and eating-related attitudes, as well as reducing the rate of short-term relapse (Beaumont, Russell, Touyz, Buckley, Lowinger, et al., 1997; Goldstein, Wilson, Thompson, Potvin, & Rampey, 1995; Romano, Halmi, Sarkar, Koke, & Lee, 2002). Fluoxetine is currently the only Food and Drug Administration-approved

treatment for any eating disorder. A variety of psychotherapeutic interventions have been examined for effectiveness in the treatment of bulimia, including cognitive behavioral therapy (CBT), interpersonal therapy (IPT), and dialectical behavioral therapy (DBT), amongst others. CBT is currently considered the most effective form of treatment for bulimia, preferably alongside the use of an antidepressant (Walsh, Fairburn, Mickley, Sysko, & Parides, 2004). Unfortunately, much like in the treatment of anorexia, there are still many more treatment nonresponders than responders, indicating a continued need for further research into treatments, comorbid factors and diagnoses, and their etiology. Additionally, although numerous eating disorder interventions have been evaluated, the majority have only achieved modest success. A recent meta-analysis conducted by Stice et al. (2007) reported that out of 51 eating disorder treatment programs, only 9 served to reduce risk factors and/or the symptoms of eating disorders and have these changes still be present at follow-up.

Almost all treatment methodologies for eating disorders are taken from treatment protocols for other disorders. In large part this is because so little is understood about the underlying neurological and pathophysiological presentations of eating disorders. Understanding an eating disorder is difficult, if not impossible, without a comprehensive picture of the role of interpersonal, intrapersonal, sociological, cultural, and physiological factors in the disorder. While large amounts of research investigating food- and body-related factors have been conducted, it was not until very recently that the pathophysiology of eating disorders was considered (Chavez & Insel, 2007).

Recent investigations into the neurological underpinnings of mental disorders have illuminated abnormal activity in the central nervous system, encouraging some

scientists to claim that a mental illness may, in fact, be a brain disorder. Investigators have begun to supplement the observational and behavioral tools of psychology with the tools more commonly reserved for medical illness, i.e., positron emission tomography (PET) scans, functional magnetic resonance imaging (fMRI), and single photon emission computed tomography (SPECT), amongst others. It is hoped that insights provided by these modern tools of neuroscience will provide more information on how to effectively treat eating disorders.

Modern advances in our understanding of these illnesses give us the opportunity to re-evaluate the etiology of this disease. For instance, weight loss and binge/purge cycles are now felt to be the outward manifestation of more significant underlying psychological illness. Although these are the features still used to identify and diagnose the disorders based on Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR) categories, there is the possibility that the grouping of symptoms into classification lists by behavior is overly simplified. Numerous studies have elucidated many other factors in eating disorders, including not only biological factors such as genetic predisposition and neurological dysfunction, but also personality characteristics, abuse histories, traumatic incidents, and developmental challenges (Anderluh et al., 2003; Berretinni, 2004; Fairburn & Harrison, 2003; Keel & Klump, 2003). These different etiological factors are readily apparent in the manifestation of eating disorder symptomology.

While each patient presents with similar symptoms causing him or her to be recognized as having an eating disorder, there can be different underlying causes of the eating disorder. For instance, patients may present with childhood abuse/PTSD, OCD

features, depression, or anxiety as the key contributing factor. As these patients receive treatment, therapy is often tailored to assist the patient in resolving these particular issues as well as address more generalized symptoms such as body image and nutrition. Due to the large variety of underlying factors resulting in an eating disorder, it may be necessary to explore response to treatment by gauging underlying etiologic patterns rather than seeking a single therapeutic technique to treat all patients. Tailoring therapy to the unique etiology of each patient may prove to be the most efficacious form of treatment for eating disorders. Ultimately, as stated by de la Rie, Noordenbos, Donker, and Furth (2006), "Evidence based clinical practice regarding treatment for any eating disorder should be founded on research on the efficacy, effectiveness, and efficiency of different treatment options as well as the clinical and physical circumstances and the patient's preferences."

The current diagnostic categories for eating disorders do not take into consideration the variety of symptom presentations and possible etiologic patterns of each individual. There are two primary diagnostic systems used throughout the world, the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed; DSM-IV; American Psychiatric Association, 1994) and the *International Statistical Classification of Diseases and Related Health Problems* (10th revision; ICD-10; World Health Organization, 1992). Despite the careful consideration and review that both manuals undergo before publication, there is rising speculation that the real-world applicability and validity of the DSM-IV categories are limited (Wonderlich, Joiner, Keel, Williamson, & Crosby, 2007). In particular, the diagnostic categories of eating disorders are best interpreted as diagnostic constructs that are open to change and are easily falsifiable (Skinner, 1986), instead of distinct disease entities.

## Proposed Etiology of Eating Disorders

The etiology of eating disorders is virtually unknown. Numerous possibilities have been suggested, such as genetic predisposition, cultural influences, neurotransmitter imbalances, temperament, and familial influences (Smolak & Levine, 2006). Explanatory theories for the etiology of eating disorders are widespread and just as varied. As Stiegler (2007) claimed, "Arguably, eating disorders have inspired more shifts in explanatory paradigms over a shorter span of time than have any other class of syndromes in the history of psychiatry." Anorexia was initially considered to be the result of the "refrigerator mom" who drove her children into food refusal as their only means of self-assertion. Bulimia was initially seen as a "protest" against parental hostility or even cruelty that would lead afflicted girls to pursue unmet needs through rampant binging followed by a rejection of what they were given with compensatory purging (Stiegler, 2007). Throughout the 1980s and 1990s, the paradigm moved to one of conceptualizing eating disorders as culture-bound syndromes, driven by western society's over-emphasis on how one looks, how one behaves, and the overwhelming media presence. Again, new research arose that found these theories lacking. Incidents of eating disorders began to appear in cultures never touched by western societies, and historical investigations found eating disorders in cultures not subject to modern mores or media. New theories seek to link anorexia and bulimia to abnormalities in the brain and genetic causes.

There may be no single explanation for eating disorders. As with their multifaceted presentation, complete with numerous possible comorbidities, manifesting in a variety of cultures, ages, and developmental histories, their theoretical presentation may be equally complex. Eating disorders may be the result of interplay between nature

and nurture. The complexities in etiology only serve to further complicate the healthcare community's attempts to find successful prevention and treatment strategies. Therefore, a thorough investigation of the possible biopsychosocial indicators of eating disorders is necessary.

**Eating Disorder Risk Factors**

Anorexia and bulimia share some general risk factors but are also unique and distinct illnesses. Both eating disorders have gender as their most potent risk factor; being female places one at much higher risk than being male (Southgate, Tchanturia, & Treasure, 2005). Numerous characteristics developed during childhood also place one at higher risk for developing an eating disorder, such as traits on the obsessive compulsive disorder spectrum or a tendency to internalize events. Certain temperamental traits are also highly correlated with eating disorder onset such as compulsivity, characterized by a fear of mistakes, perfectionism or rigidity, and emotionality, characterized by neuroticism and behavioral inhibition (i.e., shyness and social anxiety; Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003). Risk factors unique to each disorder tend to include appetitive behaviors and body weight. Individuals with anorexia tend to have a poor appetite during childhood and a lower than average body weight, with the opposite being true in individuals who have bulimia (Southgate et al., 2005). Individuals with bulimia also tend to have more exposure to adverse events in their lives than do those with anorexia.

Anorexia and bulimia appear to have similar factors that trigger the onset of the illness. There tends to be some sort of nutritional stress, which can be the result of either

a diet, exercise, or an illness that occurs in the context of some distressing life event (Southgate et al., 2007). Maintenance of the disorders occurs through a variety of factors, where the core operating structures appear to be specific to the type of eating disorder developed. According to Schmidt and Treasure (2005), anorexia is maintained through the effect of the illness on the individual's interpersonal relationships as well as the secondary gains resultant from the illness. Fairburn and colleagues (1993) developed a maintenance model of bulimia. They found that self-esteem based on appraisals of weight and shape feeds the inception of the illness. The illness is then maintained through cycles of self-perpetuating behavior (i.e., binge/purge) that are intended to counteract low self-esteem but instead strengthen it. Figure 1 provides a working model, developed by Southgate et al. (2005), of eating disorder development and maintenance.

**Underlying Neurological and Biological Factors Support a Disruption in Collaborative Brain Function**

Anorexia presents as a unique physiological condition, quite different from bulimia. Investigative studies using functional magnetic resonance imaging found neuronal abnormalities that appear to be specific to anorexia. These abnormalities include decreased brain volume (Katzman et al., 1996), diminished cerebral blood flow and metabolism (Delvenne et al., 1995), disturbances in event-related potentials (Bradley et al., 1997), impaired cognitive performance on tests (Green, Elliman, Wakeling, & Rogers, 1996) and altered levels of neurotransmitters (Kaye et al., 1998). These abnormalities suggest a physical neural mechanism underlying anorexia. Interestingly, Uher et al. (2003) found differences in the neural correlations of individuals suffering

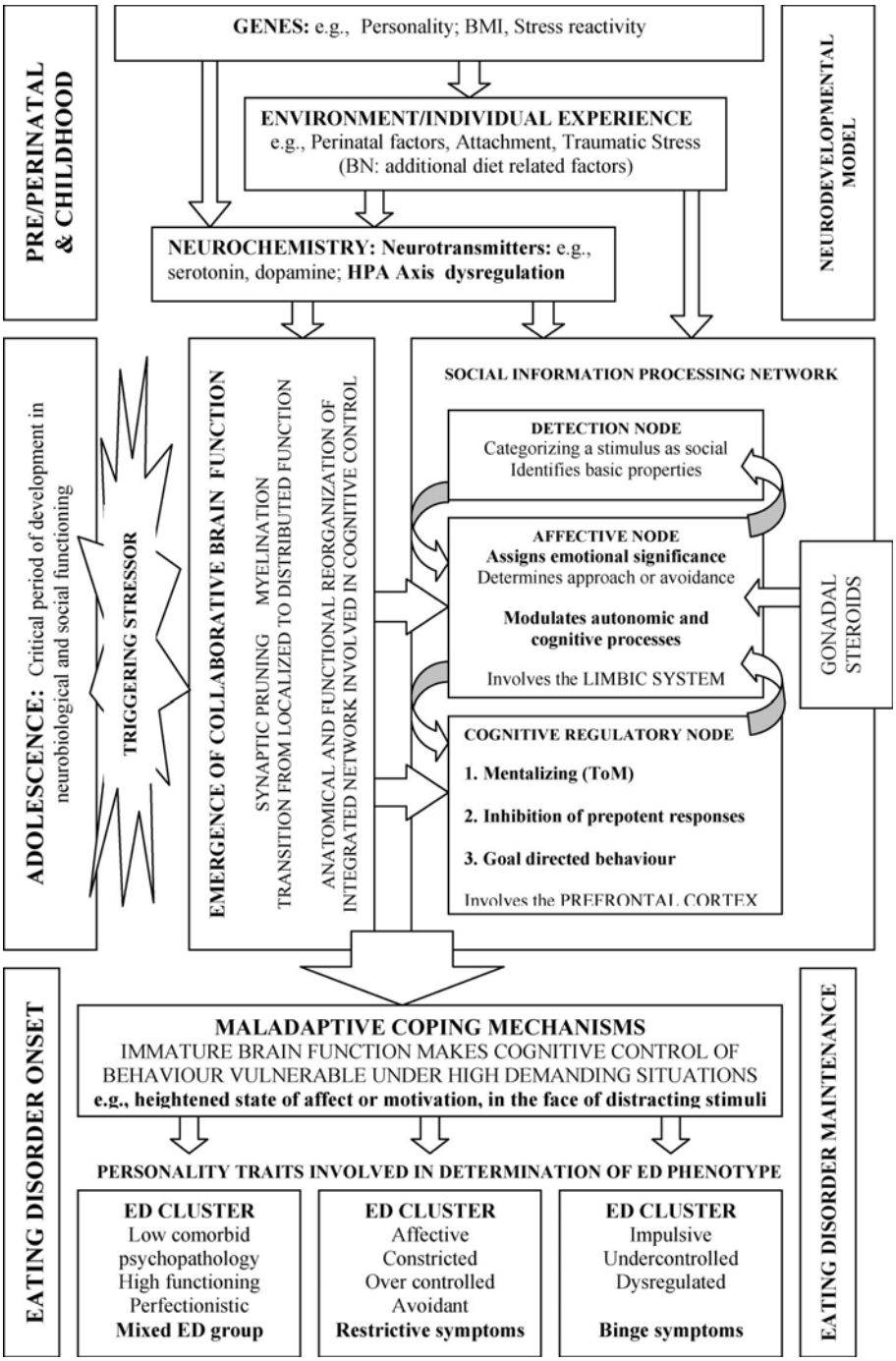


*Figure 1*. Proposed Model of Biopsychosocial Eating Disorder Inception and Maintenance (Southgate et al., 2005).

from chronic anorexia and individuals who had been recovered from anorexia for a minimum of two years. These observed differences may represent changes in brain behavior that occurred with the treatment and recovery process; in particular, they may represent changes in information and/or cognitive processes.

While bulimia does not trend toward the chronic course presented by anorexia, changes in the central nervous system are observed in the disease. For example, during the course of the illness, regional blood flow to the inferior frontal and left temporal cortical areas is elevated (Nozoe et al., 1995). It has been posited that these two areas play a significant role in the pathophysiology of bulimia. Cerebral blood flood is suggested to vary as a function of bulimia's restricting and binge/purge phases (Hirano et al., 1999). Cerebral blood flow correlates with glucose metabolism, which relegates a correlation of alterations in blood glucose levels to bulimia (Fox et al., 1988). Frank et al. (2000) examined the activity of cerebral blood flow in recovered bulimic individuals and found no significant difference from the control subjects. This supports the idea that elevated cerebral blood flow in bulimic individuals is inclusive of their pathophysiology. Upon recovery, cerebral blood flow will return to that of a non-eating disordered individual. This suggests that effective treatment and subsequent recovery should return some of the abnormal brain functions associated with bulimia to a more normalized level.

The unique presentation of these two disorders encourages further investigation into the role of neurotransmitters, cerebral blood flow, and other neurological foundations. Theories incorporating the inner workings of the brain could help to elucidate some of the unknown aspects of these disorders as well as allow for the development of new hypotheses in how to best treat and/or measure eating disorders.

The brain is composed of highly concentrated overlapping neural networks involved in the acts of desiring, seeking out, obtaining, and consuming foods. The ventromedial hypothalamus and nucleus accumbens are both highly implicated in these networks. The nucleus accumbens, in particular, is recognized as the major reward center of the brain. This nucleus is modulated by numerous neurotransmitters including dopamine, glutamate, and opioid neuropeptides (Simansky, 2005). Addiction research shows that stimulating these neural pathways and neurotransmitters may lead to physiological and behavioral pathology often combined with cravings, obsession, and overconsumption.

Dopamine action in the nucleus accumbens is of particular interest in addiction research. Some eating disorder theorists suggest that self-administration of substances and feeding behaviors can be considered in the same category as substance or alcohol abuse due to similarities in animal behavior with self-administration of these substances (Wise, 1997). One study found that both food deprivation (mimicking anorexia) and overeating (mimicking binge-eating) increased dopamine activity in the lateral hypothalamus, which led to increased activation in the nucleus accumbens. This behavioral effect is similar to the activation seen when rats lever-press for electrical stimulation of their medial forebrain bundle, of which both the lateral hypothalamus and nucleus accumbens are a part (Hernandez & Hoebel, 1988). In light of these findings, Davis and Woodside (2002) examined the role of anhedonia in individuals with eating disorders. They reported that anorexic individuals had significantly higher levels of anhedonia compared to bulimic individuals. Anhedonia, a diminished ability to experience pleasure, stems from dopamine interaction with the mesolimbic structures

making up the reward pathway. This finding suggests that the compulsive and addictive nature of anorexia may have roots in decreased dopamine activity in the reward centers of the brain. As previously mentioned, food deprivation does increase levels of dopamine in the medial forebrain bundle, demonstrating that the anorexic may be behaviorally self-medicating for this deficiency.

Some theorists claim that eating-disordered behaviors present with the same characteristics as the auto-addiction opioid theory. This theory, commonly used to describe addiction, proposes that behaviors are undertaken, such as starving or overexercising, to increase the levels of ß-endorphins in the body. These ß-endorphins are biologically identical to exogenous opioids; thus, these behaviors take on an addictive quality due to their ability to stimulate the reward centers in the brain (Marrazzi & Luby, 1986).

As previously mentioned, the brain is composed of a complex network of neurotransmitter pathways with many neurotransmitters interacting at different levels. Serotonin (5-HT) is the primary neurotransmitter implicated in eating disorder pathology. Disturbances in 5-HT levels have long been implicated in eating disorder pathology. 5-HT is implicated in a variety of psychopathologies commonly comorbid to eating disorders. For example, 5-HT has been implicated in personality and temperament traits such as behavioral inhibition, harm avoidance, and borderline personality disorder (Paris, Zweig-Frank, Kin, Schwartz, Steiger, & Nair, 2003). 5-HT is also implicated in numerous psychiatric conditions such as anxiety, fear, obsession (Barr, Goodman, Price, McDougle, & Charney, 1992), and depression (Grahame-Smith, 1992). Similarly,

serotonin (5-HT) disturbances have long been documented in individuals with eating disorders.

Research has found a significant negative correlation between eating-disordered behaviors and 5-HT levels. This supports the idea that eating-disordered behavior serves as a self-medication against high levels of anxiety. Kaye (1999) proposed that this is because the eating-disordered behavior increases the level of 5-HT in an overactive 5-HT system. Recovered bulimic individuals still exhibit disturbances in the 5-HT system, even after long-term recovery (Kaye et al., 1999; Smith et al., 1999). Disturbances in these neurotransmitters, amongst others, may play a large role in the etiology of an eating disorder (Kaye, 1999). Finally, 5-HT plays a role in satiety after food consumption (King, 2006). A recent investigation by Kaye, Frank, Bailer, et al. (2006) reported disturbances in 5-HT1A and 5-HT2A receptors as well as in the 5-HT transporter in anorexic and bulimic individuals. These disturbances are reported over the long term in individuals who were previously ill and recovered from anorexia or bulimia (Kaye et al., 1999; Smith et al., 1999).

5-HT1A is purported to play a role in mood and impulse control, anxiety, feeding behavior, and selective serotonin reuptake inhibitor (SSRI) response. Using positron emission tomography (PET), Kaye et al. (2005) found increased receptor activity of 5-HT1A in prefrontal, medial, and lateral orbital frontal, parietal, lateral temporal, and supra- and pregenual cingulated regions, as well as in the dorsal raphe nuclei in recovered anorexic-bulimic and bulimic individuals. These findings, again, exemplify the long-term changes in 5-HT receptor activity in the brain. Recovered anorexics did not show a significant difference in 5-HT1A receptor activity relative to control subjects, perhaps

providing insight as to why bulimic individuals are responsive to SSRIs but anorexic individuals are not. To date, increased 5-HT1A receptor activity has only been found in individuals exhibiting bulimic symptomology.

The 5-HT2A receptor is implicated in the regulation of mood, anxiety, antidepressant action, and feeding (Barnes & Sharp, 1999). Kaye et al. (2005) reported reduced 5-HT2A receptor activity in the parietal, occipital, and subgenual cortex in both recovered anorexic-bulimics and anorexics. Additionally, recovered anorexics showed reduced 5-HT2A activity in the mesial temporal region and pregenual cingulate. Other investigators reported a significant reduction in 5-HT2A receptor activity in the left frontal cortex, the occipital cortex, and the parietal cortex in ill anorexics (Audenart et al., 2003). Bulimics were found to have normal 5-HT2A activity (Goethals et al., 2004). While these findings are consistent with the speculation of 5-HT2A disturbances in anorexia, the disturbances of 5-HT2A in bulimia are less clear.

