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The Role of Psychological Flexibility in Eating Disorders in a Residential Treatment Sample

Phillip Ryan Mitchell

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THE ROLE OF PSYCHOLOGICAL FLEXIBILITY IN EATING DISORDERS
IN A RESIDENTIAL TREATMENT SAMPLE

by

Phillip Ryan Mitchell

A thesis submitted in partial fulfillment
of the requirement for the degree

of

MASTER OF SCIENCE

in

Psychology

Approved:

Michael P. Twohig, Ph.D.
Major Professor

Ginger Lockhart, Ph.D.
Committee Member

David Stein, Ph.D.
Committee Member

Mark R. McLellan
Vice President for Research and
Dean of the School of Graduate Studies

UTAH STATE UNIVERSITY
Logan, Utah

2013
ABSTRACT

The Role of Psychological Flexibility in Eating Disorders in a Residential Treatment Sample

by

Phillip Ryan Mitchell, Master of Science

Utah State University, 2013

Eating disorders have a dramatic effect on the lives of people who struggle with them, including cardiovascular and gastrointestinal problems and death. In addition, individuals with these diagnoses often struggle with comorbid diagnoses such as depression, anxiety, and substance abuse or dependence. By building on a conceptualization of eating disorders as a means to dysfunctionally regulate negative affect and escape unwanted thoughts, psychological flexibility, the ability of a person to contact unwanted thoughts or feelings, and behave without escape or avoidance, is investigated as a tool for treatment. Additionally, because quantitative analysis of the construct of psychological flexibility has often employed the use of college samples, this study employed a sample of participants drawn from a residential treatment facility dedicated to the treatment of eating disorders. Through regression and modeling, psychological flexibility
demonstrates its utility in the treatment of eating disorders through its relationship with body dissatisfaction and quality of life.

(48 pages)
Eating disorders (anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified) have a dramatic effect on the lives of people who struggle with them. Individuals struggling with these diagnoses often suffer from cardiovascular problems, hormonal irregularities, and gastrointestinal issues, such as wearing away of the esophageal lining. The biggest concern to clinicians is mortality resulting from physical complications related to an eating disorder or suicide. There is a growing body of research that attempts to describe the cognitive and emotional aspects of these diagnoses. Research has illustrated how suppressing emotions and thoughts, or avoiding situations that may make these thoughts and emotions arise, can function to maintain or increase the severity of these eating disorders. Therefore, a useful tool for clinicians would be a therapy that directly targets these types of cognitive/emotional avoidance, such as psychological flexibility. Psychological flexibility is the ability of an individual to contact unwanted thoughts and feelings and behave in a manner that is valued, even in the face of these negative experiences. The vast majority of research that has looked at the
relationship of psychological flexibility and eating disorders has used typical college samples to gather data, with encouraging results. However, it is important to examine whether these relationships work within those struggling the most with eating disorders. This study employed a sample of individuals from a residential treatment facility—where people live for several months and focus exclusively on getting treatment for their eating disorders. The results from this study demonstrate that psychological flexibility is useful for treating eating disorders, by influencing an individual’s body dissatisfaction and quality of life (two critical outcome measures of eating disorder severity).
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Phillip Ryan Mitchell
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CHAPTER I
INTRODUCTION

Eating disorders are an insidious problem in Western culture and a growing problem world-wide. The National Comorbidity Replication Survey (Hudson, Hiripi, Pope, & Kessler, 2007) report lifetime prevalence rates for eating disorders are between 0.6 and 4.5% and they are often comorbid with Axis I disorders such as depression and anxiety (Hudson et al., 2007). Additionally, these disorders have high mortality rates related to suicide and related pulmonary conditions and have negative physiological effects associated with the gastrointestinal and endocrine system (Berkman, Lohr, & Bulik, 2007; Birmingham, Su, Hynlisky, Goldner, & Gao, 2005; Mitchell & Crow, 2006). With these negative effects and comorbidity, it is necessary to understand possible mechanisms for the development and maintenance of these disorders so that treatments can be effectively developed and implemented.

Several researchers are now pointing to eating disorders as dysfunctional emotion regulation behaviors, which attempt to control or get rid of negatively evaluated internal states (Gilboa-Schechtman, Avnon, Zubery, & Jeczimien, 2006; Hayaki, 2009; Haynos & Fruzetti, 2011; Smart & Wegner, 1999). This research pointed out that overt behaviors related to self-imposed starvation and the binge-purge cycle were representative of individuals who were experiencing distressing events without functional behaviors to remediate these negative affect states. While these dysfunctional behaviors can be viewed as effective ways of controlling unwanted inner experiences, they come at a high cost, which is evident in the ill-health effects and ultimate mortality (Mitchell & Crow, 2006).
Unfortunately, there exists a paradoxical effect related to these dysfunctional control behaviors in that they may increase the frequency or intensity of the distressing thoughts and feelings that the individual is trying to escape from, then intensifying their application of these control behaviors to achieve escape or avoidance (Wegner, Schneider, Carter, & White, 1987).

The nature of dysfunctional emotion regulation and its relationship to eating disorders pathology makes it prudent to investigate direct attempts at interrupting that mechanism. Psychological flexibility is a construct that is being researched precisely this domain. Psychological flexibility is the ability of an individual to engage in behaviors that serve valued ends, despite how uncomfortable the situation they find themselves (Hayes, Luoma, Bond, Masuda, & Lillis, 2006). Within the construct of psychological flexibility there are two components that are crucial for understanding the construct: experiential avoidance and valued living. Experiential avoidance is another form of dysfunctional emotion regulation that works to avoid both the internal experiences described previously, as well as the environmental contexts that occasion those experiences (Hayes et al., 2006). In only using avoidance as a means for coping with unwanted internal experiences and the places and situations that give rise to them, the individual’s behavioral repertoire is often narrowed in ineffective ways. Psychological flexibility and experiential avoidance are inversely related, however they are not simply opposing constructs. Valued living is another important characteristic that is understood to be a verbally constructed appetitive phenomena that directs behavior across contexts. If experiential avoidance is the stick that motivates escape behavior, then valued living is
the carrot that motivates approach.