Research consistently shows disturbances in 5-HT1A and 5-HT2A receptor activity in recovered eating-disordered individuals. Similarly, the disturbances in dopamine (DA) activity in the brains of eating-disordered individuals are becoming well documented. It is currently unknown whether neurotransmitter disturbances precede the eating disorder or the eating disorder causes the disturbance, but the involvement of these neurotransmitters in the eating disorder is evident. Evidence concerning the effect of disturbances in synaptic pruning in adolescence and the dysregulation in the hypothalamic-pituitary-adrenal axis (HPA axis) further support these hypotheses. Kaye et al. (2005) hypothesized that eating-disordered individuals have a dysregulation in the function of their neural circuitry, most likely relating to disturbances in any number of

the components of the circuit. These disturbances could be in the form of interacting receptors or in the molecules forming the intracellular communication translating the receptor signals. While it is known that receptor activity is a combination of many factors such as neuronal firing, exocytosis, reuptake, and other intracellular mechanisms, the technology to pinpoint exactly where in this process the disturbance lies is still unavailable. Part of the challenge facing the pharmaceutical community in creating effective drugs to treat eating disorders is diagnosing how to best normalize the disruption in neurotransmitter activity.

**Neuropsychological Models of Brain Development and Their Impact on EDOs**

There are currently numerous models of the potential vulnerabilities and risk factors for eating disorders. The focus has recently moved toward the examination of the neurological, genetic, and biological underpinnings of these disorders. Bulimia and anorexia present with different underlying physiological mechanisms. These differences may explain why anorexia and bulimia respond to psychopharmaceutical and therapeutic interventions differently. For example, bulimia is generally more responsive to fluoxetine than anorexia. Anorexia is more likely to be a chronic condition than bulimia. Furthermore, numerous investigations found that individuals suffering from anorexia tend to be repeatedly hospitalized (Keel & Klump, 2003; Milos et al., 2003).

One neurodevelopmental model of eating disorders relies on the HPA axis as a primary mechanism underlying the chronic stress and maladaptive coping strategies seen in individuals with eating disorders (Connan, Campbell, Katzman, Lightman, & Treasure, 2003). This model stresses the importance of genetic factors, childhood experiences, and

the biopsychosocial environment in modifying the HPA axis. These alterations lead to maladaptive cognitive, emotional, and social functioning. The HPA axis, along with its control mechanisms located in the central nervous system, enables the metabolic resources necessary for all of our behaviors (Lovallo & Thomas, 2005). Operating in all stages of life, from sleep to severe stress, the HPA axis is also responsive to our private emotions and thoughts. Research indicates that the HPA axis produces large stress responses when exposed to novel stimuli but that with further exposure, these responses decrease. Interestingly, though, Mason (1968) claimed that the stressfulness of a response is not purely reflexive but is also modified by previous experience and the nature of the environment.

This is important to consider in eating disorders because when an individual is exposed to consistently traumatic experiences, the HPA axis is altered in response to the high levels of glucocorticoids necessary to maintain a state of alertness (i.e., a state of fight or flight). Over time, high levels of glucocorticoids damage the hippocampus, impairing memory formation as well as affecting levels of epinephrine and cortisol. These changes result in a disruption in the ability to maintain normal cognitive functioning (Lovallo & Thomas, 2005). The HPA axis also plays a large role in the regulation of 5-HT neurotransmitters, with chronic stress leading to decreases in hippocampal 5-HT1a and 5-HT1b and an increase in cortical 5-HT2a. These changes, as will be illuminated later, have been noted to be significant in individuals with an eating disorder and are also highly correlated with suicidal behaviors (Lopez, Vazquez, Chalmers, & Watson, 1997).

Adolescence is an extremely important time for brain development. During the

adolescent years, the brain begins the process of synaptic pruning, resulting in the elaborate branching-out of dendrites and increasing levels of myelination. Synaptic pruning is associated with vital refinements to brain systems through increasing efficiency and efficacy by removing redundant neural connections (Southgate et al., 2005). Myelination allows for more rapid communication throughout the brain by speeding up neural transmission. These changes allow for the collaboration of widely dispersed circuitry throughout the brain and the integration of a variety of brain regions, ultimately allowing top-down cognitive control of behavior (Luna & Sweeney, 2004). The enhanced communication between areas of the brain such as the prefrontal cortex, basal ganglia, thalamus, and frontal cortex set the stage for the maturation of the brain in such a way that reflective and inhibitory processes are more consistent and efficient. This stage of brain development in adolescence leads to the development of "collaborative brain function."

Synaptic pruning and the HPA axis are implicated as causal factors in developing a vulnerability to an eating disorder. Southgate and colleagues (2005) proposed that because of the alteration in the HPA axis, leading to a poor coping response to stress, certain individuals are in a persistent state of intense and demanding emotional distress. This state, when it persists through adolescence, interrupts the process of synaptic pruning, thereby disrupting the brain's transition from localized function to collaborative brain function. Secondary effects commonly seen in eating disorders also play a role in disrupting this critical time of brain development. Poor nutrition eventually disturbs the regular maturational processes of the brain and can also disrupt normal hormonal changes. The effects of a lack of complete development of collaborative brain function

can cause specific behaviors to arise that are often noted in an individual with an eating disorder. For example, behavioral focus may be on internal or external stimuli that are immediately gratifying or rewarding but may be harmful in the long run. The lack of integrative brain function inhibits the top-down control of behavior and therefore may lead to the preservation of maladaptive behaviors.

**Role of the Social Information Processing Network in Eating Disorders (Nelson et al., 2005)**

The neurological underpinnings of an eating disorder play an important role in how individuals with anorexia and bulimia interact with and perceive the world around them. Nelson and colleagues (2005) introduced the social information processing network (SIPN) to elucidate the variety of factors interacting to define how an individual experiences the world. The SIPN is composed of cognitive, affective, and detection nodes that mature during the process of synaptic pruning. During adolescence, the SIPN is modulated by gonadal hormones. With the onset of puberty, changes in hormones impact the cells in the affective node, thus affecting the intensity and valence of social stimuli. A hypersensitivity to interpersonal relations is created and can lead to increased emotional responsiveness in scenarios concerning self-esteem, acceptance, rejection, and motivation (Southgate et al., 2005). Research has found eating disorders to often be triggered by interpersonal problems, and it is possible that lack of maturation in the SIPN forms a neural vulnerability in the affective realm that leaves one susceptible to eating-disordered behavior.

Researchers have recently begun investigating executive functioning in individuals with eating disorders. Particular focus has been on inhibitory processing in order to examine the role of impulsivity in individuals with anorexia and bulimia. Congruent with the clinical presentation of individuals with anorexia, restricting subtype, there are increased levels of inhibitory processing. Interestingly, individuals with bulimia and those with anorexia, binge/purge subtype tend to have poorer inhibitory processing, hinting at higher levels of impulsivity (Rubia, Smith, & Taylor, 2005). Numerous other investigations into set-shifting abilities and cognitive flexibility consistently show deficits in overall executive functioning. Individuals with eating disorders appear to be able to inhibit or withhold the activation of new behaviors and may even have difficulty beginning new behaviors but have an extremely difficult time disrupting or stopping ongoing behaviors. From a wider perspective, these disorders can be conceptualized as being composed of a continuous cycle of behaviors that is difficult to break, particularly for individuals who have impediments in executive functioning.

Gillberg and colleagues (1996) suggested that individuals with anorexia struggle with goal-directed behavior. Their research indicated that under certain circumstances, individuals with anorexia will display a cognitive style that ultimately hampers task completion. Goal directedness is characterized by an individual's ability to take a step back from the minute details and see the bigger picture. Individuals with anorexia tend to have superior performance in attending to details and excelling in tasks requiring directed effort than those with bulimia and nonclinical controls. On the other side of this cognitive continuum, individuals with anorexia tend to have very weak performance with respect to incidental learning. Frith (1989) labeled this cognitive style "weak central coherence" in

which these individuals persist on focusing on details even when the instructions of the task at hand call for global information processing. These behaviors are adaptive neither for the task at hand nor for normal, healthy functioning. The cognitive style of an individual with anorexia reflects an overwhelming tendency to use localized rather than distributed information processing networks (Southgate et al., 2005). These findings, again, reflect the importance of synaptic pruning during the adolescent's development as well as the impact of a dysregulated HPA axis.

It has long been accepted that individuals with eating disorders struggle with emotional experiences, expression, and regulation (Kucharska-Pietura, Nickolauo, Marsiak, & Treasure, 2004; Schmidt, Jiwany, & Treasure, 1993; Zonnevylle-Bendek, van Goozen, Cohen-Kettenis, van Elburg, de Windt, & Stevelmans, 2004; Zonnevylle-Bendek, van Goozen, Cohen-Kettenis, van Elburg, & van Engleand, 2002). Individuals with eating disorders also tend to have comorbid alexithymia, suggesting poor emotional intelligence. Furthermore, Friederlich and colleagues (2005) found a disturbance in the emotional processing of pleasant stimuli using a startle eye blink paradigm. Both anorexics and bulimics failed to show the activation in the appetitive-motivational system that control subjects portrayed when shown positive stimuli. Anorexics tend to also have further emotional dysregulation that becomes a pervasive form of anhedonia and disrupts their internal reward system, therefore limiting or completely eliminating their experience of pleasure (Davis & Woodside, 2002).

Animal studies have evidenced that lesions in the ventral striatum, which includes the nucleus accumbens, block response to the startle reflex in positive states (Koch, Schmid, & Schnitzler, 1996), as does destroying dopaminergic neurons in this same

region. This information becomes important when one is considering the role of the dopamine systems in eating disorders. Impaired dopamine function is found in both bulimic and anorexic individuals. Individuals who have recovered from anorexia still display increased D2/D3 receptor binding in the antero-ventral striatum (Frank et al., 2005). These abnormalities may be responsible for the low appetitive drive and general anhedonic response seen in individuals with anorexia. Contrastingly, individuals with bulimia portray reduced D2/D3 binding in the antero-ventral striatum, similar to what is noted in individuals with substance abuse (Wang, Volkow, Logan, et al., 2004). Such neurological deficiencies and cognitive dysfunctions inevitably impact individuals' perception of the world around them.

**Social and Cultural Factors in Eating Disorder Development and Maintenance**

After a review of the literature, Smolak and Levine (2006) reported strong evidence suggesting that weight concerns, dieting, and body dissatisfaction predict the inception of eating pathology. Interestingly, a large meta-analysis performed by Keel and Klump (2003) found evidence suggesting that bulimia is a culture-bound syndrome, while anorexia is not (although cultural influences are found to aid in the maintenance of anorexia). The results of both of these investigations hint at the important role of societal and cultural influences in eating disorders. In light of these findings, it is unsurprising that new hypotheses regarding the roles of societal and cultural factors in eating disorder conceptualization are gaining momentum. Two newer hypotheses, the eating disorder continuum hypothesis and social cognitive theory, offer a viable beginning for the conceptualization of eating disorder etiology.

The eating disorder continuum hypothesis is based on the concept that disturbed eating behaviors are a matter of degree (Scarano & Kalodner-Martin, 1994; Tylka & Subich, 1999). It places unrestrained (or asymptomatic eating) at one end of the continuum and clinical eating disorders (or anorexia/bulimia) at the other end of the continuum. Between the two endpoints lie milder forms of disordered eating. Literature supports the hypothesis that certain characteristics of clinical eating disorders align themselves with the eating disorder continuum hypothesis. Because a majority of women divulge the use of unhealthy eating behaviors and suffer psychological and physiological consequences as a result, numerous clinicians have suggested that eating disorders ought to be conceptualized on a continuum (Scarano & Kalodner-Martin, 1994; Tylka & Subich, 1999). These clinicians note that examining different levels of eating disturbance may illuminate etiological factors involved in the development and treatment of eating disorders (Tylka & Subich, 1999). The eating disorder continuum hypothesis also sheds light on the role of social-cultural factors in eating disorders.

Scarano and Kalodner-Martin (1994) reported that women who exhibit various eating disturbances have similar psychological and behavioral characteristics as individuals with eating disorders, differing only in terms of severity. For example, body dissatisfaction, feeling fat, food preoccupation, weight preoccupation, and the fear of becoming fat increase as an individual's level of disturbed eating increases. Quantitative differences have been similarly noted between subjects occupied with clinical, subclinical, and unrestrained eating in the areas of interoceptive awareness, interpersonal relationships, and feelings of ineffectiveness; difficulties in these arenas increase as disturbed eating behaviors increase. More recently, Tylka and Subich (1999) examined

personality and cognitive facets commonly found in eating disorders along the eating disorder continuum. Their study further highlighted the differences between the asymptomatic, symptomatic, and eating disorder continuum groups, finding a linear relationship amongst the three groups. Similarly, they found a linear relationship with internal dieting locus of control and internalization of the thin-ideal stereotype (Figure 2).

In light of the large influence of society, culture, and the continuum of eating disorder pathology, it is important to examine the role of the environment in the development of an eating disorder. For example, Becker et al. (2002) assessed the effect of the introduction of television on disordered eating behaviors in a Fijian population with no previous media exposure. With the introduction of television to this culture, disordered eating behaviors and attitudes rose significantly. This investigation provides a clear indicator of the negative impact of television, especially in influencing body-image

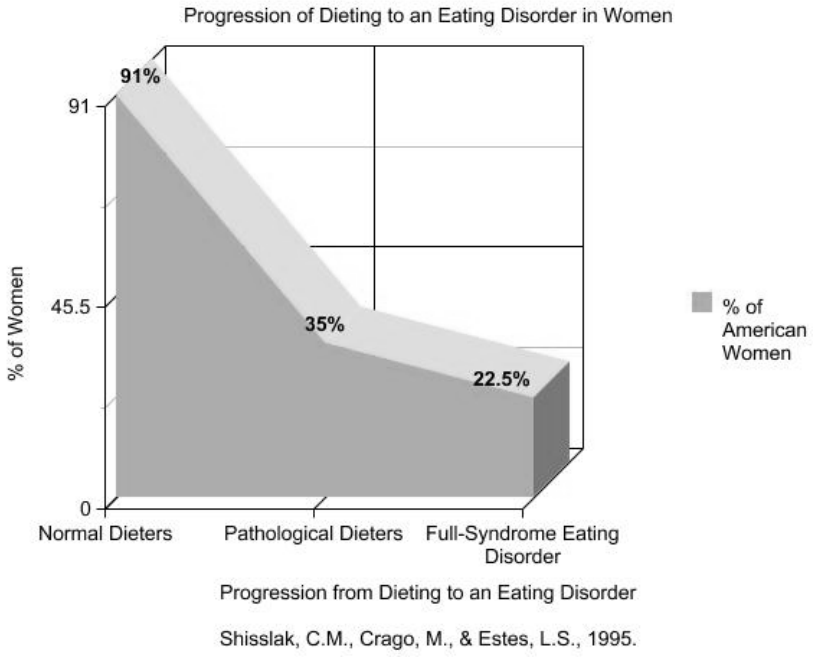


*Figure 2.* Likelihood of a Diet Progressing Into an Eating Disorder.

schemas (Figure 3). Nichter and Nichter (1991) asked adolescent girls to describe their ideal female form. The girls significantly endorsed a female who was 5 ft 7 inches, 100 lbs, and a size 5. The Body Mass Index (BMI) for their idealized female is 15.61, hugely below the recommended minimum weight of 118 lbs (BMI 18.5) for someone who is 5 ft 7 inches. The idealized female described by these adolescents would easily meet criteria for anorexia. Clearly, a normal adolescent girl cannot healthfully fit this idealized model. Yet according to this study, it is one of the main factors in the social status quo among adolescent females.

The discrepancy between girls' true forms and the idealized form may play a significant role in their self-perception (Levine & Smolak, 1998; Smolak & Levine, 1996). The more importance placed on this discrepancy, the larger the tendency toward eating-disordered behavior. Correspondingly, some researchers have proposed that the pressure to be thin influences two variables core to creating eating-disordered behavior: the internalization of the thin ideal and disturbance in body image (Stice, Nemeroff, & Shaw, 1996). Because the standard of thinness promoted by society is impossible for most women to achieve, they are left feeling negative about their own bodies. This negativity results in more body-image disturbance. As Frederickson and Roberts (1997) pointed out, women in America are often socially conditioned to base their sense of self-worth on their appearance. It is now considered normative for women to have a moderate degree of body dissatisfaction and to use diet and exercise to manipulate their weight and body in an attempt to conform to the portrayed media ideal (Gordon, 2000).

An overwhelming amount of evidence suggests that many of the factors involved in the maintenance of an eating disorder are learned behaviors (Smolak & Levine, 2006).

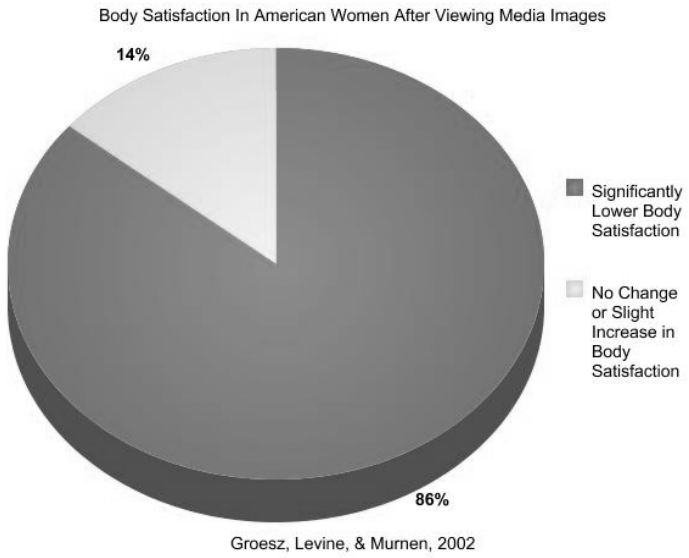


*Figure 3.* Body Satisfaction in American Women After Viewing Media Images.

Because of the apparent role of modeled and learned behaviors in eating disorder pathology, numerous researchers are now ascribing to a sociocultural model of eating-disorder conceptualization based on social cognitive theory (SCT; Bandura, 1986). SCT views behavior as the result of transactions between three factors: (a) an individual's cognitive and emotional processes; (b) patterns of behavior and competencies; and (c) the context or environment. The compilation of these three factors creates the learned behaviors and cognitions through which an individual navigates the world, known as an individual's *schema*. A schema represents the mental structures that help people manage their interactions with the environment in consistent, stable, and meaningful ways (Solso, MacLin, & MacLin, 2005).

Research on eating disorders supports the three factors of SCT in its conceptualization of eating disorder pathology. For example, Frederickson and Roberts

(1997) suggest that women are socialized to equate self-worth with their appearance. This objectification of themselves is the result of psychological variables working in conjunction with sociocultural factors, leading to body shame and body-image disturbance. Similarly, it has been suggested that pressures to be thin are predictive of negative affect and poor social support in women (Maine, 2000; Pipher, 1994; Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999). Research utilizing the eating disorder continuum hypothesis supports these suggestions. Stice et al. (1996) found that pressure to be thin predicts the unique variance in body dissatisfaction, even beyond the variance accounted for in the internalizing of the stereotypical thin ideal.

It is important to note that while the environment and culture may have a strong impact on eating disorder inception and maintenance, they have only an indirect relationship with actual eating disorder psychopathology. The other two factors of SCT, an individual's cognitive and emotional processes and patterns of behavior, also play a key role in an eating disorder's etiology. While each of the three factors of SCT has a role in eating-disordered behavior and pathology, the result is a specific type of schema present in eating-disordered individuals (Levine & Smolak, 2006). This schema, called a *body image schema*, organizes various mechanisms of body image such as shape, weight, appearance, and health (Smolak & Levine, 2006). The body image schema is the result of the interplay of SCT factors, particularly highlighting experiences and cognitions around teasing, mass media, standards of beauty, and the thin ideal in the individual's environment. The prevalence of the expectations and ideals in westernized cultures and societies support the role of the sociocultural model of eating disorders in the eating disorder continuum model hypothesis. The schema is not necessarily applied to every

interaction an individual has, but it is activated in many normal, everyday interactions such as looking in the mirror, meeting someone new for the first time, shopping for new clothes, being presented with certain foods, and/or spending time with friends.