Given the dysfunctional emotion regulation associated with eating disorder diagnoses, psychological flexibility is considered a mechanism that may help in alleviating eating disorder symptoms. Employing a wide range of methodologies, researchers have begun to gather data that sheds light on the relationship between psychological flexibility and eating disorders. Research has shown that psychological flexibility is negatively correlated with elevated scores on eating disorder severity, and those with eating disorder diagnoses indicate increased experiential avoidance versus a control sample (Rawal, Park, & William, 2010). Additionally, psychological flexibility is negatively correlated with eating disorder related cognitions (Masuda, Price, Anderson, & Wendell, 2010). Masuda and colleagues also performed a series of hierarchical regressions that support the mediating nature of the construct, which is consistent with its description from Hayes and colleagues (2006); and this mediating relationship has been bolstered in additional research with eating disorder symptomology (Masuda, Boone, & Timko, 2011).

If psychological flexibility does have a relationship with respect to psychological well-being, then it is a crucial feature in the treatment of those with eating disorders, especially when these disorders have dysfunctional emotion regulation as a core feature. In helping clients understand new ways in which they can interact with unwanted internal and external experiences, which are consistent with valued living, therapists gain a valuable tool in their therapeutic practice. However, to date, the majority of the research into the relationship between psychological flexibility and eating disorders has employed
college samples involving individuals who may or may not have a diagnosable eating disorder. It is critical to understand this relationship in a sample of individuals with an eating disorder diagnosis to assess the value of using it as a treatment target. This study will examine three interrelated constructs related to eating disorders: psychological flexibility, body dissatisfaction, and eating disorder quality of life at both admission and discharge from an eating disorder treatment facility. First, it is important to look at the relationships these constructs have by generating correlations. Second, simple regressions will be performed that will use psychological flexibility to predict scores of body dissatisfaction and eating disorder quality of life. Finally, by controlling for admission levels of psychological flexibility, body dissatisfaction and eating disorder quality of life, two models will be generated examining the ability of psychological flexibility at discharge to account for variance seen in body dissatisfaction and eating disorder quality of life at discharge. Results from this study will help shed light on the utility of targeting psychological flexibility in the treatment of eating disorders to have an effect on both body dissatisfaction and quality of life.
CHAPTER II
LITERATURE REVIEW

The current version of the American Psychiatric Association’s (APA) *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; APA, 2000) has three diagnoses in the eating disorder section: anorexia nervosa (AN), bulimia nervosa (BN) and eating disorder not otherwise specified (EDNOS). AN is primarily characterized as severe restriction of caloric intake to the point that an individual is 85% of their ideal bodyweight. BN is characterized as a cyclical disorder with periods of *binging*, where individuals may consume several thousand calories in a single event, and *purging*, a compensatory behavior where the individual expels the content of the binge cycle. EDNOS is a very loosely defined disorder that consists of subclinical (and yet still severe) levels of the other two diagnoses. For example, an individual struggling with AN may be well under their ideal bodyweight, but the DSM diagnosis dictates an amenorrheic period of three months and that individual may not meet that criteria. Additionally, researchers have begun investigating *binge-eating disorder*, which is associated with the BN cycles, but lacks the compensatory purging cycle and is not included as a diagnosis in the DSM, but is considered for future versions of the DSM (Hudson et al., 2007). According to the National Comorbidity Survey Replication (NCSR), lifetime prevalence estimates for diagnoses of an eating disorder are 0.6% for AN, 1.0% for BN, 2.8% for binge eating disorder, 1.2% for subthreshold binge eating disorder, and 4.5% for any binge eating disorder (Hudson et al., 2007).
Negative Effects and Comorbidity

The biggest concern to clinicians and physicians is the possibility of mortality related to their client’s eating disorder. Birmingham and colleagues (2005) reported standard mortality ratios of 10.5% in those with anorexia nervosa. However, this is usually the result of several years of medical and psychological complications that accompany eating disorders. Health problems related to the gastrointestinal tract, the endocrine system, cardiovascular and pulmonary conditions are related to either restrictive dietary practices related to AN, or the binge/purge cycle related to BN and binge eating disorder (Mitchell & Crow, 2006). Additionally, Berkman and colleagues (2007) reported that AN has the highest mortality rate among any psychiatric disorder. Their meta-analysis reported that 22% of individuals with AN indicated that they had attempted suicide at some point in their life.

Additionally, these eating disorders are often comorbid with several Axis I disorders. The NCSR found that of those diagnosed with an eating disorder, between 56% and 94.5% (depending on participant diagnosis) met criteria for an additional Axis I disorder (Hudson et al., 2007). For example, of those who met criteria for BN, 80.6% also met criteria for an anxiety disorder in their lifetime. Lifetime comorbidity for mood and impulse-control disorders were also elevated across diagnoses, where they report that of those diagnosed with AN, 39.1% met criteria for a diagnosis of major depressive disorder (lifetime comorbidity) as well as 50.1% of those diagnosed with BN. However, a causal pathway was not determined in this study and it remains unclear if these disorders develop simultaneously and share a common diathesis, or if one is a result of the other.
Individuals with eating disorders may also engage in deliberate self-harm, non-suicidal self-injury, and substance abuse or dependence as a means of dysfunctionally regulating emotions. Muehlenkamp, Peat, Claes, and Smits (2012) reported 19.4% of respondents endorsed a history of disordered eating and nonsuicidal self-injury. They also reported that those that reported both self-injurious behaviors and disordered eating expressed increased levels of depressive symptoms and emotion dysregulation. Ruuska, Kaltiala-Heino, Rantanen, and Koivisto (2005) reported that in a sample of adolescents seeking treatment for eating disorders or eating problems, 14.7% of those diagnosed with AN and 38.1% of those with BN had engaged in deliberate self-harm. Franko and colleagues (2005) reported a study conducted over the course of almost 9 years comprised of 136 females diagnosed with AN and 110 diagnosed with BN. Their study found that 27% of the participants endorsed a lifetime history of alcohol use disorder (AUD), and that over the course of the study, another 10% developed AUD. In another study comprising of the same participants, Herzog et al. (2006) reported that 17.1% of participants had a lifetime history of drug use disorder. Krug and colleagues (2009) conducted a study with three types of AN: the restrictive (AN-R) and binge-purge (AN-BP) subtypes, as well as a subsample who had transitioned from AN to BN (AN-Cross). This study reports that 8.8% of those with AN-R, 26.3% of those with AN-BP, and 35.2% of those with AN-Cross endorsed lifetime substance abuse. In general, these authors conclude that each of these dysfunctional behaviors may function as a means to regulate negatively valenced internal events.
Development and Maintenance of Eating Disorders

With such high rates of comorbid anxiety and mood disorder diagnoses there is strong support for increased negative affect in those with eating disorders. This leads some researchers to suggest that eating disorders develop as a means of dysfunctional emotion regulation. For example, Haynos and Fruzetti (2011) provided a model of AN describing how those struggling with this eating disorder present with heightened reactivity to food and body related stimuli. This reactivity is compounded by a perpetual state of starvation and fatigue from exercise. The authors described how when presented with emotional stimuli, individuals struggling with AN reacted in a dysregulated manner. These reactions include over exercising and restricting as compensatory behaviors that negatively reinforced dysregulated emotional reactions. Furthermore, individuals with eating disorders broadly reported having a diminished ability for proper emotion regulation (Gilboa-Schechtman et al., 2006). These authors described deficient emotional processing as a key feature that went across eating disorder diagnoses. They report that participants with AN and BN displayed similar levels of diminished emotional awareness.

In BN, Hayaki (2009) argued that the binge-purge cycle that typifies this disorder is a means of controlling negative affect. The author found that when global emotion dysregulation was controlled, negative reinforcement expectancies related to eating are unique predictors of BN symptoms, especially in the presence of negative affect. Thus, the binge-purge cycle is a negatively reinforced reaction to negative evaluated internal stimuli. Additionally, if emotion regulation is a feature that is seen across eating disorder diagnoses, then there should be a comparable method of controlling negative affect in
AN. Polivy and Herman (2002) described the restricting feature of AN as a means for controlling negative thoughts and emotions by focusing on weight and body image. Peñas-Lledó, Vaz Leal and Waller (2002) reported that overexercising, a common feature associated with AN, is an additional means of controlling negative affect functioning to take awareness away from negative internal states and refocus on controllable external behaviors.

Emotion and/or thought suppression represents another means through which individuals with eating disorders may dysfunctionally regulate affective states (Peñas-Lledó et al., 2002). Thought suppression is a construct pioneered by Wegner and colleagues (1987) and is described as a private event control technique, where the individual takes an active role in trying to avoid thoughts of a specific nature, but has a paradoxical effect in that it increases preoccupation of that specific thought. Thought suppression research has been applied to a wide variety of psychopathology, and has grown to be considered a transdiagnostic feature of several dysfunctional behaviors and psychopathology (Wenzlaff & Wegner, 2000). When applying the concept of thought suppression to eating disorders, the effect of trying to suppress thinking about food or body image issues, will exacerbate those same thoughts, with distressing results. Shouse and Nilsson (2011) reported that college women who were more emotionally aware and engage in self-silencing (a form of thought control) related to eating behaviors were more likely to report disordered eating. Additional research has demonstrated that self-silencing may function as a mediating mechanism where thin-body idealization leads to body dissatisfaction and disordered eating (Morrison & Sheahan, 2009). Finally, Smart
and Wegner (1999) compared individuals with disordered eating characteristics with those without those characteristics and report higher levels of thought suppression, as well as the paradoxical effect of thought intrusion, in the participants with those characteristics.

Related to the concept of emotion or thought suppression, alexithymia is a construct that describes the deficient ability to describe, differentiate, or process emotions (Taylor, Ryan, & Bagby, 1986). Corcos and colleagues (2000) reported that female participants diagnosed with AN endorsed higher rates of alexithymia (56.3%) than those with BN (32.3%), and that both groups were higher than controls (12.2%). When researchers controlled for depression, the levels of alexithymia did not vary between eating disorder type. Speranza, Loas, Wallier, and Corcos (2007) concluded in a 3-year study of those with eating disorders that those with greater alexithymia are more symptomatic at follow-up, and alexithymia is stable over time. It may be that this emotional deficiency functions as a precursor to thought or emotion suppression, but more research will have to illuminate any longitudinal relationship.

Considering the data that points to increased dysregulation of emotive reactions and decreased emotional awareness, it would seem prudent to target an individual’s willingness to engage with emotionally distressing thoughts and feelings, as well as stimuli and contexts that elicit those responses as a form of treatment. By engaging with negative affect in a more healthy way, it may provide a means for effectively treating eating disorders and interrupting the chain of negative outcomes, both psychological and physiological, associated with these disorders.
Psychological Flexibility

One construct that specifically targets the control agenda and other forms of dysfunctional regulation of internal experiences is psychological flexibility (PF), which is “the ability to contact the present moment...and to change or persist in behavior when it serves valued ends” (Hayes et al., 2006, p. 7) and is understood to be a variable, trait construct indicating psychological health. This contact with the present moment includes both public and private events and is further described with relational frame theory (RFT; Hayes, Barnes-Holmes, & Roche, 2001). PF as a variable exists on a spectrum where on one end (flexible), the individual is able to behave in a manner consistent with valued ends and, on the other, the individual will behave so that they can escape these events, both public and private.

Moreover, as a consequence of this type of responding, individuals may engage in a form of dysfunctional regulation of private events in which they will attempt to control the form and/or frequency of those events as well, otherwise known as experiential avoidance (EA; Hayes et al., 2006). The person engaging in experiential avoidance may avoid the external contexts that give rise those private events to avoid experiencing them. The final piece of the definition of PF is concerned with behaving according to personal values. Values are defined as verbally derived distal reinforcers that people are willing to work towards, but never achieve. For example, a person may value being honest. The person must constantly work towards that value and behave in a way consistent with it, as opposed to reaching a state where they have attained honesty. Taking all of this into consideration, PF is the process of choosing behaviors that are consistent with an
individual’s values, in the presence of specific internal and external stimuli, regardless of
the verbally conditioned properties of those stimuli.

**Psychological Flexibility and Eating Disorders**

PF has demonstrated relationships to other outcome variables related to
psychopathology through correlation, mediation and other statistical and empirical
analyses (Ruiz, 2010). PF, and its relationship to eating disorders, has also been examined
in several different ways. Additionally, because PF is the construct that an ACT
intervention targets, it has also been studied in the form of treatment outcome
interventions and it has demonstrated effectiveness as a targeted treatment construct.

In the past, researchers have used the Acceptance and Action Questionnaire
(AAQ) and its various forms to measure PF (Bond et al., 2011). When reviewing research
that has used the AAQ, some researchers have indicated that their use of this
questionnaire is to measure experiential avoidance and some indicate they are measuring
PF; it is really a semantic issue dependent on the intention of the researcher. The theory
underlying these constructs dictates that an individual who demonstrates greater PF will
show lesser experiential avoidance and vice versa. Whether PF or experiential avoidance
is discussed is a result of what the researcher is trying to emphasize.