The activation of the body image schema results in internal dialogues involving personal interpretations, thoughts, and conclusions about different interactions and situations (Cash, 1997). For someone with a negative body image schema, this internal dialogue represents a disparity between an investment in the value of a thinner shape or lower weight and an individual's self-perception. This disparity is often seen in individuals who are at risk for disordered eating (Smolak & Levine, 2006)—hence the application of SCT to eating-disorder conceptualization. Ainsworth, Waller, and Kennedy (2002) suggested that bulimic behaviors were often engaged in order to "block" the aversive body image schema. Similarly, Stein and Corte (2003) argued that a disturbed body image motivates eating, body, and weight attitudes characterizing both anorexia and bulimia. They also found that women with a negative body image schema and few positive self-concepts were more vulnerable to societal commentary about food and body concerns as well as the thin ideal. This further supports the strong impact of cultural and societal ideals.

One of the key roles of the body image schema in eating-disorder maintenance is that it creates a cyclical and self-reinforcing thought process (Smolak & Levine, 2006). The bias inherent in the negative body image schema controls individuals' everyday interactions with the world and themselves. Behaviors resulting from the thought processes wrapped up in the schema often lessen the immediate negative affect attached to the schema but have the long-term consequence of further strengthening the bias

(Smolak & Levine, 2006). The result of this cycle is the acquisition of eating-disordered pathology in a previously vulnerable individual or the worsening of pathology in an already-afflicted individual.

Tylka and Subich (2004) suggested five variables active in eating disorder vulnerability. The higher an individual's loading on these factors, the more apt he or she is to have eating-disordered behaviors or pathology. The five factors are (a) body image disturbance, (b) the internalization of the thin ideal, (c) poor family social support, (d) poor friend social support, and (e) negative affect. Tylka and Subich's model brings together not only the environmental and cultural influence of the pressure to be thin, but also personal and social variables. The model is a more precise examination of the SCT model for eating-disorder conceptualization. This model also lends credence to the eating disorder continuum hypothesis by illuminating the fact that not all individuals will have strong loadings on all five variables.

The primary predictor of eating disorder symptomology is body-image disturbance (Phelps, Johnston, & Augustyniak, 1999). Researchers have long accepted the role of body-image disturbance in eating disorder etiology (Frederickson & Roberts, 1997; Stice et al., 1996; Thompson et al., 1995; Tylka & Subich, 2004). Numerous studies indicate that individuals who have more body image disturbance are more likely to attempt to modify their bodies through the use of maladaptive weight-control techniques. Furthermore, higher levels of disturbed body image are negatively correlated with the ability to identify emotions as well as hunger and satiety signals (i.e., interoceptive awareness). Frederickson and Roberts (1997) suggested that the decrease in interoceptive awareness is due to the shame an individual feels when he or she has body-

image disturbance. This shame leads the individual to suppress hunger and satiety cues in order to lose weight or otherwise attempt to change his or her body.

Negative affect presents as both neuroticism and low self-esteem in individuals with disordered eating or eating disorders (Tylka & Subich, 2004). Negative affect accounts for many of the smaller variables making up the personal affective and cognitive aspect of SCT. For example, in Tylka and Subich's model, negative affect subsumes many other variables related to eating disturbances such as anxiety, depression, lack of impulse control, maladaptive coping, and irrational cognitions. Low self-esteem plays a large role in negative affect and is highly predictive of future eating disorder symptomology. Some theorists propose that negative affect is the key predisposing factor to internalizing the thin-ideal stereotype (Thompson et al., 1999). Furthermore, negative affect has a key role in the disturbed interoceptive awareness experienced by individuals with eating disorders. The lack of interoceptive awareness is not only related to the avoidance of hunger and satiety cues but, as Tylka and Subich (2004) have suggested, also related to the avoidance of all internal states including emotions. This suggestion has been supported in examination of the eating disorder continuum hypothesis in high school- and college-aged women (Pike, 1995; Tylka & Subich, 1999). Similarly, Mazzeo and Espelage (2002) reported that alexithymia, the inability to describe emotions verbally, is also a unique predictor of some eating-disorder variance.

Body-image disturbance and negative affect are at the core of the body-image schema. Intertwining these two components leads to a clear vulnerability to eating-disordered symptomology. When other variables such as poor relational or social support factors, genetic factors, and issues relating to trauma are heightened in certain

individuals, their predisposition toward having an eating disorder similarly is heightened. This conceptualization points to a diathesis-stress model for eating disorders. The eating disorder continuum model, in particular, supports a diathesis stress etiology, and it is further illuminated by the concepts set forth in SCT, in the body image schema, and ultimately in Tylka and Subich's (2004) multidimensional model of eating disorders.

Thus far, research has indicated that individuals with eating disorders exhibit dysfunctional emotional processing. This dysfunction appears to be correlated with appetitive responses generally related to the dopaminergic systems and reward pathways. Synaptic pruning and disturbances in the HPA axis also appear to play a role in the disruption of the development of collaborative brain function as well as an individual's ability to handle stress in an adaptive way. The combination of these factors hints at neurological underpinnings that are suggestive not only of potential vulnerability to acquiring an eating disorder, but also, almost certainly, of risk factors in both the onset and the maintenance of an eating disorder. These neurological vulnerabilities combine to create a biological environment susceptible to the pressures of western society. Today's world is rich with pressures that aid in both the development and maintenance of eating-disordered thoughts and behaviors. Clearly, effective treatment for an eating disorder must target a myriad of variables, often unique to each patient.

## Treatment of Eating Disorders

The treatment of such multifaceted and unique disorders has challenged the health care community for decades, if not centuries. The social, cultural, neurological, and biological risk factors and vulnerabilities explored thus far are just the tip of the iceberg.

Treatment response can be just as varied as the symptom presentation of these disorders. Thus far, this review has highlighted the importance of both the affective and the cognitive nodes of the social information processing network (SIPN; Nelson et al., 2005); the possibility of intense emotional dysregulation and impulsivity in individuals with bulimia, binge/purge subtype; as well as the inhibitory, detail-focused processes of individuals with anorexia. Treatment clearly needs to address all of these concerns, be it through pharmacotherapy, psychotherapy, or a combination approach. Studies investigating the use of pharmacotherapy in lieu of psychotherapy find it less effective, with the greatest rates of success seen when antidepressants are paired with psychotherapy.

**Psychopharmaceutical Treatment of Eating Disorders**

Anorexia and bulimia present a decades-long challenge to the psychiatric community. Since their emergence as a significant clinical entity, numerous studies have largely resulted in a knowledge base consisting more of "what not to do" than "what we should do" for treatment. A variety of medication trials have been performed with reports in the literature ranging from case reports of a few patients to the occasional double-blind, randomized, controlled trial. So far, only fluoxetine has received U.S. Food and Drug Administration (FDA) approval for the treatment of bulimia. There is currently no FDA-approved drug for the treatment of anorexia, although fluoxetine was found useful in the maintenance of recovery for anorexia (Holtkamp et al., 2005).

The lack of medications in the treatment of eating disorders remains a serious concern due to the continued difficulty in treating these patients and the high morbidity

and mortality rates resulting from them. Treatment is often costly and slow, with estimates of length of recovery ranging from 57 to 79 months (APA Work Group on Eating Disorders, 2000). A review of early studies using psychopharmacotherapy to treat eating disorders demonstrates a focus on the most serious and acute manifestations including malnutrition or weight loss and binge/purge cycles. Unfortunately, decades of research using weight gain and decreased binge/purge cycles as primary outcome measures in medication trials have provided few treatment options for either illness (Krüger & Kennedy, 2000). Although this may also be related to difficulties of studying this patient population, treatment providers are left sorely lacking tools to assist in the treatment of these serious and potentially lethal mental illnesses.

Bulimia has the best developed treatment literature for psychopharmacology. Studies have mainly focused on the use of selective serotonin reuptake inhibitors (SSRIs) for treatment, although anticonvulsants, opiate antagonists, serotonergic agonists, and lithium have been investigated for efficacy as well (Mitchell, de Zwann, & Roerig, 2003). Bulimic individuals tend to respond best to antidepressants, particularly SSRIs. This treatment results in significant reductions in eating-disordered behaviors such as binge eating and purging. Not surprisingly, these antidepressants also decrease comorbid psychopathologies such as anxiety disorders and affective disorder symptoms. As previously mentioned, fluoxetine is the only FDA-approved medication for the treatment of bulimia. In light of this, it has received the most attention in terms of research. One seminal research study examined patient response to fluoxetine, at 20 mg/day or 60 mg/day, as compared to a placebo. The higher dose of fluoxetine was reported to be visibly superior to the placebo (Fluoxetine Bulimia Collaborative Study Group, 1992).

A few small studies have examined the use of other medications as off-label treatment for bulimia. Topiramate, an anticonvulsant that is proposed to work on voltage-gated sodium channels, glutamate receptors, and gamma-Aminobutyric Acid (GABA) receptors, has shown some efficacy with bulimic patients (Zhu & Walsh, 2002). A case study by Knable (2001) reported a significant decrease in the patient's weight concern as well as a significant decrease in her desire to binge, purge, and self-mutilate following the use of topirimate to treat her epilepsy. Much more research is clearly needed before efficacy can be claimed for the treatment of bulimia with this medication.

So far, only two studies have examined the use of opiate antagonists for bulimia *(*Alger, Schwalbers, Bigaouette, Michalek, & Howard, 1991; Mitchell et al., 1989). Both studies focused on the use of naltrexone, a drug commonly prescribed to help alcoholics stop craving alcohol, in purging bulimics. Naltrexone is a competitive antagonist at the mu-opioid and kappa-opioid receptors and thus modulates the dopaminergic mesolimbic pathway affecting opiate receptors. The ineffectiveness found with naltrexone for the treatment of bulimia is not surprising considering the findings of Davis and Woodside (2002), who reported significantly less dopaminergic involvement for the purging bulimic than for the anorexic or other subtypes of the bulimic spectrum such as compulsive overexercisers.

Current literature has a dearth of studies examining the long-term efficacy of antidepressants on bulimia. This is particularly concerning in light of the aforementioned long-term disturbances in serotonergic activity in the brain. Another concern is the lack of research examining the different subtypes of bulimia. Current research on bulimia typically focuses on the prototypical purging subtype. Exercising bulimics portray

different DA activity than do purging bulimics. These differences may affect the efficacy of a drug on different bulimic subtypes.

Psychopharmacology for anorexia initially focused on SSRIs, with some efficacy being seen with them for relapse prevention in weight-restored anorexics (Holtkamp, 2005). A large variety of pharmacotherapy options have been explored with the anorexic population such as antipsychotics, narcotic antagonists, antihistamines, lithium, zinc, antidepressants, and tetrahydrocannabinol (THC; Attria & Schroeder, 2005; Mitchell et al., 2003). New research on the psychopharmacology of anorexia has focused on atypical neuroleptics in order to better address the resistance to treatment commonly seen in anorexia. The most difficult part of treating an anorexic patient is that such patients present with two very serious concerns: the psychopathology of an anorexic and the physiological attributes of a person close to emaciation presenting with symptoms such as osteoporosis and amenorrhea (Mitchell et al., 2003). Separating the immediate physiological concerns from the psychological concerns can be difficult to treat psychopharmacologically. As the anorexic individual is restored to a healthy weight, he or she may present with very different biological patterns and thus have different responses to medications.

Typical pharmacotherapy for the acute anorexic focuses on medications designed to induce weight gain such as antidepressants, lithium, and anticonvulsants. Treatment trials with this methodology are not proven to have long-term efficacy. Use of SSRIs in the treatment of acute anorexia also has limited results. Attia and Schroeder (2005) reported no benefit of fluoxetine use in inpatient anorexics as compared to placebo. They hypothesized that underweight anorexics may have neurochemical disturbances

disrupting the drugs' mechanism of action, specifically disturbed 5-HT function. Problematically for this hypothesis, 5-HT disturbance appears to remain in anorexic individuals even after recovery (Kaye et al., 2005). The purported inaction of SSRIs on the acute anorexic may be caused by an unknown mediating factor, perhaps the body's physical state. Interestingly, fluoxetine is reported as effective in weight maintenance as it is in weight restoration, although results are still inconclusive (Kaye et al., 1999).

Numerous other medications are used to induce weight gain in the anorexic individual. The opiate antagonists naloxone and naltrexone both result in consistent weight gain in underweight anorexics. A randomized, placebo-controlled, double-blind study on anorexics using naltrexone similarly reported significant improvement in eating-disordered behaviors such as binge-eating and purging (Marrazzi, 1995). These findings offer further support for the autoaddiction opioid theory of anorexia and should prompt future investigation into an addiction model of anorexia. A small study of lithium reported short-term weight gain in an anorexic population, but additional research is needed (Gross, Ebert, Faden, et al., 1981). Because of its appetite-stimulating effects, THC was examined in an anorexic population. No benefit of weight gain was seen, and several participants dropped out of the study due to the side effects of the THC such as paranoia, interpersonal sensitivity, and sleep disturbance (Gross, Ebert, Faden, et al., 1983). Use of THC is this population was not shown to be efficacious.

Finally, antipsychotics have been heavily examined in the anorexic population with studies dating as far back as the 1960s. Initial studies did show enhanced weight gain, particularly on chlorpromazine, but participants displayed negative side effects such as seizures and increased purging (Attia & Schroeder, 2005). Pimozide was also

examined in an anorexic population, and although it did result in weight gain, there was no improvement in patient behaviors or attitudes. Due to the significant negative side effects and the minimal clinical efficacy of the traditional antipsychotics, they have not been considered for mainstream anorexic treatment. The new atypical antipsychotics present a new option for eating disorder clinicians due to their more manageable side effects. In particular, olanzapine has been examined in several studies. It is currently associated with some behavioral and psychological improvement as well as improved weight gain (La Vie, Kaye, & Grey, 2000; Powers, Santana, & Bannon, 2002). Clinical trials are needed to further support the efficacy of this medication for anorexia.

Most treatment practitioners working in the field of eating disorders recognize the benefit of a combined pharmacotherapeutic and psychotherapeutic approach to treating these multifaceted disorders. A review completed by Shapiro and colleagues (2007) found only six studies examining the combined effects of pharmacotherapy and psychotherapy in the treatment of bulimia. Combined treatment was associated with a greater decrease in both binge and purge behaviors (Goldbloom, Olsted, Davis, et al., 1997; Walsh, Wilson, Loeb, et al., 1997). Combined treatment studies for anorexia are few and far between, with neither pharmacotherapy nor psychotherapy showing consistent positive outcomes. The numerous factors neurologically, socially, culturally, cognitively, and biologically account for the difficulty in finding successful treatment strategies for both bulimia and anorexia. To date, a myriad of different approaches are used psychotherapeutically to treat these disorders.

## Psychotherapeutic Models for the Treatment of Eating Disorders

Seven primary treatment models for eating disorders are commonly used today. They are (a) psychodynamic therapy, (b) cognitive behavioral therapy (CBT), (c) enhanced cognitive behavioral therapy (eCBT), (d) integrative cognitive-affective therapy (ICAT), (e) interpersonal therapy (IPT), (f) dialectical behavioral therapy (DBT), and (g) the addiction model.

Psychodynamic psychotherapy is one of the oldest and most respected forms of treatment for mental illness. The psychodynamic philosophy places emphasis on internal conflicts and motives as well as unconscious forces. Through this focus on unconscious motives and conflicts, therapists are able to help the patient decipher the root causes of their behaviors (Trull & Phares, 2001). While there are numerous psychodynamic theories, such as self psychology and object relations, the underlying core concept of treatment does not vary significantly. The underlying approach of psychodynamic therapies is that underlying causes for disordered behaviors must be addressed and resolved or else the behavior will continue to return. While traditional psychodynamic views of maladaptive food behaviors were fixated on the sexual nature and interpretation of the individual's relationship to food, modern psychodynamic practitioners in the field of eating disorders have a distinctly different view of maladaptive food behaviors (Furumoto & Keating, 1995).

Modern psychodynamic theorists posit that adaptive behaviors arise when developmental needs are not met (Costin, 1999). Adaptive behaviors then function as substitutes for the developmental deficits and protect the individual against resulting pain, frustration, and/or anger. The primary difficulty with these adaptive behaviors is that they

never become internalized, as they do not have the ability to replace the behaviors needed for healthy development. For individuals struggling with eating disorders, some of these adaptive behaviors can go on to threaten long-term health and may even lead to death. For example, an individual who was never taught to self-soothe may rely on food for comfort and thus binge eat when distressed. Another common example is an individual who never developed an internal locus of control. When all control in the individual's life is externalized, events may feel chaotic, and severe restriction and control of food may provide an internal sense of stability and safety. Gabbard (2000) provided a comprehensive summary of the multiple developmental deficits that an individual may experience and how eating-disordered behavior may serve as adaptive behavior:

> (1) a desperate attempt to be special, (2) an attack on the false sense of self fostered by parental expectations, (3) an assertion of a nascent true self, (4) an attack on a hostile maternal introjects viewed as equivalent to the body, (5) a defense against greed and desire, (6) an effort to make others—rather than the patient—feel greedy and helpless, (7) a defensive attempt to prevent unmetabolized projections from the parents from entering the patient, and (8) an escalating cry for help to shake the parents out of their self-absorption and make them aware of the child's suffering.

In the psychodynamic framework, symptoms are considered to be expressions of a struggling inner self. For an individual with an eating disorder, this struggling inner self is using the disordered eating and weight-control behaviors as the primary method of expressing the underlying problems or issues. These symptoms, then, are actually the only method of communication for the pain and anger that the individual has, and thus efforts to take them away are avoided (Costin, 1999). Ultimately, once the individual has learned to internally meet his or her needs and no longer struggles with his or her initial

development deficits, the eating-disordered behaviors will no longer be necessary and will subside on their own.

Regardless of the primary framework of the psychodynamic approach, the primary goal in therapy is to help the patient gain insight into how his or her past, personality, and personal relationships interact and how this interaction relates to the eating disorder. While an understanding of these factors and their interplay is clearly valuable to an individual with an eating disorder, the psychodynamic approach to treating eating disorders has two problems. First, patients with eating disorders are often in such a state of depression, starvation, and/or compulsivity that their ability to explore their histories and interpersonal relationships is almost nonexistent. In consideration of this, medical stability, suicidal tendencies, starvation, compulsive binging/purging, and other harmful behaviors will have to be addressed before any psychodynamic work can commence (Costin, 1999). Second, it may take years for the patient to come to a full understanding and integration of the factors leading up to his or her eating disorder. During this time, the individual's harmful behaviors may persist or worsen. This second concern calls for an intervention with more immediate benefits.

Cognitive behavioral therapy (CBT) is recognized as the most efficacious form of treatment for bulimia and is often referred to as the Gold Standard (Fairburn, 2006). Recently, the National Institute for Clinical Excellence (NICE) assigned CBT a grade of A as an empirically supported treatment modality for bulimia. Despite these accolades, 40% of individuals with bulimia who complete CBT will have relapsed by a 60-week follow-up (Fairburn, Cooper, Doll, O'Connor, Bohn, & Hawker, 2008). The primary mechanism of action in CBT is to help the client identify and change maladaptive

cognitions. Cognition is a mental perception or awareness of one's world. Cognitions are fundamental to an individual's ability to navigate the world in a successful manner. They provide algorithms and patterns that make sense of the overwhelming sensory experience of daily life. Individuals who struggle with depression, low self-esteem, anxiety, and a myriad of other life-disrupting conditions typically struggle with harmful, or maladaptive, cognitions.