Recently Rawal and colleagues (2010) examined the relationship that experiential
avoidance has with eating disorder symptoms by means of self-report questionnaires in
two studies. The first study utilized a sample of 177 university students and reported that
AAQ-II scores had a correlation of $r = .38$, $p < .001$ with the Eating Disorder
Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994), which is a self-report measure of eating disorder symptomology. After determining correlation, the AAQ-II was used in a regression and they found that eating disorder levels, as well as anxiety, depression and gender, accounted for 36% of the variance seen in experiential avoidance. The researchers went on to study experiential avoidance in a clinical sample ($N = 13$) diagnosed with AN. This sample was compared to a control ($N = 13$) and the results found that experiential avoidance was greater in the clinical sample, compared to the control, $t(1,24) = 7.40, p < .001$. These results indicated that experiential avoidance did indeed have a relationship with eating disorder pathology and that in comparison with a control sample, those diagnosed with AN show greater levels of experiential avoidance.

However, the way that experiential avoidance is discussed in this paper is somewhat contrary to the way it is described by Hayes and colleagues (2006). In this research, the authors described variations in experiential avoidance as a result of the variations in eating disordered behaviors, not the other way around. It is more common to use eating disorder behaviors as the outcome variable and experiential avoidance as a predictor of eating disorder behaviors. It is more likely to see the research question as how do variations in experiential avoidance impact eating disordered behaviors, as opposed to how do changes in eating disorder behaviors impact PF.

Masuda and colleagues (2010) examined how PF related to eating disorder related cognitions. Utilizing a college sample of 440 students with the AAQ-16, Bond and Bunce (2003) found that PF negatively correlated with general psychological ill health, emotional distress (both with $r’s = -.46, p < .01$) and conviction of disordered eating-
related cognitions ($r = -.32, p < .01$). Furthermore, these researchers went on to report a series of hierarchical regressions in which PF was added in a second step. PF was able to account for increased variance in their models in both psychological ill health ($R^2\Delta = .14, p < .001$) and emotional distress ($R^2\Delta = .15, p < .001$). These data again demonstrated the relationship that PF shared with eating disorder symptoms (specifically cognitions and distress), but went on to show that PF had explanatory power in describing the eating disordered individual.

Masuda and colleagues (2011) further examined the role that PF plays in eating disordered symptoms. To do this, they recruited a college sample of 209 and asked them to complete a series of self-report measures including the AAQ-16 (Bond & Bunce, 2003) and measures related to eating disorder pathology and cognitions. Their results showed a negative correlation between PF and eating disorder pathology, as measured by the Eating Attitudes Test (EAT-26) (Garner, Olmstead, Bohr, & Garfinkel, 1982), $r = -.33, p < .01$. PF was also negatively correlated with disordered eating cognitions, as measured by the Mizes Anorectic Cognitions Questionnaire—Revised (MAC-R; Mizes et al., 2000), $r = -.46, p < .01$. These researchers then went on to hypothesize and test PF’s ability to mediate the relationship between self-reported self-concealment with eating disorder pathology and, separately, with disordered eating cognitions. The Self-Concealment Scale (SCS; Larson & Chastain, 1990) measures an individual’s tendency to hide negatively evaluated facts about themselves. PF demonstrated a mediating relationship, using the Baron and Kenny (1986) method, between self-concealment and eating disorder pathology, as well as disordered eating cognitions. This mediating
relationship is predicted in PF theory (Hayes et al., 2006). Note that PF is described as an ubiquitous construct that accounts for why private events arise (such as negative body image appraisals) for all people, but it is only those who are low in PF who will act with pathological eating disordered behaviors to control those unwanted thoughts and feelings. Individuals with greater PF may have the same type of negatively evaluated private events, but will choose to act in ways that are concordant with their values.

If PF shares these relationships with eating disordered pathology, then it should be possible to demonstrate the effectiveness of targeting PF specifically as a treatment for eating disorders. To date, there are a few empirical studies that have examined this hypothesis.

**Targeting Psychological Flexibility**

Juarscio, Forman, and Herbert (2010) conducted a small trial that compared cognitive therapy with ACT for the treatment of pathological eating in a college sample ($N = 55$). Their results described a significant interaction effect of Time x Condition (cognitive therapy vs. ACT), $F(1, 53) = 4.71, p = .03$, with the ACT condition demonstrating a larger effect size in decreasing eating pathology from pretreatment to posttreatment ($d = 1.89$), compared to the cognitive therapy condition ($d = .48$). Additionally, there was some preliminary evidence that global functioning was increased in the ACT condition more than the in the cognitive therapy condition, however, they suggest that this finding should be explored further. This suggests that targeting PF might be a means of remediating eating disorder pathology.
Additionally, a case series design has been employed to look at the effectiveness of targeting PF in three female participants diagnosed with AN (Berman, Boutelle, & Crow, 2009). These participants enrolled in 17 one-hour sessions of ACT. They completed the EDE-Q at pretreatment, posttreatment, and a 1-year follow-up. The authors reported that two of the participants had improved “substantially” and that the third participant improved modestly, but reported better gains in quality of life and pursuit of valued living. These treatment gains were maintained at 1-year follow-up.

Again, these findings point to the effectiveness of targeting PF to effect treatment gains. It is not the thoughts and feelings themselves that are of importance to ACT, but rather how the individual chooses to respond to those thoughts and feelings. When a person takes a more open stance towards unwanted private events, and chooses to respond in a manner that is consistent with his or her values, the person may be able to see a reduction in eating disordered symptoms, its comorbid effects (such as depression or anxiety), and increases in overall quality of life and psychological health.

Proposed Research Study Rationale

However, there are still gaps in the literature as to how PF relates to eating disorders. For example, the majority of the research cited is based on university students. While it is statistically probable that some of the participants met diagnostic criteria, the majority of the data represents typically functioning individuals. It is important to understand how PF functions for those individuals who are at severe levels of eating disordered behavior.

Theoretically, the reviewed literature predicts that individuals diagnosed with
eating disorders should also report lower levels of PF, compared to that of a typically functioning population. If that is true, then it should be possible to increase levels of PF, through ACT, and record changes in eating disorder pathology. Thus, the question becomes: What relationship exists between PF and eating disorder related constructs in a sample of residents at a residential treatment facility? Masuda and colleagues (2011) reported that PF functioned as a mediator. PF should be able to account for variance observed in eating disorder related constructs (such as body dissatisfaction and quality of life) at both admission and discharge to the treatment facility. Moreover, the level of PF at discharge may demonstrate increased explanatory power in a model accounting for variance in those same eating disorder related constructs. This leads to a series of testable hypotheses. This study will evaluate the role of PF in eating disorders using a sample of individuals in residential treatment for eating disorders. Specifically, the following predictions were tested.

1. Psychological inflexibility, at both admission and discharge, will demonstrate a moderate to strong correlation with body dissatisfaction and quality of life, seen at admission and discharge.

2. A. Psychological inflexibility at admission and discharge, as well as body dissatisfaction at admission, will significantly account for variance in body dissatisfaction at discharge.

   B. Psychological inflexibility at admission and discharge, as well as quality of life at admission, will significantly account for variance in quality of life at discharge.

3. A. Psychological inflexibility at discharge will have a stronger relationship
with body dissatisfaction at discharge than body dissatisfaction at admission and psychological inflexibility at admission.

B. Psychological inflexibility at discharge will have a stronger relationship with quality of life at discharge than quality of life at admission and psychological inflexibility at admission.
CHAPTER III

METHODS

Participants and Recruitment

Participants in this study are female residents of a mountain-west eating disorder treatment facility between the ages of 11 and 47 between January 2007 and September 2011. To be admitted to the facility, residents had to meet several criteria. First, residents had to meet DSM-IV-TR diagnostic criteria for AN, BN, or EDNOS. Initially, this diagnosis was determined by a home physician and/or psychologist who made the recommendation for admission to residential treatment. Then, upon admission to the treatment facility, diagnosis was achieved by consensus by a treatment team consisting of licensed therapists, physicians, nurse practitioners, nursing assistants and dieticians. Additional psychological diagnoses may have been present. Second, only individuals who were medically stable were admitted to the facility and, therefore, it is unlikely that residents are not physically able to complete the self-report assessment battery. Finally, admitted residents must have had the cognitive ability to engage in psychological treatment; thus, residents will not have intellectual disabilities that prevent self-report assessment.

Procedure

Data Collection and Storage

This study employed existing data that had been collected at the residential
treatment facility between January 2007 and September 2011. Upon admission to the treatment facility, residents were oriented to the facility, interviewed by staff about history of their eating disorder, family and personal history, and received a medical examination by nurse practitioners. During this period, residents were also given an informed consent form to participate in the ongoing data collection. Residents then completed the online assessment battery, consisting of validated and reliable self-report assessments, generally within the first 48 hours after being admitted to the facility. The responses to the self-report battery will be used in this proposed project.

Residents who signed the informed consent form were instructed to sit at a computer that then prompts them for responses for the items on the self-report measures. Responses to the self-report measures and related demographic information were stored on a secure online website (HIPAA compliant and provide exclusively to health-care companies). This data were de-identified to prevent possible violations of confidentiality. The individual resident’s de-identified responses were then collected and scored in an electronic database by a research assistant who is not familiar with the residents. This electronic database is at a separate site, again preventing possible violations of confidentiality. This same online assessment process was completed before the residents discharge from the facility, generally within 48 hours of discharge.

**Measures**

**Acceptance and Action Questionnaire-II.** The Acceptance and Action Questionnaire-II (AAQ-II; Bond et al., 2011) is a 7-item self-report questionnaire that measures psychological inflexibility. Each item is rated on a 7-point scale ranging from
“never true” to “always true.” A total score is summed and greater scores represent increased levels of psychological inflexibility. The test has good test-retest reliability ($\alpha = .88$) and convergent validity with BDI-II ($r = .71, p < .001$).

**Eating Disorder Inventory 3rd Edition.** The Eating Disorder Inventory—Third Edition (EDI-3; Garner, 2004) is a self-report assessment for use with individuals with eating disorders. The test is composed of 91 items that respondents rate each one on a 6-point Likert-type scale from *Always* to *Never* based on how true those statements are for their symptoms. The assessment is organized onto 12 primary scales, consisting of three eating-disorder specific scales (drive for thinness [DT], bulimia [B] and body dissatisfaction [BD]) and nine general psychological scales that are highly relevant to, but not specific to, eating disorders. It also yields six composites: one that is eating-disorder specific (Eating Disorder Risk Composite; EDRC) and five that are general integrative psychological constructs. The three eating-disorder specific scales will be used in this analysis. Garner reported the test-retest reliability for DT at .95, for B at .94, for BD at .95, and for EDRC at .98. Additionally, the manual reports good internal consistency: between .63 and .97 for these subscales (depending on diagnosis). The BD subscale was used in this study.

**Eating Disorder Quality of Life.** The Eating Disorder Quality of Life (EDQOL; Engel et al., 2006) instrument is a 20-item disease-specific health-related quality of life measure. The instrument taps into four subscales commonly impacted by an eating disorder: psychological, physical/cognitive, financial, work/school, and a total score, with each measure having a raw and mean score. The instrument has demonstrated good
psychometric properties and sensitivity to change with an eating disorder population.

Engel and colleagues reported 1 week test-retest reliability at .93 for the total score and report convergent validity correlations ranging from .41-.73 for each of the subscales (all significant at the $p < .01$ level). The total score was used in these data analyses.

**Data Analysis**

The data were analyzed using Statistical Packaging for Social Sciences (SPSS, version 19) and generated a mean, standard deviation and correlation coefficient for each of the three variables at both time points (admission and discharge) to explore relationships between constructs. Additionally, after logarithmic transformation, the EDI-3 BD and EDQOL at discharge were analyzed in three regressions each. Finally, two general models were generated examining each variable’s accounting of variance in EDI-3 BD and EDQOL at discharge. Significance for each analysis was set at $p = .05$ level.
CHAPTER IV

RESULTS

The participants in this study are all female between the ages 11 - 47 ($M = 18.61; SD = 5.38$). Fifty-four percent were diagnosed with AN, 19.9% were diagnosed with BN, and 26.1% were diagnosed with EDNOS. Each participant completed a battery of measures when they were admitted to the program and the same measures at discharge. Because this analysis was conducted as part of an ongoing data collection initiative at a residential treatment facility, some measures were added at a later point in time than others. Thus, the sample size is different for some of the measures. Additionally, this data was captured at a moment when some individuals were still in treatment, or left the facility before completing the assessment battery, and is the reason why there is discrepancy between the admission and discharge sample sizes.