Individuals who struggle with eating disorders tend to have rather insidious cognitions concerning their identity, body, food, and other concepts tied to the inception and maintenance of an eating disorder. These maladaptive cognitions are held sacred because they provide a sense of control and order to an otherwise chaotic universe (Costin, 1999). Eating-disordered behaviors such as binge eating, food restriction, purging, and overexercising are all products of beliefs, attitudes, and assumptions about the meaning of body weight and eating. These beliefs, attitudes, and assumptions are typically distorted and in extreme situations may lead to an eating disorder. One of the primary jobs of a therapist, regardless of orientation, is to begin to address, challenge, and change these disordered thought processes. As mentioned previously, this is the primary mechanism of action for a CBT therapist.

Costin (1999) identified four primary functions that cognitive distortions serve for an individual suffering from an eating disorder.

1. Cognitive distortions provide a sense of being in control and being safe.

   *Example:* Cognitive distortions such as all-or-nothing thinking and extreme thinking provide a strict system of rules concerning acceptable and forbidden foods. Costin discussed one bulimic woman who allowed herself no fat in her diet. If she did

happen to eat something with fat in it, then she felt as though she had "failed her system" and would subsequently binge on all of the forbidden foods and then purge.

2. Cognitive distortions further reinforce the eating disorder as an integral part of the person's identity.

   *Example:* Eating-disordered behaviors such as restriction, overexercising, and weight issues make the person feel unique and special. The individual becomes identified to others as an individual with an eating disorder and further internalizes this identification until the individual does not know who he or she would be without the eating disorder. The maladaptive thoughts and beliefs create the individual's sense of self.

3. Cognitive distortions enable the individual to replace reality with a system that supports the individual and allows him or her to rationalize his or her behaviors.

   *Example:* Individuals struggling with an eating disorder use their distorted system of rules to create a safe world to navigate. Magically thinking that one's worries will disappear as long as one weighs only 78 pounds creates a system where the individual focuses solely on obtaining the goal weight at the cost of any other indicators of reality.

4. Cognitive distortions help provide a justification or explanation of the individual's behaviors to other people.

   *Example:* Physiological maladies are often drawn upon as explanations as to why an individual can't and/or won't eat. Allergies pertaining to sugar, dairy, wheat, and other common ingredients are often created to explain an individual's severe restriction from a certain food group. Statements such as "I already ate" are meant to

soothe concerned family and friends. For an individual struggling with an eating disorder, "eating a meal" can be equivalent to eating a small handful of grapes or a cup of air-popped popcorn.

As shown, cognitive distortions provide an insidious mechanism for the eating disorder to infiltrate the core of a person's sense of self. If these distortions are not appropriately addressed, the distortions and the corresponding symptomatic behaviors will persist.

Despite the reported therapeutic efficacy of these primary types of treatment for eating disorders, they still result in disappointingly low rates of total remission. There are only two major studies examining the efficacy of CBT. Rates of recovery stall around 40%. In a large study funded by the National Institute of Mental Health, recovery rates in the intervention sample were 40% while the treatment-as-usual group saw recovery rates of 29% (Agras et al., 2000). The McKnight Foundation found a recovery rate of 41% in the intervention sample and 31% in the treatment-as-usual group (Mitchell, Halmi, Wilson, Agras, Kraemer, et al., 2000). Due to the low rates of recovery, newer forms of therapy are constantly being investigated for the treatment of eating disorders.

Fairburn, Cooper, and Shafran (2003) recently developed a form of CBT specifically designed to treat individuals with eating disorders. This form of therapy, called *enhanced CBT* (eCBT), includes the core premises of CBT while adding four additional factors specific to the eating-disordered population. eCBT places a special emphasis on interpersonal difficulties, clinical perfectionism, mood intolerance, and low self-esteem. This form of treatment was designed for outpatient therapy and has two treatment models. One is for an eating disorder patient with a BMI greater than 17.5; it

takes 20 weeks to complete the entire sequence. The second sequence is for individuals with a BMI lower than 17.5 and takes 40 weeks to complete. Fairborn et al. (2003) devised a complete treatment strategy around four stages. The first stage incorporates case formulation with early behavioral change. This stage is designed around biweekly treatment sessions. The second stage reviews the case formulation and incorporates a more in-depth intervention for the individual's problems in areas specific to the four factors listed above. Stage 3 contains the majority of the treatment utilizing CBT concepts but also includes modules specific to the four factors. Finally, during Stage 4, the therapist works to help the individual devise a relapse prevention plan and encourage the continuation of recovery. Results on the efficacy of eCBT are not yet available, as Fairborn and colleagues are currently involved in a large study.

Another variation of the CBT paradigm is integrative cognitive-affective therapy (Mitchell, Agras, & Wonderlich, 2007). This form of therapy emphasizes self-oriented cognitions, interpersonal schemas, emotional experiences, interpersonal patterns, and cultural experiences. Integrative cognitive-affective therapy is strongly based in personality, attachment, and self-discrepancy theories. The theoretical background of this theory is that individuals who suffer from bulimia experience a self-deficit between their actual self and their ideal self. Due to this deficit, they develop an internal aversion to their sense of self and corresponding negative affect. As they expect to be rejected for not living up to the expectations of others, they develop maladaptive interpersonal patterns to stave off abandonment and/or rejection. Similar to eCBT, this intervention is conceptualized in four distinct phases. The first phase incorporates the first three sessions of therapy. During this phase, the therapist focuses on increasing client motivation and

psychoeducation. The second phase occurs over the next five sessions of therapy and is focused on normalizing eating behaviors and helping the client develop coping skills. Phase 3 spans Sessions 9 to 18 and contains the bulk of the work concerning intrapersonal (cognitive) and interpersonal factors. In Phase 3, the therapist and client focus on the primary factors, mentioned above, that make up the bulimic individual's pathology. Finally, Phase 4 focuses on the development of a maintenance and relapse prevention plan.

Another primary form of therapy used to treat bulimia is interpersonal therapy (IPT). IPT is based on the premise that interpersonal factors play a significant role in the inception and maintenance of many disorders. It was originally developed as an intervention for depression (Weissman & Markowitz, 1995). IPT for bulimia focuses on four areas of interpersonal concern: interpersonal role disputes, interpersonal deficits, role transitions, and grief (Jacobs, Robinson-Welch, & Wilfley, 2004). Typically, IPT engages several therapeutic tools used to address these four areas, which include but are not limited to communication training, feedback on problematic interactive patterns, identification and exploration of feelings, and expectation modification. To date, IPT is the only form of therapy that has outcomes comparable to those of CBT (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Fairburn, Jones, Preveler, Hope, and O'Connor, 1993). Research suggests that this type of therapy may take longer to have an effect, as end-of-treatment assessments are less favorable than those of CBT. Interestingly, at 1-year follow-ups, the differences between individuals treated with CBT, as compared to those treated with IPT, are insignificant. IPT therefore presents as a viable treatment alternative for individuals with bulimia who are reluctant to engage in CBT or are

struggling with interpersonal problems.

Dialectical behavioral therapy (DBT), pioneered by Marsha Linehan, has become popular as a treatment choice for those struggling with an eating disorder. DBT was originally designed to treat individuals suffering from borderline personality disorder and/or struggling with suicidal and self-injurious behaviors (Linehan, 1993). The methodology of DBT has since been modified for use in the treatment of eating disorders and was shown to be effective in a small study conducted by Safer, Telch, and Agras (2001). The treatment focus in DBT is behavioral change and strategies to help individuals learn to accept themselves. The intervention is focused on the relationship between the change process and self-acceptance. Linehan developed four skill modules involved in skill acquisition: mindfulness, interpersonal effectiveness, emotion regulation, and distress tolerance. Researchers hypothesize that DBT is effective in treating bulimia because it targets the individual's problems with emotion dysregulation, which may be one of the core factors underlying bulimia (Mitchell et al., 2007).

Finally, some eating disorder practitioners use an addiction or disease model to treat eating disorders. This model is also known as the abstinence model. This model is adapted from the disease model of alcoholism (Rosenberg, Devine, & Rothrock, 1995; Rosenberg & Rosen, 1994; Weisner, 1995). In this model, alcoholics are considered powerless because the disease of alcoholism creates abnormal and addictive responses in their body to the consumption of alcohol. Because of these abnormal and addictive responses, the individual considers him- or herself to be powerless over the alcohol and turns to a greater "power" for help. The Twelve Step program of Alcoholics Anonymous was designed around this principle and is used worldwide to treat alcoholism. When

practitioners apply this model to the treatment of eating disorders, they simply replace the word "alcohol" with "eating disorder," resulting in addiction support groups such as Eating Disorders Anonymous (EDA) and, similarly, Overeaters Anonymous (OA). The primary treatment approach of EDA and OA is to help the participant create and maintain abstinence from foods that are considered to have addictive qualities, such as sugar and white flour. As the participants work to gain and maintain abstinence from these addictive foods, they work through the Twelve Steps of EDA.

The initial application of the addiction analogy of alcoholism to compulsive overeating stems from the idea that if ingredients in alcohol could lead to an addictive state, then certain foods might have the same effect. Despite the large numbers of individuals utilizing the twelve-step model to help overcome their harmful relationship with food, there is also no proof that the addiction model is a successful treatment model for eating disorders or compulsive overeating (Costin, 1999). According to Hatsukami, Owen, Pyle, and Mitchell (1982), the addiction model for the treatment of eating disorders appears to have been readily adopted by practitioners due to the absolute dearth of other treatment models. In 1993, the American Psychiatric Association (APA) cautioned against the use of twelve-step models as the primary treatment for bulimia and anorexia in their treatment guidelines for eating disorders. The APA stated concerns that due to

> the great variability of knowledge, attitudes, beliefs, and practices from chapter to chapter and from sponsor to sponsor regarding eating disorders and their medical and psychotherapeutic treatment and because of the great variability of patients' personality structures, clinical conditions, and susceptibility to potentially counter therapeutic practices, clinicians should carefully monitor patients' experiences with the Twelve Step programs. (APA, 1993)

**Psychotherapeutic Treatment Outcomes in Bulimia**

In order to fully conceptualize the purpose and use of different treatment modalities, it is important to define what recovery from bulimia is and the goal for intervention. Typically, there are six primary objectives in the successful treatment of bulimia (Mitchell et al., 2007). The first goal is to eliminate binge eating patterns and compensatory behaviors. Second, the goal is to help the individual return to a normal and healthy pattern of eating. Third, the importance of medical stability is taken into consideration, and physical complications from the disorder are assessed. The fourth objective is to address the underlying psychological concerns of the individual. This typically includes addressing issues with self-esteem, interpersonal concerns, body image dissatisfaction, and any other dysfunctional thought or behavioral patterns. Finally, treatment must take into consideration comorbid conditions and address them effectively. For example, it is quite common for individuals suffering from bulimia to have comorbid diagnoses of depression and anxiety. The final objective of the treatment of bulimia is to prevent relapse.

Literature on treatment modalities for bulimia is widespread. Since its original description in 1979, a breadth of information regarding this disorder has developed. Mitchell, Agras, and Wonderlich (2007) noted six main treatment modalities for bulimia that are commonly used to treat this disorder. The first of these interventions has already been discussed at length—psychopharmaceuticals. Optimal treatment results are seen where there is a combination of psychopharmaceuticals and some form of psychotherapy. The other five primary forms of treatment intervention for bulimia have already been discussed. They are (a) cognitive behavioral therapy (CBT), (b) enhanced cognitive

behavioral therapy (eCBT), (c) integrative cognitive-affective therapy (ICAT), (d) interpersonal therapy (IPT), and (e) dialectical behavioral therapy (DBT).

Fairburn, Agras, Walsh, Wilson, and Stice (2004) found that the best predictor of a good outcome at 8 months follow-up from treatment was an early reduction in purging behavior. Unfortunately, there have been no studies supporting this as a positive outcome variable 8 months posttreatment. Other researchers indicate that normalization in eating patterns is a positive outcome predictor at 3 months posttreatment (Burton & Stice, 2006), but again there are no longer term follow-ups available. Clearly, while many variables are hypothesized concerning the inception of, maintenance of, and recovery from bulimia, none of these variables have been clearly indicated through research.

While a wide range of treatment strategies for bulimia exist, the effectiveness of such strategies is low. Wilson (1996) and Agras (1993) maintained that recovery is only achieved by about 50% of patients who enter treatment. Such low numbers prove that further investigation into existing methods is needed. Complications with treatment are often related to the difficulty in defining the etiology of eating disorders as well as the role of societal and cultural factors. Researchers appear to have reached a consensus regarding the importance of cognitions and interpersonal relationships in the maintenance of bulimia. However, many clinicians and researchers also report reaching an impasse when trying to identify which forms of therapy are most appropriate for each individual. For example, Nevonen and Broberg (2006) suggested that IPT is more effective with bulimic individuals struggling with impulsivity and affective instability, whereas CBT is the treatment of choice for those whose EDO has an origin in body image, eating concerns, and a focus on weight.

Unfortunately, these aspects of an individual's eating disorder may not emerge until late in treatment. A recent factor analysis examining commonly used treatment strategies for eating disorders reported that only 6% of respondents used a standardized treatment manual, while 98% of the respondents used an eclectic methodology combining strategies of all seven treatment methods described above (Tobin, Banker, Weisberg, & Bowers, 2007). Empirically validated outcome strategies are almost impossible to assess in such a varied and diverse treatment arena. Thus far, current research has only focused on outcome data, which merely highlight success or failure in treatment.

**Psychotherapeutic Treatment Outcomes in Anorexia**

The treatment of anorexia suffers from much darker outcome results than bulimia. Longitudinal studies on anorexia report high mortality rates. Studies with follow-ups completed 5 to 10 years after treatment report mortality rates of 3% to 6%, range 0% to 11.5%, while studies with a longer period between treatment and follow-up portray an even grimmer picture, with mortality rates between 0% and 17.5% (Fichter, Quadflieg, & Hedlund, 2006). Several studies have attempted to establish outcome predictors. Following regression analysis, 11 variables predictive of outcome appear to be similar across studies: duration of the eating disorder, age of onset, family of origin, age at onset of menstruation, sexual problems, psychiatric comorbidity, perfectionism, impulsivity, self-evaluation, extroversion, and low body weight (Fairburn, Cooper, Doll, & Welch, 1999; Keel & Mitchell, 1997; Lindberg & Hjern, 2003; Quadflieg & Fichter, 2003; Steinhausen, 2002). These predictive variables may present in a wide variety of constellations. Other problems with the development of effective and empirically based

treatments for anorexia include the small sample sizes of the studies and their contrasting results.

A recent investigation by Fichter, Quadflieg, and Hedlund (2006) followed 103 individuals for 12 years who were admitted for the treatment of anorexia. Follow-ups were conducted at 2, 6, and 12 years posttreatment. The investigation of Fichter et al. (2006) provided important results due to the long-term follow-up and large sample size. The results of this study were illuminating and shed light on a series of factors that ought to be incorporated into the successful treatment of anorexia. These variables, shown in Table 1, exemplify factors consistent across a large sample size.

The greatest predictor of poor outcome for anorexia is sexual problems. This variable includes body contact, sexual arousal, and related feelings. Two conceptualizations are obvious when discussing the role of this variable in the maintenance of anorexia. Sexual problems may relate to factors involving maturation, self-identity, self-esteem, as well as societal conceptualizations of the sexualized female form. This variable highlights the possible interaction of sexual abuse and lingering intimacy concerns in the maintenance of anorexia. Participants in this study who had experienced sexual abuse before the age of 11 had significantly higher sexual problems and poorer outcome rates. This demonstrates the importance of treating sexual problems in individuals struggling with anorexia. The second primary predictor of poor outcome of treatment in individuals with anorexia was impulsivity, also a commonly accepted predictor of bulimia. The last two significantly predictive variables were duration of inpatient stay and duration of eating disorder. The more time an individual spent in an inpatient setting, the worse his or her prognosis became. Similarly, the longer an

Table 1

*Final Predictive Model of Poor Eating Disorder Outcome 12 Years After Index Treatment in Anorexia Nervosa (Fichter et al., 2006)*

| | **$R^2$ = 0.45 predictor** | Wald statistics | Odds ratio | 95% CI |
|---|---|---|---|---|
| 1 | High intensity of sexual problems | 6.9** | 5.08 | 1.51–17.09 |
| 2 | Impulsivity | 4.9* | 3.71 | 1.16–11.87 |
| 3 | Long duration of index inpatient treatment | 4.2* | 1.52 | 1.02–2.28 |
| 4 | Long duration of eating disorder | 9.7** | 1.27 | 1.09–1.47 |

**Note. 95% CI = 95% confidence interval.**
*** $p < .05$.**
****$p < .01$.**

individual suffered from anorexia, the greater his or her chances were of achieving a poor outcome.

While the predictors found in the investigation of Fichter et al. (2006) are important, Fichter et al. also reported interesting results regarding individuals in remission from anorexia. The typically accepted definition for recovery from anorexia, weight being within 15% of ideal body weight (le Grange & Rock, 2005), may not be an adequate predictor of eating disorder remission or recovery. Despite no longer meeting the diagnostic criteria for anorexia, these individuals still maintain significantly different characteristics from the normal population. For example, recovered individuals maintained significantly higher levels of eating-disordered behaviors and general psychopathology. Recovered anorexics also maintained certain pathological attitudes in regard to the thin ideal and body image. Interestingly enough, there was no difference between non-eating disordered women and the recovered individuals in regard to eating-

disordered characteristics related to binge eating, atypical compensatory behaviors, or restrictive eating patterns. These results further highlight the difficulty in treating body image disturbance despite an alleviation of food-related eating disordered behaviors. Le Grange and Lock (2005) completed a review of all literature reporting on therapeutic treatments and/or efficacy for anorexia. They identified eight uncontrolled and five controlled studies examining the treatment of adolescents with anorexia, and seven controlled investigations on the treatment of adults with anorexia. There is clearly a dearth of literature and research examining the treatment of this life-threatening disorder.

In 1975, Minuchin and associates completed what is now considered a seminal work for the treatment of adolescents with anorexia. In an uncontrolled investigation at the Child Guidance Clinic in Philadelphia, PA, they treated 53 adolescent patients with family therapy and wrote an article discussing their results (Minuchin, Baker, Rosman, Liebman, Milman, & Todd, 1975; Minuchin, Rosman, & Baker, 1978). The individuals in this study were largely comprised of adolescents (only 3 were greater than 18 years of age) diagnosed with anorexia for 8 months or less. Minuchin and associates reported very high success rates. Eighty-six percent of the patients were reported recovered at the time of follow-up. While the results appear very promising, it is important to remember that duration of illness is most likely a primary predictor of outcome and that the short duration of the illness might have positively impacted recovery rates. Regardless, the work of Minuchin and his associates was impactful for two reasons: (a) the number of recovered individuals was large, and (b) the theoretical underpinnings of their approach could be replicated.

Minuchin and associates (1975) conceptualized a “psychosomatic family” that

was at the core of an individual's struggle with anorexia. The "psychosomatic family" consisted of a particular family process in which family members were enmeshed, rigid, and highly avoidant of conflict. This family system sets the stage for an individual, when coupled with the developmental demands of adolescence, to develop anorexia as a way of navigating the system. Minuchin and associates (1975) cautioned against seeing their theory as an etiologic concept of how anorexia develops, urging readers instead to consider the eating disorder within this framework as a constantly shifting and evolving part of the family process. Therefore, the ultimate goal of the Minuchin treatment was to alter the family's interactive engagement.

It wasn't until 1987 that a group of researchers at the Maudsley Hospital in London attempted a systematic investigation of the Minuchin methodology. Russell, Szmukler, Dare, and Eisler (1987) examined the effectiveness of individual outpatient therapy as compared to family therapy in adolescents with anorexia. While Russell and associates largely recreated the environment of Minuchin's treatment, they added in several new key variables. For example, they engaged the parents in the refeeding process of the adolescents and maintained parental involvement in this matter until weight was restored. They also did not begin to address individual or family concerns until after weight restoration was achieved. Russell et al. (1987) compared family therapy to a systematized supportive individual therapy conceptualized as "treatment as usual." Results of these studies showed promise for the method conceptualized by Minuchin and associates (1975). At the 5-year follow-up, only 36% of patients who received individual therapy reported a favorable outcome, whereas 90% who received family therapy had a positive outcome.