For the measure of PF, the AAQ-II, the mean at admission is 30.42 ($SD = 9.75; N = 109$) and at discharge is 18.84 ($SD = 8.69; N = 79$). For the measure of body dissatisfaction, EDI-3 BD, the mean at admission is 27.51 ($SD = 11.52; N = 167$) and at discharge is 14.77 ($SD = 12.06; N = 119$). For the quality of life measure, EDQOL, the mean at admission is 47.81 ($SD = 18.83; N = 168$) and at discharge is 22.91 ($SD = 21.92; N = 119$). Additionally, a correlation matrix was generated for each measure at both measurement times and is reported in Table 1. Note that the majority of the measures have statistically significant correlations. These correlation coefficients demonstrate that these constructs are interrelated, but are separate measurements.

For the hypothesis testing, data was analyzed in a series of steps. Initially, the data
Table 1

Correlation Matrix for AAQ-II, EDI-3 BD and EDQOL at Admission and Discharge

<table>
<thead>
<tr>
<th>Variable</th>
<th>Admission</th>
<th></th>
<th></th>
<th>Discharge</th>
<th></th>
<th></th>
</tr>
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<tbody>
<tr>
<td></td>
<td>AAQ-II</td>
<td>EDI-3 BD</td>
<td>EDQOL</td>
<td>AAQ-II</td>
<td>EDI-3 BD</td>
<td>EDQOL</td>
</tr>
<tr>
<td>Admission</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AAQ-II</td>
<td>r</td>
<td>.44**</td>
<td>.53**</td>
<td>.54**</td>
<td>.33**</td>
<td>.24*</td>
</tr>
<tr>
<td>p</td>
<td>-</td>
<td>.00</td>
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<td>.00</td>
<td>.01</td>
<td>.05</td>
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<tr>
<td>EDI-3 BD</td>
<td>r</td>
<td>.44**</td>
<td>.50**</td>
<td>.09</td>
<td>.33**</td>
<td>.14</td>
</tr>
<tr>
<td>p</td>
<td>.00</td>
<td>-</td>
<td>.00</td>
<td>.43</td>
<td>.00</td>
<td>.13</td>
</tr>
<tr>
<td>EDQOL</td>
<td>r</td>
<td>.53**</td>
<td>.50**</td>
<td>.23*</td>
<td>.26**</td>
<td>.34**</td>
</tr>
<tr>
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<td>.00</td>
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</tr>
<tr>
<td>Discharge</td>
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<td></td>
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</tr>
<tr>
<td>AAQ-II</td>
<td>r</td>
<td>.54**</td>
<td>.09</td>
<td>.23*</td>
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<td>.68**</td>
</tr>
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<td>p</td>
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<td>-</td>
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<td>.00</td>
</tr>
<tr>
<td>EDI-3 BD</td>
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<td>.33**</td>
<td>.26**</td>
<td>.68**</td>
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<td>p</td>
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<td>-</td>
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<tr>
<td>EDQOL</td>
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<td>.14</td>
<td>.34**</td>
<td>.60**</td>
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<td>p</td>
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<td>.12</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>-</td>
</tr>
</tbody>
</table>

* Correlations significant at 0.05 level.
** Correlations significant at 0.01 level.

for the outcome measures (EDI-3 and EDQOL) demonstrated skew and had to be transformed logarithmically. Because the EDI-3 had an item-response values that included 0, it was necessary to add one point to each participant’s item responses on the EDI-3 to make the logarithmic transformation possible on each participant’s BD subscale score. This transformation allowed for the EDI-3 and EDQOL scores to meet the assumption of normality necessary for regression analysis and modeling.

After completing the transformation of the data, it was possible to perform the series of regression analysis that account for the variance in discharge scores of EDI-3 BD and EDQOL. The results of these regressions are reported in Table 2. For the EDI-3 BD, recall that the hypothesis is that AAQ-II and EDI-3 BD at admission will account for
Table 2

Independent Regressions of AAQ-II at Admission and Discharge, and EDI-3 BD and EDQOL at Admission on EDI-3 BD and EDQOL at Discharge

<table>
<thead>
<tr>
<th>Variable</th>
<th>$R^2$</th>
<th>$B$</th>
<th>$SE(B)$</th>
<th>$\beta$</th>
<th>$t$</th>
<th>$df$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDI-3 BD (discharge)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AAQ-II (admission)</td>
<td>.12</td>
<td>.04</td>
<td>.02</td>
<td>.34</td>
<td>2.90</td>
<td>64</td>
<td>.01**</td>
</tr>
<tr>
<td>AAQ-II (discharge)</td>
<td>.31</td>
<td>.08</td>
<td>.01</td>
<td>.56</td>
<td>5.74</td>
<td>72</td>
<td>&lt; .01**</td>
</tr>
<tr>
<td>EDI-3 BD (admission)</td>
<td>.06</td>
<td>.02</td>
<td>.01</td>
<td>.24</td>
<td>2.62</td>
<td>114</td>
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<td>EDQOL (discharge)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AAQ-II (admission)</td>
<td>.03</td>
<td>.01</td>
<td>.01</td>
<td>.18</td>
<td>1.55</td>
<td>69</td>
<td>.13</td>
</tr>
<tr>
<td>AAQ-II (discharge)</td>
<td>.36</td>
<td>.03</td>
<td>.00</td>
<td>.60</td>
<td>6.58</td>
<td>77</td>
<td>&lt; .01**</td>
</tr>
<tr>
<td>EDQOL (admission)</td>
<td>.09</td>
<td>.01</td>
<td>.00</td>
<td>.30</td>
<td>3.37</td>
<td>119</td>
<td>&lt; .01**</td>
</tr>
</tbody>
</table>

* Regression significant at .05 level.
** Regression significant at .01 level.

Some variance seen in the discharge score, but that the AAQ-II at discharge would have a greater ability to account for variance in the EDI-3 BD at discharge. For the EDQOL, the hypothesis is essentially the same, exchanging the EDQOL at admission and discharge for EDI-3 BD scores. The AAQ-II at admission score is not statistically significant in accounting for variance in EDQOL discharge scores; but the EDQOL at admission and AAQ-II at discharge are significant in accounting for variance, with the AAQ-II score at discharge having a greater ability to account for variance.

Finally, to fully answer the question guiding this research, it was necessary to employ general linear models for both the EDI-3 BD and EDQOL at discharge. Results of this model can be seen in Table 3. Both models are statistically significant in accounting for variance in discharge scores, with the AAQ-II at discharge demonstrating the strongest relationships with the outcome variables.
Table 3

General Model Accounting for Variance at Discharge for EDI-3 Body Dissatisfaction (BD) and Eating Disorder Quality of Life (EDQOL) Using AAQ-II at Admission and Discharge, and EDI-3 BD and EDQOL at Discharge

<table>
<thead>
<tr>
<th>Variable</th>
<th>$R^2$</th>
<th>df</th>
<th>$F$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDI-3 BD (discharge)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Corrected model</td>
<td>.34</td>
<td>3</td>
<td>10.53</td>
<td>&lt; .01**</td>
</tr>
<tr>
<td>AAQ-II (admission)</td>
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<td>.85</td>
<td></td>
</tr>
<tr>
<td>AAQ-II (discharge)</td>
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<td>18.71</td>
<td>&lt; .01**</td>
<td></td>
</tr>
<tr>
<td>EDI-3 BD (admission)</td>
<td>1</td>
<td>3.17</td>
<td>.08</td>
<td></td>
</tr>
<tr>
<td>EDQOL (discharge)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corrected model</td>
<td>.42</td>
<td>3</td>
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<td>&lt; .01**</td>
</tr>
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<td>AAQ-II (admission)</td>
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<td>6.15</td>
<td>.02*</td>
<td></td>
</tr>
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<td>AAQ-II (discharge)</td>
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<td>40.40</td>
<td>&lt; .01**</td>
<td></td>
</tr>
<tr>
<td>EDQOL (admission)</td>
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<td>4.34</td>
<td>.04*</td>
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</tr>
</tbody>
</table>

* Significant at .05 level.
** Significant at .01 level.
CHAPTER V
DISCUSSION

The question guiding this research is: What relationship exists between PF and eating disorder related constructs in a sample of residents at a residential treatment facility? As can be seen from the means and standard deviations reported, participants are endorsing movement on each measure towards healthier functioning. Over the course of their treatment at the residential facility, these participants have made considerable improvement related to all three constructs that were measured in this study. A series of hypotheses were put forward and quantitatively analyzed to help explain what changes are occurring. First, the correlations (see Table 1) describe relationships between constructs at both time points, and all but two of those correlations are moderate to strong and statistically significant (the two that failed to reach significance are correlations between admission and discharge scores, which were not a specific focus of this project). Additionally, it should be noted that the correlation coefficients increase in value from admission to discharge. This is most likely an artifact due to the measurement, but does not discount the relationship between each construct.

Having established the relationships between these constructs at admission and discharge, this leads to testing of the second hypotheses related to regression analyses. The hypotheses related to the discharge levels of both the EDI-3 BD and EDQOL are that the AAQ-II at discharge would be significant in accounting for their variance. Table 3 shows that these hypotheses are confirmed. The AAQ-II at discharge is able to account for 31% of the variance in EDI-3 BD at discharge and 36% of the variance in EDQOL at
discharge. These results are important, and help to answer the research question, because a significant portion of the variability in outcome scores is described by the attempts of a person to control or escape unwanted internal experiences. The inference is that by increasing the level of a person to interact flexibly with their thoughts, they will be able to decrease the level of overall body dissatisfaction and quality of life directly impacted by an eating disorder.

Obviously this does not account for all of the variance in body dissatisfaction and quality of life but it is an important portion of their variance. The EDI-3 BD, EDQOL and AAQ-II at admission were also used in these regressions in accounting for variance in the outcome variables. This is because there is an assumption that the individuals come in to treatment with already extant levels of PF, body dissatisfaction and quality of life and should be analyzed as explanatory variables separate from PF at discharge. AAQ-II and EDI-3 BD at admission account for a small amount of variance (12% and 6%, respectively) for EDI-3 BD at discharge, but only EDQOL at admission (9%), and not AAQ-II at admission, account for variance in EDQOL at discharge. So, all but one of those hypotheses were confirmed, demonstrating that they do have some value to add in understanding EDI-3 BD and EDQOL at discharge (with the exception of AAQ-II at admission).

While these simple regressions help in understanding the degree to which they can account for variance independently, these constructs do not exist in a conceptual vacuum. Instead, these constructs are interrelated (as demonstrated in Table 1) and can exhibit influence one another. So, a final pair of hypotheses was put forward to model the
ability of PF at discharge to describe a relationship with EDI-3 BD and EDQOL at discharge, while holding AAQ-II, EDI-3 BD, and EDQOL at admission constant (see Table 3). The model demonstrates that AAQ-II at discharge has a stronger relationship with EDI-3 and EDQOL at discharge when holding the other variables constant. Moreover, this model demonstrates that those other variables lose their explanatory power. So the final pair of hypotheses is confirmed; the AAQ-II at discharge holds more explanatory power in EDI-3 BD and EDQOL at discharge. It is interesting to note, the only other variable that, while small, is still statistically significant in the model is AAQ-II at admission and is also the only variable that failed to demonstrate significance in the simple regression for the EDQOL at discharge. It is possible that this variable is significant in the model and not the regression because it does not account for variance in EDQOL at discharge, but would account for variance in AAQ-II at discharge, which is significant in both regression and modeling. However, more quantitative analysis is necessary to test this.

In returning to the original research question, how does PF relate to eating disorder related constructs, the answer becomes clearer in light of the present findings? There is a strong relationship with a person’s attempts to escape or avoid unwanted internal events and the degree to which they are dissatisfied with their body and negatively evaluate their quality of life. What this study shows is that as a group, individuals who are healthy enough to be discharged from a residential treatment facility share levels of PF that are consistent with healthier functioning; they interact with negatively evaluated thoughts in a way that does not result in behavior that is consistent
with an eating disorder. Indeed, private and public events that may bring about unwanted thoughts and feelings may still show up for these participants; however they have learned a new way to deal with them. They do not engage in behaviors that are directed at suppressing, escaping or avoiding any of these. The correlations demonstrate that these constructs are strongly related, and the results of the regressions and modeling describe how PF can account for different levels of body dissatisfaction and quality of life. With greater ability of an individual to interact flexibly with their internal events, they express less dissatisfaction with their bodies and their quality of life is improved.