More recently, other investigators have compared different forms of family therapy for the treatment of adolescent anorexia. These studies found that regardless of the type of family therapy (conjoint family therapy vs. separated family therapy), individuals receiving family therapy still had significantly better outcome results than did those just receiving individual therapy (Eisler, Dare, Hodes, Russell, Dodge, & Le Grange, 2000; Morgan & Hayward, 1988). Eisler et al. (2000) also found results for families and individuals who participated in conjoint family therapy. At 5-year follow-up, 75% of patients who received some form of family therapy had a positive outcome, as compared to 15% who had good outcomes with individual therapy. Another study investigated outcomes with behavioral systems family therapy as compared to ego-oriented individual therapy (Robin, Siegel, Moye, Gilroy, Dennis, & Sikand, 1999). Robin and associates found that individuals who received family therapy were significantly more likely to return to normal weight and to have regained their menses. Interestingly, the two groups showed no difference in regard to changes in depression, eating attitudes, and eating-related family conflict. These results emphasize the potential role of the family in the physical aspect of recovery but not in the more cognitively oriented aspects of recovery. This is an essential differentiation, as almost all studies, to date, have defined an individual as recovered from anorexia if he or she has returned to normal weight.

In comparison to studies examining adolescents with anorexia, studies examining interventions for adult individuals with anorexia are just as difficult to find. Before le Grange and Lock's review (2005) of the treatment of anorexia, there had only been seven studies examining outpatient therapy outcomes of individuals with anorexia. These

studies examined a variety of therapies such as individual therapy, cognitive behavioral therapy, individual therapy combined with family therapy, group therapy, dietary counseling, and nutritional advice. Similar to the studies on individuals with bulimia and adolescents with anorexia, all outcome results were based on posttreatment reported results via either a self-report questionnaire or a clinical interview.

In 1987, the first controlled trial investigating outpatient treatment results on adults with anorexia was published. Thirty-six patients were randomly assigned to receive either individual or family therapy following discharge from the hospital (Russell, Smuzkler, Dare, & Eisler, 1987). Although those who received individual therapy showed an initial improvement over those receiving family therapy at the 5-year mark, there was no significant difference between the two groups. Another study found similar results when examining two different types of individual therapy, focal therapy and cognitive analytic therapy, when compared to family therapy (Dare, Eisler, Russell, Treasure, & Dodge, 2001). No single form of therapy proved more efficacious than the others at the time of treatment termination. Numerous other studies investigating and comparing a variety of treatment modalities for anorexia have found similar results, with no significant difference between the groups at time of termination (Channon, De Silva, Hemsley, & Perkins, 1989; Crisp, Norton, Gowers, Halek, Bowyer et al., 1991; Treasure, Todd, Brolly, Tilley, Nehmed, et al., 1995).

Only two studies examining controlled treatment effects on adults with anorexia had significant results. One study compared nutritional counseling to outpatient CBT in 33 women posthospitalization (Pike, Walsh, Vitousek, Wilson, & Bauer, 2003). This investigation was more empirically sound than those previously noted because it utilized

manualized treatment interventions. It is also important to note that this investigation was significantly more intense than the previous studies and consisted of 50 treatment sessions over a course of 12 months. Results of this study indicated a significantly lower nonresponse rate for individuals in the CBT group. Furthermore, when the researchers applied Fairburn and Cooper's (1993) criteria of a good outcome –i.e., no binge eating or purging behaviors, weight restored, and < 1 standard deviation (SD) from the norm on the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), none of the individuals in the nutritional counseling group were shown to have a good outcome, while 17% of those in the CBT group did. While the results of this study are mildly discouraging, they portray the reality of current treatment outcomes for women struggling with anorexia.

Another interesting study examined two of the specialized therapies purported to be the most efficacious for eating disorder treatment. McIntosh et al. (2005) examined the effectiveness of CBT and IPT compared to a treatment-as-usual group. Interestingly, after 20 sessions for 20 weeks, the control treatment group had treatment outcomes that were superior to those of either CBT or IPT. The results of this study, as well as the previously discussed studies, indicate the difficulty in creating and implementing an effective treatment strategy for anorexia. These studies reported results of "good" and "intermediate" outcomes as anywhere from 29% to 63%, with the majority of individuals falling into the 60% range. It is also extremely important to remember that in all but one study, there were no follow-up data. The previously discussed long-range follow-up data of 12 years posttreatment paint a much grimmer picture, with mortality rates of up to 17.5% and only 52.4% of participants obtaining recovery (Fichter, Quadflieg, & Hedlund, 2006).

In an unprecedented investigation, Kordy and associates (2002) examined 422 bulimic and 233 anorexic patients over the course of 2.5 years. Utilizing the Longitudinal Follow-up Evaluation, they attempted to establish operational definitions for partial and full remission, relapse, and recovery for individuals with anorexia and bulimia (Frank et al., 1991). Given the previously discussed difficulties with defining outcome in this population, their task was not an easy one.

Utilizing the operational definitions provided in Table 2, the treatment progress of a total of 655 patients was assessed using the Kaplan-Meier Survival Analysis to determine the chances for remission or recovery as well as those for relapse or recurrence. Partial remission was the most commonly observed clinical phenomenon. Twenty percent of the anorexic patients and 30% of the bulimic patients had progressed to this stage of treatment at the time of entering the posttreatment stage. These proportions did increase through the posttreatment stage to 55% for anorexics and 60% for bulimics. Full remission or recovery was a significantly rarer occurrence (Figure 4), with only 7% of anorexics and 18% of bulimics achieving full remission. Only 6% of anorexics and 16% of bulimics were considered recovered (Figure 5).

Kordy and associates' (2002) research pinpointed important findings about the stability of remission and recovery in these disorders. Full remission and recovery was more stable than partial remission. Out of the 22 anorexic patients who obtained full remission or recovery during the 2.5 years of observation, only 2 relapsed (1 in Month 6 and 1 in Month 21). Partial remission was much less stable, with 35% of the anorexic patients obtaining partial remission relapsing. During the first 7 months of achieving

Table 2

*Kordy and Associates (2002) Operational Definitions of Possible Eating Disorder Treatment Outcomes*

| **Symptom** | **Concept** Partial Remission (1 month) | Full Remission (3 months) | Recovery (12 months) |
|---|---|---|---|
| *Anorexia: Restricting Type* | | | |
| Underweight ($kg/m^2$) | BMI > 17.5 | BMI > 19 | BMI > 19 |
| Fear to gain weight | -- | No extremes | No extremes |
| Weight reduction by (#/wk) | Vom.[a] & Lax[b]. = 0 | Vom. & Lax. = 0 | Vom. & Lax. = 0 |
| Binges (#/wk) | 0 | 0 | 0 |
| *Anorexia: Binge Purge Type* | | | |
| Underweight ($kg/m^2$) | BMI>17.5 | BMI>19 | BMI>19 |
| Fear to gain weight | -- | No extremes | No extremes |
| Binges (#/wk) | ≤ 1 | 0 | 0 |
| Weight reduction by (#/wk) | Vom. & Lax. = 0 | Vom. & Lax. = 0 | Vom. & Lax. = 0 |
| *Bulimia* | | | |
| Binge/Purge (#/wk) | ≤ 1 | 0 | 0 |
| Preoccupation with figure | -- | No extremes | No extremes |
| Weight reduction by (#/wk) | Vom. & Lax. ≤ 0 | Vom. & Lax. = 0 | Vom. & Lax. = 0 |

*Relapse:* change from partial or full remission to full syndrome according to DSM-IV
*Recurrence:* change from recovery to full syndrome according to DSM-IV

**[a] Weight reduction by vomiting**
**[b] Weight reduction by laxative abuse**

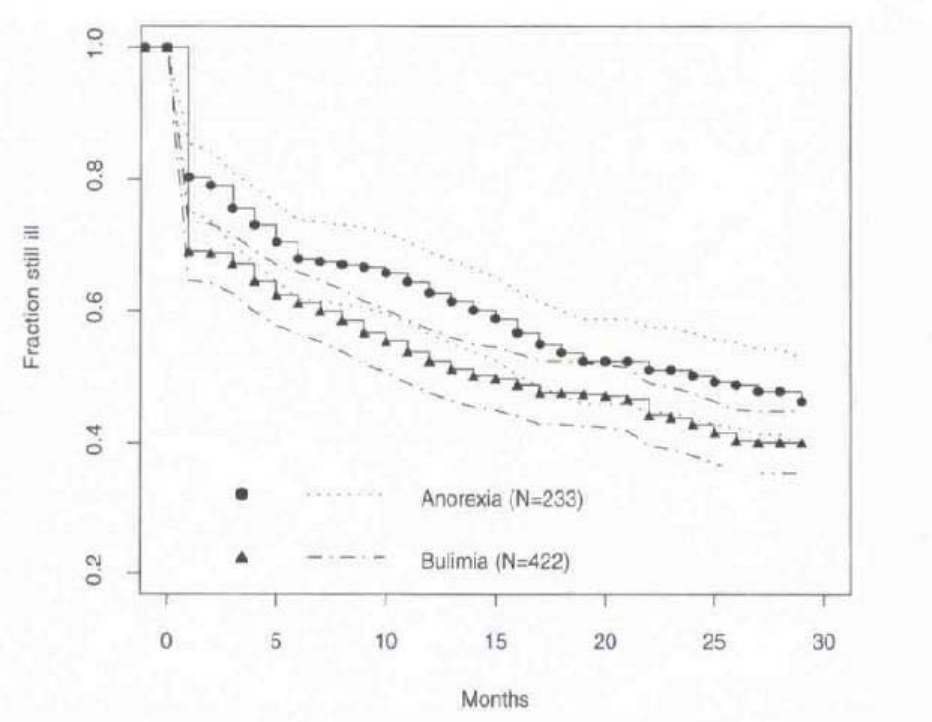


*Figure 4.* Graphical Representation of Partial Remission in Eating Disorder Treatment (Kordy et al., 2002).

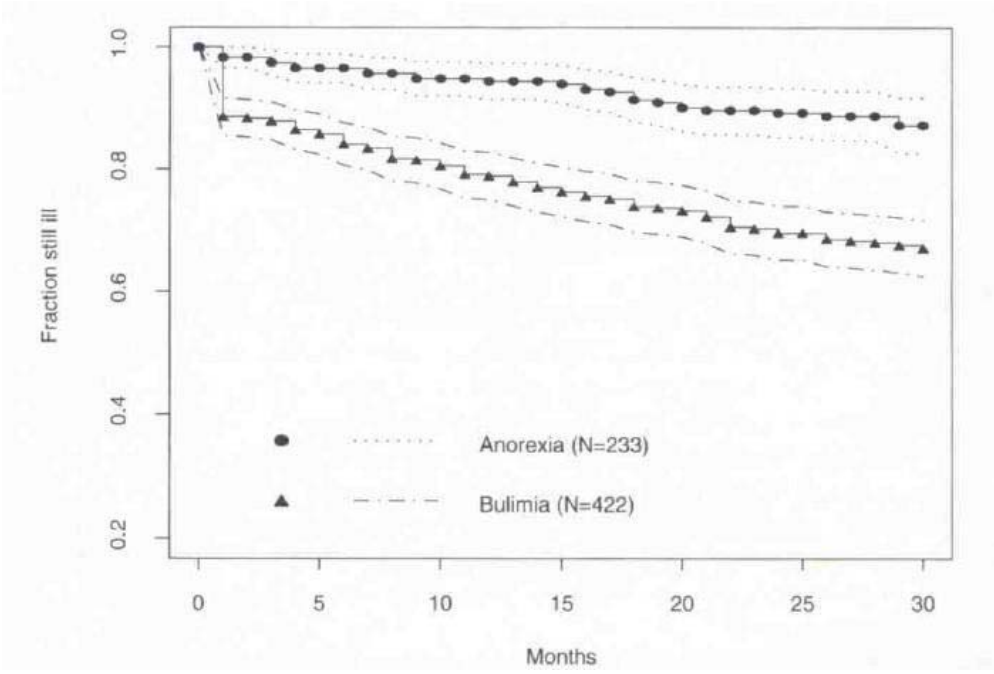


*Figure 5.* Graphical Representation of Full Remission in Eating Disorder Treatment (Kordy et al., 2002).

partial remission, the risk of relapse for these patients was 5 times higher, with risk rates steadily decreasing after this period. Bulimic participants showed no significant differences between relapse risk for partial/full remission and recovery. Interestingly, patients in full remission or recovery were significantly more vulnerable to relapse during Months 4 through 6 of their remission-recovery period. After the 6-month mark, risk of relapse decreased dramatically. Of those who obtained partial remission at the posttreatment stage, only 40% maintained this state, with 60% relapsing. Up to the 6th month, the risk for relapse for partially remitted bulimics displayed similar relapse risk percentages as for the fully remitted/recovered patients.

Kordy's (2002) work was the first attempt to operationally define the treatment trajectory for this patient population. The results indicate the difficulty in treating these patients and the necessity for long-term care, significantly highlighting the most vulnerable stages of the treatment process. Clearly, treatment of these disorders is difficult and may at times seem impossible. With such a widely varied etiology, brain abnormalities lasting past recovery, and such a large variety of risk factors involved, how can a single treatment be successfully applied to all individuals struggling with this disorder? Each individual presenting with an eating disorder may, in actuality, be unique in etiology and symptom presentation of the eating disorder. The unfortunate outcome of this is that no treatment center or clinician will be able to apply a manualized treatment to all clients and expect strong outcome results.

Dishearteningly, too many patients and clients slip through the cracks of eating-disorder treatment due to the lack of knowledge and/or research regarding the efficacious treatment of their illness. One way to shed light on the highly varied presentations of

these disorders, and to prevent patients and clients from being treated by a therapeutic modality that does not address their concerns, is to monitor treatment progress throughout the treatment program. This review has examined numerous longitudinal studies for the treatment of eating disorders, and yet no single review was anything but either pre- and/or post-treatment. A continued mechanism of evaluation of treatment progress will increase the treatment provider's ability to change treatment modalities should treatment progress not be ideal.

## Measuring Treatment Outcomes

In 1984, the World Health Organization implemented project "Health 2000," committing member states to the development of measures to assess and assure quality health care services (World Health Organization, 2001). This initiative began a serious push toward the medical field's focus on outcome measurements. As health management systems grew increasingly powerful and cost-effectiveness plans began to take priority, measures of health care quality, effectiveness, and treatment outcomes began to have serious social and political implications. Similarly, as consumers began to demand more transparency from the medical field, expectations of health care providers to provide empirically based results of reported successes also continued to rise. Today, while the numbers of outcome studies are still small, they are growing, driven by organizations as large as the National Institute of Mental Health and as small as private clinics seeking cost containment through ultimate efficiency.

Part of the increased focus on outcome measurements for psychotherapy stems from the realization that patient deterioration in psychotherapy is well documented,

although very little is known about the prevalence, rate, or magnitude of the deterioration. Some researchers posit that rates of deterioration are as low as 5% but may be as high as 15% (Lambert & Bergin, 1994). The suggested rates of deterioration are not specific to any patient population, theoretical orientation, or treatment modality, and are even consistent in group and family therapies (Mohr, 1995; Shapiro & Shapiro, 1982; Smith, Glass, & Miller, 1980). Rates of deterioration have even been noted in groups that receive no treatment (Lambert, DeJulio, & Stein, 1978). The possibility of patient deterioration is a threat to all clinicians and needs to be more fully researched in order to provide effective client care.

**Outcome Research Modalities**

The majority of treatment outcome research is designed as efficacy research. These investigations seek to minimize variance between external variables and maximize the control of internal variables. Through stringent methodologies, treatment effects are thus isolated and measurable. While efficacy research is accepted as the "Gold Standard" in this research arena (Kendall, 1998; Wells, 1999), results are typically not generalizable, given the strict controls implemented on treatment in the research environment. Due to lack of generalizability noted in efficacy research, clinicians are starting to turn to the use of effectiveness research in order to measure and assess the success of various treatment modalities. Effectiveness research relies upon routine clinical practice to assess the real-world clinical success of different treatments. The focus here is on ecological validity rather than internal validity, such as that sought in efficacy treatment. This shift in focus allows clinicians and researchers to draw

generalizable conclusions about the success of different treatment modalities. In summary, efficacy research attempts to measure the potential success of a treatment in ideal treatment conditions. Effectiveness research examines how well a treatment works in a real-world setting (Howard, Moras, Brill, Martinovich, & Lutz, 1996).

While efficacy research and effectiveness research elucidate important aspects of treatment efficacy, they both focus solely on group response to treatment (Howard et al., 1996), neglecting to maximize the effect of treatment being studied. In 2001, Lambert proposed the use of treatment outcome management procedures in order to improve treatment effects. Drawing upon the idea of patient-focused research (Howard et al., 1996), Lambert suggested that treatment response should be measured continually, with feedback provided to clinicians in order to enable clinicians to track treatment and make treatment plan modifications as necessary.

Patient-focused research seeks to answer the most pressing question facing treatment providers: Is this patient responding to this treatment? To answer this question, Howard and associates (1986; 1993) utilized dose-response and phase models of treatment effectiveness to develop a method of patient profiling designed to provide continuous feedback on individual patient treatment responses. Data pertinent to treatment success are continuously collected and modeled on a graph. This data are then compared to an expected progress pattern developed for each patient based on clinical characteristics. Patient-focused research aids in the early identification of patients who are not responding to treatment at expected levels, allowing for alterations in the treatment plan in an effort to change the treatment outcome. Lambert, Hansen, and Finch (2001) stated three defining qualities for patient-focused research: (a) evaluates

individual patient progress over the course of treatment, (b) provides regular feedback to treatment providers, and (c) "attempts to answer the question, Is this particular treatment working for this patient?" (p. 159).

Efficacy, effectiveness, and patient-focused research are all valuable contributors to the wealth of research available concerning treatment outcomes. While the three are complementary procedures, there are distinct and important differences in their processes. Although efficacy and effectiveness research are widely recognized as the foundation of evidence-based practice, they are characterized by a top-down approach to patient care. Because of their methodology, they do not allow for the consideration of patients' individual differences. Patient-focused care, on the other hand, presents a bottom-up approach to care that is driven by patient-specific information and geared toward enhancing patient outcomes.

**Patient-Focused Research Models**

To date, there are two primary patient-focused research models for measuring, or modeling, therapeutic outcome results: the Brigham Young University Model and the Stuttgart-Heidelberg Quality Assurance Model (Percevic, Lambert, & Kordy, 2004). The researchers at Brigham Young University have largely focused on predicting treatment failure. Their research has developed operational definitions for treatment success and treatment failure (Lambert & Finch, 1999; Wells, Burlingame, & Morrell, 2002). A main theoretical underpinning of the Brigham Young University model is that early treatment response predicts outcome and treatment nonresponse is indicative of treatment failure.

Therefore, through appropriate outcome measurements, clinicians can identify early nonresponders and change treatment accordingly.

Utilizing a system developed by Lambert and associates to track patient progress session to session, clinicians are able to receive a large amount of data regarding their patients' potential clinical outcomes. Lambert and colleagues engaged in a series of investigations analyzing the impact of feedback to clinicians on overall patient outcomes (Lambert, Whipple, Smart, Vermeersch, Nielsen, et al., 2001; Lambert, Whipple, Vermeersch, Smart, Hawksin, et al., 2002). These investigations found that feedback significantly improved the outcome of patients who were deteriorating in treatment or at risk for dropping out. Lambert and associates (2003) demonstrated that feedback on client outcomes, when provided regularly to clinicians, provides benefits that are not only substantial, but also replicable. One controlled investigation reduced patient deterioration rates to 13% in the clinician feedback group as compared to 21% in the treatment-as-usual group. Improvement and recovery rates were similarly affected, improving to 35% in the feedback group from a baseline of 21%.

The Stuttgart-Heidelberg quality assurance model was developed in Germany at the Center of Psychotherapy and Research, Stuttgart. The model, similar to the Brigham Young model, began using continuous treatment outcome monitoring to develop a more thorough understanding of symptom course (Kordy, Hannöver, & Richard, 2001; Kordy & Lutz, 1995). The researchers found the symptom courses of their patients graphed as linear trends moving toward improvement. Each patient presented with independent change rates and significant residual fluctuation. Spitzer (2001) defined similar courses of treatment as "random walks." The Stuttgart group adopted the random walk model as the

theoretical basis for the Stuttgart-Heidelberg quality assurance model.

The primary benefit of outcome monitoring utilizing the random walk model is that it allows healthcare providers to integrate an adaptive allocation of therapeutic resources and a cost efficiency component to the treatment of their clientele. Through an assessment of current outcome and intended outcome, clinicians are able to provide appropriate treatment, i.e., they know when to terminate therapy for a client who is no longer suffering and know when to extend treatment to those who are not responding to treatment as expected. The Stuttgart model differs from the Brigham Young University model in that it does not rely upon prior data to assess whether the patient is responding as expected. Early nonresponse to treatment is not an indication of the patient's overall outcome. Instead, the model focuses on the client in the "here and now," assessing the presence of dysfunction throughout the course of therapy. The presence of dysfunction at any given point is indicative of further therapeutic intervention being necessary.

Research supports the adaptation of therapeutic treatment time to match client distress. Percevic (2003) reported that utilizing the random walk model with continuous outcome monitoring, 85% of the experimental group achieved significant clinical improvement, as compared to 65% of the control group. In this same study, Percevic also examined therapy duration and found that with the appropriate allocation of therapeutic resources and monitoring, treatment duration could drop to as few as 19 sessions (down from 52) with the same levels of client improvement.

Although relying on a different theoretical foundation, both feedback models clearly result in benefits to both clients and clinicians. Percevic and associates (2004) hypothesized that much of the benefit noted by clients is due to an "attention effect."

Simply stated, therapists paid more attention to client progress when provided with continuous feedback about their client. Many theorists also apply the law of diminishing returns to therapy, believing that the more therapy one receives, the less effective it becomes over time, and that therapy may, if utilized for too long, actually become detrimental to the client (Howard, Kopta, Krause, & Orlinsky, 1986). Therefore, the appropriate allocation of therapeutic resources is similarly key in effective client care.

**Conclusion**

With a reported treatment deterioration rate of 5% to 15% expected across all patient populations (Lambert & Bergin, 1984) combined with the notoriously difficulty in treating eating disorders, only 13% of anorexics and 34% of bulimics achieve full remission or recovery by the end of the posttreatment stage (Kordy et al., 2002). Accurate outcome treatment measures are crucial. The varied etiology and symptom presentation of these disorders make treatment extremely difficult. Patients respond in a variety of manners to different treatment paradigms as well as at different rates (Kordy, Haug, & Percevic, 2006), further enhancing the difficulty in providing effective patient care.

Given the longstanding nature of an eating disorder, it is not unusual for a patient to progress through a variety of treatment stages as his or her treatment progresses. As patients work toward recovery, their treatment must be appropriately tailored to meet their needs. Again, one is reminded of the law of diminishing returns in therapy (Howard et al., 1986) as well as the importance of appropriate allocation of therapeutic resources. One of the most difficult aspects of treatment to assess is when it is the appropriate time to transition from one therapeutic modality to the next, be it higher or lower level care

(Kordy et al., 2006). A number of researchers have relied upon a theoretical backing similar to the Brigham Young model to assess patient outcomes and appropriate treatment transition points. For example, Agras and associates (2000) reported that patients who responded early to treatment had better outcomes than those who responded to treatment later. Outcome monitoring systems have been implemented with this ideology in mind, with an eye toward assessing when patients are not meeting treatment goals. This allows clinicians to plan interventions that inhibit patient deterioration or dropout.

Recently, Percevic and associates (2006) reported that symptom change across therapy was negatively correlated with immediate treatment responders making strong gains at first but then decreasing gains as therapy continued. Inversely, slow or non-responders at the beginning of therapy were seen as making large treatment gains later in therapy. This has important implications for the treatment of eating disorders. Often, patients considered to be non-responders are moved to a higher level of care and quick responders are held to the standard course of therapy designed by the treatment program. Both of these actions have important implications for the successful allocation of therapeutic resources and the overall treatment of patients.

Current treatment for an eating disorder is designed to meet the standard demanded either by insurance companies or by treatment providers. As providers become more accustomed to a standardized treatment course, it is often easier to simply "go through the motions" with each new patient rather than assess patients for their individual treatment needs. If, however, as research has shown (Agras et al., 2001), patients do respond to treatment at a variety of speeds and levels, then treatment does need to be

tailored to match each patient, allowing for the most effective use of therapeutic resources and the most beneficial client care. Kordy and associates (2006) called this type of treatment *Individually Tailored Service Allocation*.

Individually tailored service allocation implies that treatment outcomes cannot be known from the beginning of intake; instead, treatment must be consistently monitored and feedback supplied to the treatment team. Feedback monitoring reports allow the treatment team to regularly assess the patient's treatment progress and reassess expected outcomes as treatment continues. This feedback allows for the tailoring of individual treatment plans to consistently meet the unique and varied needs of each patient (Kordy et al., 2006). This approach allows for an integration of all of the successful therapy approaches for eating disorders and consistent monitoring and tailoring of these approaches to help patients obtain optimal treatment. Furthermore, should a patient be an early responder, patient monitoring will disrupt the cycle of the rule of diminishing returns by ensuring that a change in service provision will match patient needs.

The purpose of this investigation is to examine the effect of Individually Tailored Service Allocation on therapeutic outcomes in an eating disorder program. This study will use the Outcome Questionnaire–45 and the Outcome Questionnaire Analyst to assess patient progress and provide progress reports to therapists. In consideration of the literature reviewed, several questions present themselves: (a) Does treatment in a partial hospital program for eating disorders significantly affect global psychological dysfunction over time? (b) Does initial disease severity affect change in global psychological dysfunction over time? (c) Does provision of feedback about treatment response to therapists significantly affect global psychological dysfunction over time? (d)

Does feedback moderate the relationship between nonresponse to treatment and treatment outcome over time? (e) Does therapist receipt of treatment response feedback account for unique variance in differences in length of treatment? (f) Does feedback account for variations in the point at which global psychological dysfunction reaches its maximum ? (g) Does therapist receipt of treatment response feedback account for unique variance in the rate of change from the point of maximum global psychological dysfunction to discharge from treatment?

## Aims, Hypotheses, and Exploratory Questions

### Aim 1

To examine change in global psychological dysfunction over the course of treatment in a partial hospital eating disorder program.

**Hypothesis 1a**. A significant decrease in average global psychological dysfunction will be observed over the course of treatment for patients in a partial hospital eating disorder program.

**Hypothesis 1b**. Significant interindividual differences in intraindividual change will be observed—that is, the pattern of change of individual patients will vary relative to the sample-level trajectory of change.

### Aim 2

To examine the effect of the initial level of global psychological maladjustment on change in global psychological dysfunction over the course of treatment in a partial hospital eating disorder program.

**Hypothesis 2**. Initial disease severity will account for a significant amount of

variance in the hypothesized decrease in average global psychological dysfunction over the course of treatment. Decrease in global psychological dysfunction will be greater for patients with lower initial levels of disease severity.

**Exploratory question 2**. To what extent do low self-esteem, interpersonal problems, affective problems, perfectionism, and facets of disease severity impact change in global psychological dysfunction over the course of treatment? Of the facets shown to be related to change over the course of treatment, is the nature of the relationship such that more severe maladjustment impacts the decrease in global psychological dysfunction?

**Aim 3**

To examine the effect of enabling greater individualized tailoring of service allocation through provision of feedback about response to treatment on decrease in global psychological dysfunction over time in a partial hospital eating disorder program.

**Hypothesis 3a**. Differential treatment outcomes in the form of between-individual rates of change will be shown for patients whose therapists receive feedback such that patients whose therapists receive feedback will demonstrate a significantly greater decrease in patient global psychological dysfunction as compared to those to whose therapists' feedback is not provided.

**Hypothesis 3b**. Consistent with the notion of Individually Tailored Service Allocation (Kordy et al., 2006) in which patient information at intake is not considered wholly predictive of treatment outcome, therapist receipt of treatment response feedback will account for unique variance in between-individual rates of change.

**Hypothesis 3c**. Therapist receipt of feedback will moderate the relationship between nonresponse to treatment and treatment outcome over time such that nonrespondent patients whose therapists receive feedback will show a significantly greater decrease in global psychological dysfunction as compared to those to whose therapists' feedback is not provided.

**Aim 4**

To examine the effect of enabling greater individualized tailoring of service allocation through the provision of feedback about response to treatment on length of treatment in a partial hospital eating disorder program.

**Hypothesis 4**. Therapist receipt of treatment response feedback will account for significant unique variance in length of treatment in the partial hospital program (PHP) over and above that accounted for by disease severity at time of intake, such that total treatment length will be shorter for patients whose therapists receive feedback as compared to those to whose therapists' feedback is not provided.

## Methods and Analyses

The following section provides a thorough review of the methods and analyses utilized for this research investigation. First the demographics of the participating patients and clinicians will be presented, followed by an examination of the measures used. Finally, a comprehensive introduction to Hierarchical Linear Modeling concludes this chapter.

### Participants

A total of 58 patients receiving treatment at the Valenta Inc., Eating Disorders Program (Valenta) participated in this study. Patients with fewer than six Outcome Questionnaire (OQ) measurement occasions (i.e., a minimum of 3 weeks of treatment at Valenta) were removed from the data set. Final data analysis included 51 adult women with a primary diagnosis of either anorexia nervosa or bulimia nervosa (Table 3). Patients ranged in age from 18 to 49 ($M$ =24.14, $SD$ = 7.03). Upon intake, patients were administered a battery of assessments including the Outcome Questionnaire-45 (OQ-45) and Eating Disorders Inventory 3 (EDI-3). Demographic data were collected as part of the routine intake assessment interview. Patients were randomly assigned to one of the two treatment conditions: experimental (feedback) or control (no feedback). During the course of the study, the Outcome Questionnaire-45.2 was used to assess patient global psychological dysfunction twice a week. All patients completed the OQ-45 as part of their check-in procedures every Monday and Thursday morning throughout their course of treatment at Valenta. The OQ-45 was administered using a Dell Axim X5 Pocket PC with the Outcome Questionnaire Analyst (OQ-A) software.

Table 3

*Characteristics of Patients*

| Variables | N | % | $\bar{x}$ | SD | Range |
|---|---|---|---|---|---|
| Gender | | | | | |
| Female | 51 | 100% | | | |
| Diagnosis | | | | | |
| Anorexia Nervosa | 24 | 47.1% | | | |
| Bulimia Nervosa | 27 | 52.9% | | | |
| Age eating-disordered behaviors began | | | 14.9 | 4.7 | 8-33 |
| Duration of eating disorder | | | 9.5 | 6.6 | 1.3-27 |
| Number of outcome questionnaires administered | | | 25.8 | 11.4 | 6-53 |
| Eating Disorders Inventory– 3 Composite Scales | | | | | |
| Global Psychological Maladjustment | | | 48.2 | 6.9 | 33-61 |
| Ineffectiveness Composite | | | 48.2 | 8.2 | 31-64 |
| Interpersonal Problems Composite | | | 51.0 | 7.3 | 34-65 |
| Affective Problems Composite | | | 48.1 | 8.1 | 32-68 |
| Overcontrol Composite | | | 46.5 | 8.8 | 29-63 |

**Participating Clinicians**

Three therapists from Valenta participated in this study. Two were licensed marriage and family therapists, and the third therapist was a social worker. All therapists worked under the guidance of the Valenta Medical Director as well as with a nutritionist. The purpose of the study was explained to the participating therapists, and they were made aware that they would receive feedback on only half of their patient load. Assignment of patients to therapists was performed using routine intake procedures. It was assumed that the numbers of participants in the experimental and control conditions seen by each therapist were equal, and therefore no steps were necessary to alleviate potential therapist assignment effects. As indicated in Table 4, Therapist 3 was randomly assigned roughly 25% more patients who were in the feedback group than either Therapist 1 or Therapist 2. A multiple regression analysis was conducted to evaluate the prediction of therapist assignment and feedback group assignment on OQ change score.

Table 4

*Participant Assignment to Therapist and Feedback Groups*

| Measure | | Feedback group: No feedback Count | No feedback Row *N* % | Feedback Count | Feedback Row *N* % | Total Count | Total Row *N* % |
|---|---|---|---|---|---|---|---|
| Therapist | 1* | 9 | 52.9% | 8 | 47.1% | 17 | 100.0% |
| | 2* | 8 | 53.3% | 7 | 46.7% | 15 | 100.0% |
| | 3** | 4 | 21.1% | 15 | 78.9% | 19 | 100.0% |

* Marriage and Family Therapist
** Social Worker

$R^2 = .02$, $F(2, 48) = .49$, $p = .615$. Neither therapist assignment nor feedback group assignment significantly predicted OQ change scores. A one-way analysis of variance was conducted to evaluate the relationship between therapist assignment and the OQ change score, assessing for overall change in global psychological dysfunction at treatment end. The analysis of variance (ANOVA) was not significant, $F(1, 49) = .001$, $p = .98$, indicating that therapist assignment did not result in a significant change in global psychological dysfunction. Based on these results, it has been assumed that the unequal distribution of the feedback and no feedback group patients amongst the therapists will have no significant impact on subsequent analyses.

## Measures

### The Outcome Questionnaire-45 (OQ-45)

The OQ-45.2 is part of a quality management system developed by Lambert and colleagues (see Lambert, Hansen, et al., 2001) in 1996. It was developed in part from the work of Howard et al. (1986) examining the dose-effect relationship in therapy. Lambert

and Hansen et al. (2001) use the OQ-45 as an operationalization of patient treatment outcomes. A brief, self-report measure, the OQ-45 is used to track both the magnitude and rate of change throughout the course of treatment (Brown, Burlingame, Lambert, Jones, & Vaccaro, 2001). The questionnaire is composed of 45 questions, each based on a 5-point Likert scale (0 = never; 1 = rarely; 2 = sometimes, 3 = frequently; 4 = almost always) and results in scores ranging from 0 to 180. Patient progress, as assessed by the OQ-45, is based on monitoring three aspects pertinent to therapeutic outcomes: (a) subjective discomfort, (b) interpersonal relationships, and (c) social role performance. These three measurements result in three subscale scores. The OQ Total Score provides a global assessment of patient functioning (Lambert, Whipple, et al., 2001).

Lambert and associates (1996; 2004) found the OQ to have adequate internal consistency ($r$ = .93). The OQ also has a satisfactory test-retest value at the 3-week mark ($r$ = .85; Lambert, Burlingame, et al., 1996; Lambert et al., 2004). The OQ also has normative data based on data collected throughout the United States (Lambert, Burlingame, et al., 1996; Lambert, Hansen, et al., 1996; Umphress, Lambert, Smart, Barlow, & Clouse, 1997). The OQ-45 is indicated to be reliable, valid, and sensitive to change (Lambert et al., 1998). To facilitate the interpretation of scores, criteria for reliable and clinically significant change have been developed based on the criteria outlined by Jacobson, Follette, and Revenstorf (1984). The cutoff point for differentiating between a normal and a dysfunctional state has been set at a score of 64, and the reliable change index has been calculated to be 14 points. Based on these criteria, patient outcome can be determined by comparing OQ-45 scores to previous feedback reports, as well as through examining projected outcome curves.

**Feedback on Progress**

The OQ system enables the generation of feedback reports that can inform therapists of patient progress in treatment. Feedback reports take the form of a progress graph in which patient status is communicated using a system of color-coded feedback messages (see Figure 6). Decision rules for determination of feedback message are based on baseline OQ score, number of treatment sessions completed, and change from most recent OQ score as compared with the baseline score.

In consideration of therapeutic progress, as indicated by decision rules, one of four feedback messages may be given (Lambert et al., 2001):

White feedback: "The client is functioning in the normal range. Consider termination."

Green feedback: "The rate of change the client is making is in the adequate range. No change in the treatment plan is recommended."

Yellow feedback: "The rate of change the client is making is less than adequate. Recommendations: Consider altering the treatment plan by intensifying treatment, shifting intervention strategies, and monitoring progress especially carefully. This client may end up with no significant benefit from therapy."

Red feedback: "The client is not making the expected level of progress. Chances are she may drop out of treatment prematurely or have a negative treatment outcome. Steps should be taken to carefully review this case and decide upon a new course of action such as referral for medication or intensification of treatment. The treatment plan should be reconsidered."

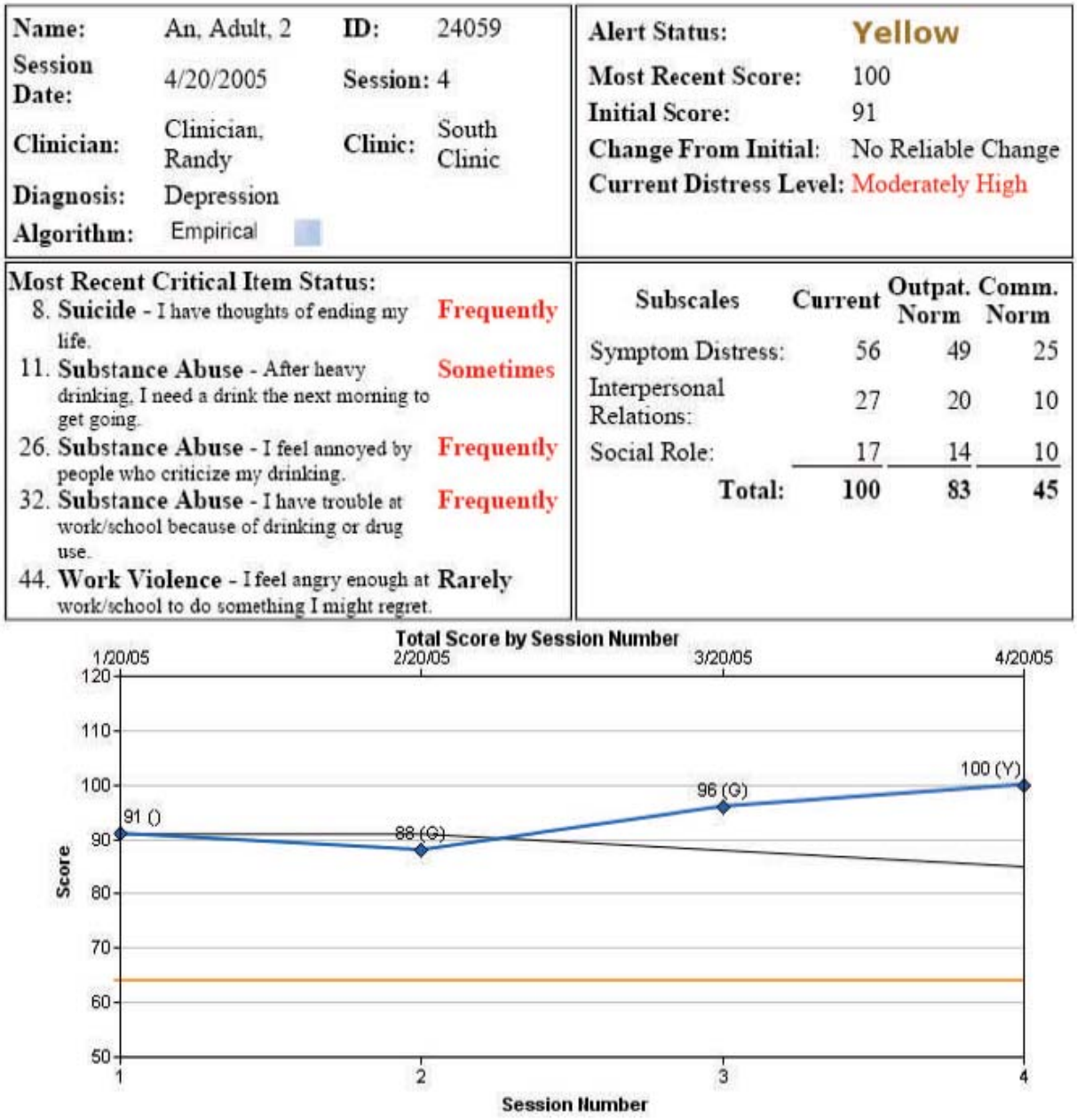


Figure 6. Sample OQ-A Feedback Report Provided to Therapists.