However, this study has several limitations that should be mentioned. First, this study employed a sample of residents in an eating disorder treatment facility who were only measured when they admitted and discharged from the facility. This data, therefore, violates the principle of independence of observation. These participants were interacting with one another and could have influenced each other in ways that the research methodology could not parse out. It would be better if the participants were independent of one another so that confidence in PF’s relationship with eating disorder related outcomes. However, as the data presents a limitation related to the fact that it comes from a treatment facility; the simple fact that the data is gathered at a residential treatment facility presents a strength of the project. This data represents a sample of individuals who have been so severely impacted by their eating disorders that they sought treatment in a facility that is dedicated to arresting external symptoms of the disorder and allowed for the magnified focus on the internal events and agendas that sought to control them.

The data is collected while the participant is still in the treatment facility, so it is
possible that participants could be malingering at the admission measurement point and responding favorably as they discharged from the facility. The data was logarithmically transformed for the discharge measurements of both EDI-3 and EDQOL because it demonstrated skew. The participants were reporting enough favorable results that the curve did not fit the assumptions for regression and modeling analyses. While it is possible that the participants were responding in a way that does not conform to assumptions of data normality, the participants are responding at a point in time when their symptoms have remised enough for therapists to discharge them from the facility. Because of this, it is also possible that participants are as well as they report, however the conclusions of this paper could be more confident if there was another data point where the participants were separate from the facility, e.g. a six-month follow-up assessment.

Additionally, the correlation coefficients increase from admission to discharge. At both measurement times, participants completed the entire battery of assessment and it is likely that through their treatment at the facility, the constructs of PF, body dissatisfaction and eating disorder quality of life became conceptually linked. Thus, the participants may be responding in a way that artificially increases their correlation coefficients, through measurement error variance, and does not accurately describe these relationships.

Finally, there are participants who did not complete the assessment at discharge. At a residential treatment facility, occasionally residents removed themselves from the program against a counselor’s or medical advice. Every step is done to prevent this type of discharge from happening, but it still occurs. There are also occasions that logistically prevent an individual from completing the discharge assessment before returning to their
home. Every effort is made to have these participants complete the assessment when they get home, but there are times were this still does not occur.

There are some considerations that should be made when attempting to generalize the findings from this study. For example, this is a sample of female participants drawn from a residential treatment facility and therefore the participants in this study have the means to afford such treatment. Thus, it may be that other individuals who are in different socio-economic situations, individuals who cannot afford residential treatment, may not respond in ways that are similar to the participants of this study. Another consideration that should be taken is the age range of participants employed in this study. This sample was aggregated from females age 11 to 47, which could present a large developmental difference. For the purpose of this discussion, it is assumed that there is a difference in the emotional development between different age groups. While, in younger participants there could be more readiness to adopt new means of interacting with private events, older participants may be more hesitant to adopt these new behaviors and letting go of control strategies. Future research that explores the differences in PF as it relates to emotional development across the lifespan would shed light on this subject.

The results from this study corroborate evidence already gathered about PF’s relationship with eating disorder related constructs and fits in with the broader research. It is evident that those with greater PF demonstrate less psychopathology (Ruiz, 2010), especially related to eating disorders (Juarascio et al., 2010; Masuda et al., 2010, 2011). This research not only corroborates that evidence, but examines it within a population of severely eating disordered sample. Where other studies employed convenience samples
from colleges, this sample demonstrates that PF’s relationship to eating disorder constructs is still true even at a more severe level. Greater PF is related to improvements in eating disorder related constructs, to the best ability there is to measure them.

In addition to ACT with its focus on PF, other forms of therapy have been developed to target types of cognitive rigidity, especially in regards to executive functioning. For example, cognitive remediation therapy (CRT; Tchanturia, Davies, & Campbell, 2007; Wykes & van der Gaag, 2001) is a therapy designed to aid the individual to reduce rigid forms of problem solving and employ novel methods. Initially employed with those diagnosed with a form of psychosis, it has recently been tested as a form of therapy with those with AN (Davies & Tchanturia, 2005). The central idea related to CRT is that an individual who display impairments in executive functioning, specifically in terms of set-shifting tasks, will maintain their anorexic behaviors even when they have a desire to change. CRT allows the individual to experience treatment gains in these areas, without the necessity of grappling with difficult issues or emotions related to their eating disorder (Davies & Tchanturia, 2005). So, it may be possible, especially with those individuals who are treatment resistant to employ CRT as a first line of therapy and, once executive functioning is improved, introduce a more cognitive-behavioral therapy (such as ACT) that helps undermine the agenda of controlling unpleasant thoughts and feelings.

Given this body of literature, it is prudent that future studies look at directly manipulating PF and how that impacts eating disorder behavior. This study implies a causal or directional model—using PF as a predictor of body dissatisfaction and quality of life—but causality cannot be demonstrated from this study. If the theory concerning
PF’s ability to account for changes in other variables, then controlled experiments are necessary. Using small sample, multiple-baseline designs and randomized control trials is one way to explore this. While the results of the present study were able to account for around 30%-40% of the variance seen in body dissatisfaction and quality of life, the rest of the variance could be explained by a number of other related variables including specific internal events. Additionally, it would be interesting to examine how eating disorder thoughts and feelings occur in individuals with increased PF. Do the thoughts and feelings disappear entirely, do they occur with less frequency, or do those thoughts and feelings appear at the same frequency and are responded to differently? It is not consistent with ACT that an individual struggling with any psychological or behavioral issues be completely free of unwanted thoughts and feelings to be healthy, it just describes how a healthy individual responds to them (Hayes et al., 2006).

An experiment with tighter control could greatly increase the body of information related to PF and eating disorders. The present study adds to the evidence that this type of research is warranted. From the results herein, inferences are made about the ability of PF to account for eating disorder related constructs. Acceptance and Commitment Therapy (Hayes et al., 2001), which specifically functions to increase PF, should be investigated as a treatment for eating disorders in an experimental design. This study demonstrates the relationship PF has with these other constructs, but it is limited to how much it can describe how the relationship functions. A well-controlled experiment that uses ACT to manipulate PF as an independent variable and eating disorder constructs as dependent variables would be able to shed considerable light on the subject where this study is
unable. There have been numerous studies that have looked at ACT as a treatment for various other disorders and ACT is demonstrating effectiveness in treating a broad range of issues (see Ruiz, 2010). By implementing a therapy that is designed to help an individual respond to unwanted internal events in a way that is consistent with their values, as opposed to strategies like suppression, escape, and avoidance, ACT may be a useful tool that therapists can draw upon to help improve the lives of individuals struggling with eating disorders.
REFERENCES