As a result of the ease with which the OQ can be scored and the elegant simplicity of the system of decision, OQ scores can be graphed and appropriate feedback message determined quickly after administration, enabling the provision of prompt feedback on

patient progress to therapists. Through the use of tools for administration and scoring such as Outcome Questionnaire Analyst (OQ-A) software, it is possible to generate near-instantaneous feedback.

**Eating Disorder Inventory-3 (EDI-3)**

The Eating Disorder Inventory – 3 (EDI-3; Garner, 2004) was created in 1983 to serve as a self-reported measure of attitudes and behaviors associated with bulimia nervosa and anorexia nervosa (Garner et al., 1983). In 1992, an update of the Eating Disorder Inventory (EDI) was released, the Eating Disorder Inventory – 2 (EDI – 2), which involved the addition of three provisional scales; the original eight scales remained intact. In 2004, Garner released the EDI – 3, which is an extension of the EDI and EDI – 2. The EDI – 3 includes scales and composites of eating disorder behavior that have been recently noted in the literature as being common to eating disorder symptomatology. The EDI – 3 is composed of 91 items that make up 12 scales: 3 eating disorder-specific scales and 9 general psychological scales: drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturity fears. The general psychological scales examine issues that are highly relevant to eating disorders but not specific to them. The new EDI also has the advantage of providing six composite scores; one is eating disorder-specific (Eating Disorder Risk), and the other five are general constructs (Ineffectiveness, Interpersonal Problems, Affective Problems, Overcontrol, General Psychological Maladjustment).

The development of the EDI was based on the assumption that disordered eating is multidimensional in nature. In light of this, the EDI is composed of eight subscales that

assess either attitudes and behaviors associated with eating and weight or personality and psychological characteristics commonly found in eating-disordered individuals. The EDI – 3 can provide normative information on eating-disordered individuals who are between 13 and 53. The EDI has been normed for all three DSM-IV-TR eating disorder diagnoses: anorexia nervosa (restricting type and purging type), bulimia nervosa, and eating disorder not otherwise specified. It has not been normed for obesity or binge eating disorder (Psychological Assessment Resources, Inc.). The EDI – 3 has strong test-retest stability coefficients. The median test-retest coefficient for the Eating Disorder Risk scales composite is .95, and the median test-retest coefficient for the General Psychological scales is .93 (Psychological Assessment Resources, Inc.).

## Predictor Variables

Primary independent variables will be effect of therapist receipt of feedback, response to treatment, and initial disease severity. A selected set of covariates will also be assessed.

### Feedback

To assess the effects of feedback to therapists on patient progress in treatment, patients to whose therapists feedback is given will be compared to those about whom feedback of progress is not made available. The treatment variable of feedback is dichotomous and specifies participant randomization to either the experimental condition, in which therapists were provided consistent feedback about patient progress in treatment, or the control condition, in which no feedback was given to therapists. All

patients will complete the OQ-45 on a regular basis over the course of their treatment, and all OQ results will be recorded for each participant. However, only information for patients randomized to the experimental group will be available for therapist review. For patients assigned to the experimental group, therapists will receive computer-generated feedback reports (Figure 6) the same day that the questionnaire is administered.

**Initial Disease Severity**

The Global Psychological Maladjustment Composite (GPMC) of the EDI-3 will be used as the primary indicator of initial disease severity. Exploratory analyses will also consider the extent to which initial severity on facets of Global Psychological Maladjustment is predictive of treatment outcome. Specifically, the extent to which low self-esteem, interpersonal problems, affective problems, and perfectionism are predictive of treatment outcome will be assessed using the Ineffectiveness Composite, Interpersonal Problems Composite, Affective Problems Composite, and Overcontrol Composite, respectively.

**Covariates**

Previous investigations on eating disorder outcomes and/or treatment efficacy have identified numerous variables that may impact treatment outcomes (Fairburn, Cooper, Doll, & Welch, 1999; Fichter, Quadflieg, & Hedlund, 2006; Keel & Mitchell, 1997; Lindberg & Hjern, 2003; Quadflieg & Fichter, 2003; Steinhausen, 2002). For the purposes of this study, three variables were identified following a review of the literature as having the largest possible impact on treatment outcomes. These variables are eating

disorder diagnosis (anorexia nervosa or bulimia), age of onset of eating disorder (age of initial symptom manifestation), and duration of eating disorder (number of years since symptom manifestation). In addition, the possible effects of therapist assignment will also be assessed.

## Outcome Variables

Primary dependent variables included (a) change in global psychological dysfunction, (b) rate of change in global psychological dysfunction, and (c) length of treatment period. The Outcome Questionnaire-45 (OQ-45) was used as the measure of patient global psychological dysfunction. These three outcome variables were considered across time in treatment. For purposes of these analyses, time was defined in terms of number of OQ-45 check-ins, or "measurement occasions." For this study, measurement occasions of OQ-45 data were collected biweekly from patients (Monday and Thursday). If a participant was missing data for a measurement occasion over the span of her treatment, it was assumed that the data were missing at random. The outcome variables were evaluated across periods of treatment, including over the entire treatment course (i.e., time from treatment intake to discharge).

### For Evaluation of Outcome for Total Course of Treatment

The OQ-45 data obtained across the participant treatment course yielded a hierarchically nested data structure, with the participant's biweekly OQ-45 scores nested within the randomized feedback condition. Hierarchical linear modeling (HLM; Version 6.0.6; Raudenbush, Bryk, Cheong, & Congdon, 2004) was used to evaluate participant

outcome for the total course of treatment. Following data cleaning and preparation for analyses, within-participant regressions were performed, and then the resulting regression coefficients were modeled as a function of the between-participant conditions, assignment to a feedback or no-feedback condition. In other words, the HLM analyses involved modeling the within-subjects (participant's biweekly OQ-45 scores) variance at Level 1 and between-subjects (or feedback condition) variance at Level 2 (Raudenbush & Bryk, 2002). HLM was chosen for these analyses for its superior ability to manage data that are collected longitudinally but at varying intervals and for its ability to manage multiple covariance structures (Gibbons, Hedeker, Elkin, Waternaux, Kraemer, Greenhouse, et al., 1993).

Hierarchical linear modeling typically consists of a level of within-subjects factors (Level 1) nested in another level of between-subjects factors (Level 2). Level 1 represents the relationship between some measure of time (i.e., OQ Administration Number) and the outcome variable for each participant (i.e., Total Score of OQ-45 for each administration). While it is possible to have more than one outcome variable, for the purposes of this investigation, only one outcome variable was identified to be analyzed. The analysis of each Level 1 variable resulted in regression coefficients. The within-subjects regression coefficients were estimated using the following equation:

$$\text{OQ-45 Score}_{ij} = \beta_{0j} + \beta_{1j}(\text{OQ-45 Measurement Number})_{ij} + r_{ij}.$$

In this equation, $\text{OQ-45 Score}_{ij}$ represents the participant's final OQ-45 score at discharge for participant $_j$ at OQ-45 measurement number $_I$. For each individual $_j$, the intercept is represented by $\beta_{0j}$ and the within-subjects slope is represented by $\beta_{1j}$.

Level 2 variables of the HLM analysis are the invariant predictor variables that define the relationship between the Level 1 variables as well as the estimated parameters from the Level 1 intercepts and slopes. For example, the patients who participated in this study were randomly assigned to either a control group or an experimental group. Group membership is an invariant variable (e.g., once in an assigned group, patients were never switched to the other group). As discussed in the literature review, therapist receipt of feedback on patient progress has been shown to positively influence patient outcomes in treatment (Harmon, Hawkins, Lambert, Slade, Whipple, 2005; Lambert, 2005; Lambert, Whipple, Smart, Vermeersch, Nielsen, et al., 2001; Lambert, Whipple, Vermeersch, Smart, Hawksin, et al., 2002; Lambert Ogles, 2009). If the covariate, or predictor, variable is patient assignment to feedback condition, then the results of the Level 2 equation, in which the Level 1 outcome variable (TotalScore) and time variable (SessNum) are nested, would represent the initial OQ score at intake and the rate of change throughout treatment as influenced by patient assignment to a feedback condition. Across-treatment change in global psychological dysfunction was defined as

$$\beta_{(i0-ij)i} = \beta_{0j} + \beta_{ij} + r_{ij}.$$

in which, for participant $_j$, $\beta_{0j}$ is the OQ-45 score at baseline, $\beta_{ij}$ is the final OQ-45 score before discharge, and $\beta_{(i0-ij)i}$ is the change between the scores at baseline and discharge. Within-subjects error variance is represented by $r_{ij}$. Across-treatment rate of change in global psychological dysfunction is defined as HLM slope from treatment entry (baseline or $\beta_{0j}$) to discharge (final measurement occasion or $\beta_{ij}$). The length of treatment period is defined as number of possible OQ-45 measurement occasions from treatment entry

(baseline or $\beta_{0j}$) to discharge (final OQ-45 score or $\beta_{ij}$).

Hierarchical linear modeling allows for the Level 1 regression coefficients to be modeled at another level. The Level 1 regression coefficients were modeled at Level 2 as a function of between-subjects differences based on the feedback condition and initial level of global psychological dysfunction. This second level is represented by the following equation:

$$\beta_{0j} = \gamma_{00} + \gamma_{01j} + \gamma_{02j} + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11j} + \gamma_{12j}$$

In the between-subjects model, $\beta_{0j}$ through $\beta_{ij}$ represent, respectively, the within-subjects intercepts and slopes. The means of $\beta_{ij}$ within-subjects regression coefficients are represented by $\gamma_0$s, covarying for the possible between-subjects effects of number of OQ administrations and feedback condition. The errors of the $\beta_{.j}$s were represented by their respective $u_{.j}$s and the variances are represented by the between-subjects error variances.

**HLM Intercept-only Model**

The intercept-only model, also known as the one-way ANOVA model with random effects or unconditional model, is used to establish a baseline. The Level 1 and Level 2 models are then compared to it. The intercept-only model equation is as follows:

$$\beta_{0j} = \beta_0 + r_i$$

The intercept-only model revealed an intraclass correlation coefficient of .83. Thus, 83% of the variance in OQ-45 scores was between-subjects (feedback versus no-feedback

groups), and 17% of the variance in OQ-45 scores was at the student level. Because variance existed at both levels of the data structure, predictors were added to each level individually.

## Results

This chapter begins with a review of the mechanisms utilized for data preparation and review. Next, the outcomes of the data screening and characteristics of the data utilized in the Hierarchical Linear Models are presented. This chapter closes with step-by-step presentation of the results of each Hypothesis and Exploration Question.

## Data Screening

All variables of interest were screened for multivariate assumptions. Variables that presented as significantly skewed, leptokurtic, or platykurtic were transformed logarithmically and screened again. Additionally, all variables were assessed for outliers. For the purposes of this investigation, an outlier was defined as any variable with a z-score greater than 3.29. Three individuals were indicated to have outliers on at least one measurement. Taking into consideration the already small sample size of this study, alternate methods for managing the outliers without having to remove patients from the data were considered. Based on the suggestion of Tabachnick and Fidell (1996), analyses were run with and without the patients who had the outliers to assess the impact of the outliers on the results. The outliers were not found to change the results of the analyses, and therefore no patients were removed from this study due to possessing an outlying variable within their data. The correlations between participant age, primary diagnosis (AN or BN), total number of OQ administrations, OQ change score, and EDI-3 composite subscale Global Psychological Maladjustment variables were low, thus alleviating concerns of multicollinearity (see Table 5). The correlations between participant age, duration of eating disorder, and age eating disorder began were

Table 5

*Correlation Table of Variables Used in Analyses*

| Variable | Age at intake | Duration of EDO | D | Therapist assignment | Age at EDO onset | Length of treatment | IC | IPC | APC | OC | GPMC |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Participant Age at Intake | 1.000 | | | | | | | | | | |
| Duration of Eating Disorder | .729** | 1.000 | | | | | | | | | |
| Diagnosis | .233 | .311* | 1.000 | | | | | | | | |
| Therapist Assignment | -.199 | -.100 | -.143 | 1.000 | | | | | | | |
| Age of Eating Disorder Onset | .506** | -.193 | -.046 | -.209 | 1.000 | | | | | | |
| Length of Treatment | -.100 | -.115 | .070 | -.196 | .012 | 1.000 | | | | | |
| Ineffectiveness Composite | .152 | .154 | .195 | -.048 | -.017 | -.051 | 1.000 | | | | |
| Interpersonal Problems Composite | .190 | .120 | .029 | -.106 | .108 | .042 | .754** | 1.000 | | | |
| Affective Problems Composite | .167 | .154 | .367** | -.021 | -.008 | .013 | .459** | .285* | 1.000 | | |
| Overcontrol Composite | .228 | .242 | -.011 | .027 | -.082 | .062 | .131 | .159 | .308* | 1.000 | |
| Global Psychological Maladjustment Composite | .187 | .199 | .323* | .015 | -.062 | .004 | .568** | .448** | .821** | .510** | 1.000 |

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

significant, as was expected. The correlations between the composite subscales of the EDI-3 were also significant, as expected.

Data were screened for missing data, and six instances of missing data were identified. Due to HLM not allowing for missing variables at Level 2 and age variables being Level-2 data, mean substitution was used to replace missing data. Mean substitution was utilized in order to preserve as much available data for analysis as possible and, due to the low number of missing data points, the possibility of the mean substitution resulting in a significant change in the values of the variables' correlations was low. This concern was addressed through examining correlation matrices before and after implementing mean substitution for the missing data. There was not a significant change between the correlation matrices.

Normality assumptions for all output variables were assessed through an examination of each variable's frequency plot of the distribution. Both nontransformed and log-transformed variables appeared to be approximately normally distributed. Prior to analyzing the data for HLM, each variable was assessed to ensure that it met the primary assumptions of HLM. The two key assumptions are that the dependent variable is normally distributed and that there is a linear relationship between the dependent and independent variables (Raudenbush, 2004). The variable (TotalScore), representing the unique total score of each OQ administration, violated the assumptions of normality ($p <$ .000). The variable was transformed using a loglinear transformation, which resulted in the variable being normalized and meeting the criteria for normality ($p > .05$). To assess the assumption of linearity, the session number (SessNum) variable was examined with TotalScore as the dependent variable to assess for a linear relationship. SessNum was chosen for this task as previous research has typically found number of treatment sessions to be a predictor of better treatment outcomes (Howard et al., 1986). An examination of the scatterplots representing the relationship between TotalScore and Sessnum indicated a linear relationship between the variables. Therefore, the data met the key assumptions of HLM.

The model-building process for the HLM began with an assessment of the intercept model, which was composed of only the outcome variable (TotalScore) and the time variable (SessNum). The intercept model indicated whether the patients experienced change across the course of treatment. Had the analysis of the intercept shown there to be no variability, no subsequent HLM analyses would have been conducted. The intercept model indicated significant change across the course of treatment (see Figure 7 and Table

6), and subsequent predictor variables were sequentially added to the model in order to explain the remaining variance in the relationship between the Level 1 variables. As each variable was added, the model was examined to assess for significant change in the amount of residual error. If a variable resulted in a significant reduction of residual error, it was thus assumed to account for a significant level of variance in the model. Variables that did not explain variance in the model were removed. The resulting model was composed of the independent variables feedback condition and the EDI-3 Global Psychological Maladajustment Composite score. The final model was represented by the following equation:

Level 1: $\beta_{0j} = \beta_0 + \beta_1(\text{SessNum})_j + r_i$

Level 2: $\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Feedback Group})_j + \gamma_{02}(\text{EDI-3 GPMC Composite})_j + u_{0j}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{Feedback Group})_j + \gamma_{12}(\text{EDI-3 GPMC Composite})_j$

## Analyses of Hypotheses and Exploratory Questions

### Hypothesis 1a

A paired-samples *t* test was conducted to compare subjects' OQ scores at intake to their OQ scores at the time of discharge as a measure of change in psychological dysfunction. There was a significant difference between the OQ intake scores ($M = 81.86$, $SD = 24.72$) and OQ discharge scores ($M = 72.02$, $SD = 27.45$); $t(50) = 3.16$, $p = 0.003$. These results indicate that there was significant change in subjects' intake and discharge OQ scores. Specifically, these results suggest that global psychological dysfunction significantly decreased over the course of treatment (Figure 7).

The course of symptom change over treatment course was also assessed through evaluation of significance of the HLM slope fixed-effects estimates. As indicated in Table 7, patients started treatment with an average OQ score of 80.77 and became significantly better by an average of 0.37 points per OQ-45 administration ($p < .05$).

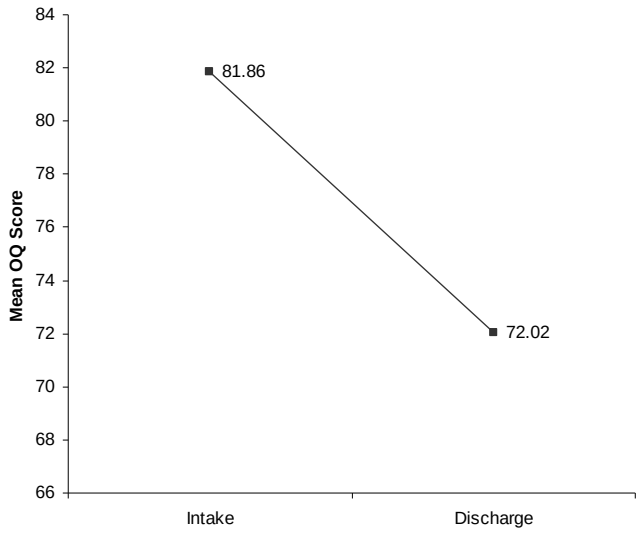


*Figure 7.* Mean change in OQ Score Across Treatment Course for All Patients.

Table 6

*HLM Parameters of Course of OQ-45 Scores Over Time in Treatment for All Patients*

| Effect | Notation | Coefficient | SE | T-ratio | P-value |
|---|---|---|---|---|---|
| Intercept | β00 | 80.77 | 3.86 | 20.70 | 0.000 |
| Slope | β10 | -0.37 | 0.15 | -2.58 | 0.016 |

**Hypothesis 1b**

Interindividual differences in intraindividual change were assessed through an evaluation of the random-effects estimates of the HLM slope at Level 1 of the hierarchical analysis. Both the intercept and number of OQ administrations were found to be reliable predictors of OQ-45 change scores; the reliability estimates equaled 0.97 and 0.84, respectively. The random-effects estimates of the HLM slope at Level 1 were significant ($p < .0005$), and therefore the null hypothesis was rejected. The intraclass correlation was examined with the following equation:

$$Y_{ij} = \mu + \alpha_i + \varepsilon_{ij}.$$

Significant interindividual differences in intraindividual change were noted; the pattern of change in individual patients did vary relative to the sample level trajectory of change. In other words, patients in the feedback group significantly varied in their change trajectories from those in the nonfeedback group (interindividual variability) but had similar change trajectories to other patients within their assigned feedback condition (Müller & Büttner, 1994). Results of the intraclass correlation indicated that 86% of the variance in patients' change in global psychological dysfunction is attributed to the between-subjects effect on mean TotalScore.

The results for Hypotheses 1a and 1b indicate that overall, participants' OQ-45 scores varied across OQ administrations, indicating that patients did experience a change in global psychological dysfunction during the course of treatment. These change patterns were indicated both interindividually and intraindividually. The model is not complete, however, as the within-subjects variance component is reported as significantly different

from zero. Other predictor variables needed to be added to the model to account for the remaining variance.

**Hypothesis 2**

A two-way repeated measures, within-subjects analysis of variance was conducted to evaluate the effect of initial disease severity at the time of intake on patient treatment outcomes. The within-subjects factors were the patient OQ scores at intake and discharge. The EDI-3 Global Psychological Maladjustment Composite and total number of OQ administrations were covariates in the analysis. The interaction and main effects were tested using the multivariate criterion of Wilk's Lambda ($\Lambda$ ). Results of the repeated measures ANOVA indicated that initial disease severity does not have a significant effect on the average change of global psychological dysfunction at Time 1, intake, and Time 2, discharge, $\Lambda = .97$, $F(1, 47) = 1.62$, $p = .21$, $\eta 2 = .03$, nor does the number of OQ administrations, $\Lambda = .99$, $F(1, 47) = .52$, $p = .48$, $\eta 2 = .01$. These results indicate that neither initial disease severity nor time in treatment have a significant relationship with a patient's change in global psychological dysfunction when one is examining patient intake and discharge outcomes.

The EDI-3 Global Psychological Maladjustment Composite scale was not indicated to have a significant effect on change in average global psychological dysfunction between intake and discharge outcome scores when examined with repeated-measures ANOVA. An analysis of the intercepts-and-slopes as outcomes model utilized GPMC to predict the Level 1 intercept and Level 1 slope of a patient's global psychological dysfunction through her course of treatment. GPMC was a significant

Table 7

*Final Estimations of Fixed Effects for the Conditional Model*

| Fixed Effect | Coefficient | SE | T-Ratio | P-Value |
|---|---|---|---|---|
| Intercept $\beta_0$ | | | | |
| Intercept1 | 83.24 | 4.36 | 19.10 | 0.000 |
| GPMC | 1.60 | 0.49 | 3.27 | 0.002 |
| FB Group | -10.13 | 6.83 | -1.48 | 0.144 |
| Slope $\beta_1$ | | | | |
| Intercept2 | -0.08 | 0.04 | -2.06 | 0.039 |
| GPMC | -0.01 | 0.00 | -2.48 | 0.014 |
| FB Group | -0.20 | 0.08 | -2.39 | 0.017 |

predictor of the Level 1 intercept *t(48)* = 3.27*, p* =.002. Overall, initial disease severity does significantly predict global psychological dysfunction at intake (Table 7).

**Exploratory Question 2**

A two-way repeated measures, within-subjects analysis of variance was conducted to assess the extent to which self-esteem, interpersonal problems, affective problems, and perfectionism moderate patient change in global psychological dysfunction between intake and discharge. The within-subjects factors were the patient OQ scores at intake and discharge. The EDI-3 composite scores for Ineffectiveness, Interpersonal Problems, Affective Problems, and Overcontrol were analyzed as covariates. The interaction and main effects were tested using the multivariate criterion of Wilk's Lambda ($\Lambda$ ). Results of the repeated-measures ANOVA indicated EDI-3 composite scores for Ineffectiveness, $\Lambda = .99$, $F(1, 45) = .54$, $p = .47$, $\eta 2 = .01$, Interpersonal Problems, $\Lambda = .99$, $F(1, 45) = .19$, $p = .67$, $\eta 2 = .00$, Affective Problems, $\Lambda = 1.00$, $F(1, 45) = .08$, $p = .78$, $\eta 2 = .01$, and

Overcontrol, $\Lambda = .99$, $F(1, 45) = .65$, $p = .42$, $\eta 2 = .01$, do not significantly moderate the patient change in global psychological dysfunction between intake and discharge. These results support the removal of these predictor variables from the HLM.

**Hypothesis 3a**

An ANOVA was conducted to determine whether feedback condition moderates the patient change in global psychological dysfunction between intake and discharge. Feedback condition was the primary predictor, and pre- to post-treatment OQ change scores was the outcome variable. ANOVA results indicate that feedback condition does not account for a significant amount of variance in the change in OQ scores administered at intake and discharge $F(1, 49) = 0.001$, $p = .98$.

**Hypothesis 3b**

To determine if therapist receipt of feedback significantly predicts treatment outcome, the Level 1 HLM slopes were examined. Level 1 of the HLM was represented by the following equation:

$$\text{OQ Score} = \beta_0 + \beta_1(\text{OQ Measurement Number}) + r$$

and Level 2 was represented by the equation:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Feedback Group})_j + \gamma_{02}(\text{GPMC})_j$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{Feedback Group})_j + \gamma_{12}(\text{GPMC})_{j.}$$

Differences in the average rate of decrease in global psychological dysfunction between

feedback conditions, after controlling for initial disease severity, were evaluated through a comparison of *t* ratio analyses. The feedback condition was a significant predictor of change in individual global psychological dysfunction across the course of treatment, $t(1309) = -2.39$, $p = .017$. These results indicate that feedback condition can significantly predict an individual's change in global psychological dysfunction over the course of treatment. Patients in the feedback condition improved their total OQ-45 score an average of 0.20 per OQ-45 administration compared to patients not in the feedback condition.

**Hypothesis 3c**

The intercepts of the Level 2 HLM variables were examined using t-ratio analyses to assess whether placement in the feedback condition impacted patient global psychological dysfunction. The analysis of the relationship between feedback group assignment and OQ-45 score at intake was not significant, $t(48) = -10.13$, $p = 0.144$ (Table 8). These results indicate that patients' OQ-45 scores at intake were not significantly different. Therefore, changes in the relationship between OQ-45 scores and feedback condition that occur during the course of treatment would be related to the impact of feedback group assignment and not caused by the initial assignment to either feedback condition.

**Hypothesis 4**

Variation in total treatment duration at Valenta attributable to therapist receipt of treatment response feedback, beyond the variation accounted for by disease severity, was assessed by an analysis of covariance (ANCOVA) with feedback condition as the

primary predictor, EDI global psychological maladjustment as a continuous covariate, and total number of OQ administrations as the dependent variable. The ANCOVA was significant $F(2, 48) = 4.18$, $p = .05$. The feedback condition was significant after controlling for EDI-3 global psychological maladjustment. The hypothesis that, after consideration of initial disease severity, patients whose therapists receive feedback have shorter treatment duration was not confirmed, however. Results of the ANCOVA indicate that patients whose therapists receive feedback have a longer course of treatment than do patients whose therapists do not receive feedback (Figure 8).

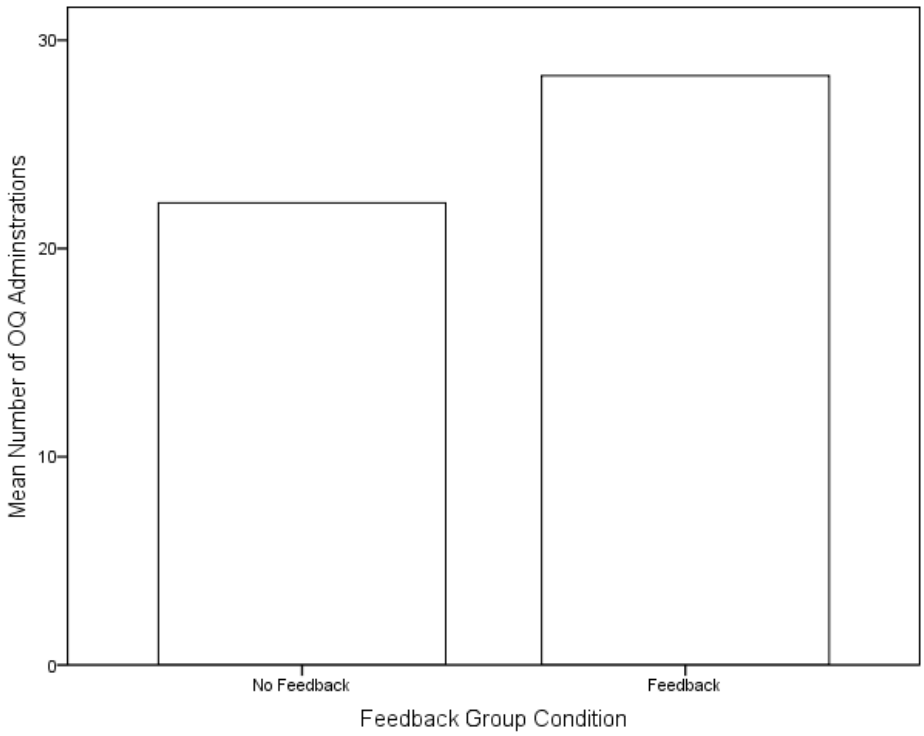


*Figure 8.* Mean Number of Total OQ Administrations by Feedback Condition.

## Discussion

This final chapter of this investigation into the impact of individually tailored service allocation opens up with a review of the importance of identifying for effective treatments for eating disorders. A discussion of the results of this investigation and their meaning and possible significance for the successful treatment of eating disorders follors. The chapter closes with important methodological considerations for this research investigations and, finally, closes with discussion of future areas for research relating to the problems presented herein.

This investigation was designed to evaluate the impact of Individually Tailored Service Allocation on eating disorder treatment outcomes. The design and implementation of successful eating disorder treatment rely not only upon understanding the risk factors and etiology of these life-destroying disorders, but also on examining the symptom course, particularly within a treatment setting. This is particularly true given the complexity of the many varied risk factors that may lead to the development of an eating disorder. As indicated by the breadth of information included in the literature review for this investigation, there have been hundreds of studies examining risk factors, etiologies, treatment strategies, treatment outcomes, and a multitude of combinations of these factors. One oft-overlooked entity, though, is the course of patient symptom presentation throughout the course of treatment.

As highlighted earlier, a wide range of treatment strategies for eating disorders exist, but the effectiveness of the treatment strategies remains low. Some researchers posit that as few as 50% of individuals with bulimia nervosa (Agras, 1993; Kordy, 2002;

Wilson, 1996) and 40% of individuals with anorexia nervosa (Kordy, 2002) will reach recovery. Such low numbers prove further investigation into existing treatment methods is needed. Complications with treatment are often related to the difficulty in defining the etiology of eating disorders as well as the role of societal and cultural factors. Researchers appear to have reached consensus regarding the importance of cognitions and interpersonal relationships in the maintenance of bulimia. However, many clinicians and researchers also report reaching an impasse when trying to identify which forms of therapy are most appropriate for anorexia. For example, Nevonen and Broberg (2006) suggested that IPT is more effective with bulimic individuals struggling with impulsivity and affective instability, whereas CBT is the treatment of choice for those whose EDO has an origin in body image, eating concerns, and a focus on weight.

Unfortunately, these different aspects of an individual's eating disorder may not emerge until late in treatment. Tobin and associates (2007) reported that 98% of clinicians who specialize in the treatment of eating disorders use an eclectic treatment approach in lieu of a standardized treatment manual. Empirically validated outcome strategies are almost impossible to assess in such a varied and diverse treatment arena. Yet with an almost 50% recovery rate for individuals with an eating disorder, it is clear that someone is doing something right in terms of eating disorder treatment. The real dilemma may not be which standardized treatment strategy a clinic or provider should adopt as the primary course of eating disorder treatment; instead, it may be how a clinician appropriately assesses which individual treatment strategies will most effectively treat this unique patient's eating disorders. Thus far, current research has only focused on outcome data or compared symptom level at intake with that at discharge.

Both strategies merely highlight success or failure in treatment and do not allow for clinician intervention based on the collected outcome data.

The results of this study indicate that variability in levels of global psychological dysfunction throughout the course of treatment appear to be the norm, rather than an exception, and this variability is related to eating disorder treatment outcomes. Interestingly, despite the widely documented recognition of several key risk factors for eating disorders as well as key predictors of treatment outcome, only initial global psychological maladjustment and feedback group assignment were indicated as significant predictors of treatment outcomes.

Global psychological maladjustment as measured by the global composite scale of the EDI-3 was a resilient predictor of treatment outcomes. Membership in the feedback group also had a significant effect on treatment, particularly over the course of treatment. Therapists may have been primed by patients' scores on the GPMC and the correlating intake OQ-45 scores, which highlighted the unique psychological maladjustment and dysfunction, respectively, for each patient. This priming could have led to greater attunement to the patient's symptom presentation and thus enhanced the possibility of a positive treatment outcome. Patients whose therapists did not receive biweekly outcome reports on their treatment progress may not have benefited from the same level of awareness provided by the global psychological maladjustment and intake OQ-45 results, as there were not regular objective reminders of the patient's current psychological status.

This current investigation is relevant to previous research, as it clearly indicates that Individually Tailored Service Allocation serves to enhance treatment outcomes in an eating disorder treatment center. These results challenge popular notions that treatment

strategies need to be manualized and streamlined in order to not only get the best results, but also identify key variables for predicting treatment outcomes. Given the widely varied and complex etiology of an eating disorder, it is not surprising that treatment trajectory and treatment outcomes vary as a function of intraindividual differences within a group, or interindividual, context. Each individual maintained a unique treatment course with widely varying scores on the OQ-45, yet all participants in the feedback condition followed the same treatment course—in other words, participants had unique treatment trajectories as indicated by the OQ-45 scores across treatment course but had significantly similar treatment trajectories within their assigned feedback condition.

The current study provides compelling evidence to suggest that eating disorder symptom manifestation and symptom regression do not follow a stable, linear course throughout treatment. Recognizing this variability and implementing treatment strategies specific to the unique symptom presentation of each patient appear to be a critical in reducing global psychological dysfunction. Counter to the existing research literature (Kordy, Hannöver, & Richard, 2001; Kordy & Lutz, 1995), the current study found that when outcome data are examined in a purely linear fashion, important fluctuations in treatment course are lost.

As highlighted by the notable discrepancy in the results for Hypothesis 2, the choice of an appropriate statistical procedure has an irrefutable ability to drastically shift the outcome of an investigation. Similarly, the choice of treatment methodology and level of Individually Tailored Service Allocation have the irrefutable ability to drastically shift treatment outcomes, as clearly indicated by the results of the investigation. The notion that eating disorder treatment is a linear process is clearly disputed by the results for

Hypothesis 2. Results examining the impact of global psychological maladjustment on the change in OQ scores from intake to discharge were not significant ($p$ = .21), nor was number of OQ administrations ($p$ = .48). According to the results of the within-subjects, repeated-measures ANOVA, neither initial disease severity nor time in treatment have a significant relationship with a patient's change in global psychological dysfunction when one is examining patient intake and discharge outcomes. This statistical procedure represents a one-size-fits-all-approach to data examination, which is unfortunately the approach taken by managed care providers and insurance agencies when parsing out coverage for eating disorder treatment. Successful eating disorder treatment may hinge on regularly examining patient progress throughout the course of treatment. Typically, eating disorder treatment is designed to meet the standard demanded by insurance companies or by the treatment providers. If, however, patients do have unique treatment trajectories, then treatment does need to be tailored to match each patient (Agras et al., 2001), allowing for the most effective use of therapeutic resources and the most beneficial treatment. The results for Hypothesis 4 provide support for individually tailored treatment allocation. These findings indicated that patients in the therapist feedback condition actually had a longer treatment course than those in the nonfeedback condition. This outcome is not likely to be looked upon favorably by healthcare organizations that are constantly seeking to reduce costs related to patient care. It will be important for researchers to continue to build the case that while longer treatment is more costly in the short run, the costs will be significantly lower if treatment is successful and future partial hospitalization or higher level care is avoided in the future.

## Methodological Considerations

There are several limitations to this study that warrant discussion. First, with a small sample size and lack of power, it may not have been possible to detect the psychological predictors that significantly predict rapidity of weight gain. Additionally, this study included a mostly homogenous sample of participants with regard to gender and ethnicity. While this sample is relatively consistent with those of other studies that have evaluated eating disorder populations, the results of this study are not generalizable to males and people of non-Caucasian ethnicities. Furthermore, all psychological measures consisted exclusively of self-report questionnaires. Due to the denial and ambivalence involved in this disorder, participants may have underreported their symptoms and difficulties. While patients were assured that their measures would remain confidential, they may have responded with the belief that their answers could have a possible impact on the length of time they spend in treatment.

Another methodological limitation of this study pertains to the use of treatment duration as a measure of outcome. Although Valenta delineated similar treatment goals and discharge criteria for patients, a patient's length of time in treatment is subject to external factors such as insurance, family concerns, financial stress, etc. Therefore, treatment duration is dependent on the physical and psychological health of the patient and includes external variables that are impossible to control methodologically. This investigation did not examine external factors that could have impacted treatment duration.

## Areas for Future Research

It is recommended that the current study be continued in order to increase sample size and strengthen the results of the investigation. It is the hope of this researcher that upon completion of this investigation, the results are utilized to inform treatment practices and support patients in receiving appropriate care. In particular, a significant amount of time and resources are utilized with the hope or aiding individuals with an eating disorder to achieve remission and recovery. The results of Hypothesis 4, which indicated that patients in the therapist feedback condition actually had a longer treatment course than those in the nonfeedback condition, are particularly provocative. This outcome is not likely to warrant applause from healthcare organizations that are constantly seeking to reduce costs related to patient care but may illuminate why current remission and reocovery rates are so dismal. Future research ought to continue to build the case that while longer treatment is more costly in the short run, in consideration of long-term outcomes, the costs will be significantly lower if treatment is successful and future partial hospitalization or higher level care is not warranted.

The results of this investigation are easily generalized to the demographic population commonly served in eating-disorder treatment programs. It is important for future research to evaluate how treatment outcomes may differ in a population of minorities or males with eating disorders. A more diverse sample will also enable researchers to examine additional cultural and societal factors not considered in this investigation and their impact on eating-disorder treatment outcomes. Important psychological differences between ethnicity and gender could be vital in determining methods of eating-disorder intervention and treatment focus.

The data collected in this investigation adequately assessed patient progress throughout the course of treatment. It would be fruitful to examine the subscales of the OQ-45 throughout the course of treatment in order to assess for emerging interpersonal concerns, social role perceptions, and symptom distress (a measure of depression and anxiety; Lambert, Hansen, et al., 2001). This would provide both clinicians and researchers with additional information needed to examine the relationships between these different facets and treatment outcomes. Examining the OQ-45 subscales throughout the course of treatment may also provide clarification as to why several of the variables that have been previously found to significantly predict treatment outcomes (i.e., age of eating disorder onset, duration of eating disorder, etc.) were not found to be significant in this model. Along these same lines, the OQ-45 is a general measure of global psychological dysfunction and is not an outcome measure specific to the assessment of eating-disorder symptoms. Using a standardized measure for assessing eating-disorder symptoms at regular intervals during the course of treatment along with the OQ-45 would provide additional, crucial information about the change trajectory of eating-disorder symptoms throughout the treatment course as well as serve as another invaluable resource for clinical intervention.

Future research could also involve continuing to follow a patient's treatment using the OQ-45 after she has completed treatment with Valenta and stepped down into a lower level of care such as outpatient individual therapy. Collecting this additional information, post-partial hospitalization, would enable researchers to assess changes in additional psychological variables as well as track whether improvements made during the course of treatment are maintained.

Finally, future research should also take into consideration the effects of other variables that have been defined in the literature as predicting treatment outcome. For example, 11 variables have been identified as primary predictors of treatment outcomes for individuals with anorexia nervosa: duration of the eating disorder, age of onset, family of origin, age at onset of menstruation, sexual problems, psychiatric comorbidity, perfectionism, impulsivity, self-evaluation, extroversion, and low body weight (Fairburn, Cooper, Doll, & Welch, 1999; Keel & Mitchell, 1997; Lindberg & Hjern, 2003; Quadflieg & Fichter, 2003; Steinhausen, 2002). These variables could possibly be assessed in conjunction with the OQ-45 biweekly administration or, should that prove too burdensome, at both intake and discharge, thus allowing for an assessment of overall change in symptom presentation throughout treatment.

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