2016

INDIVIDUALS' PERCEPTIONS OF DISORDERED EATING PATHOLOGY: DEVELOPMENT AND VALIDATION OF THE INVENTORY FOR DISORDERED EATING ATTRIBUTIONS (IDEA)

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INDIVIDUALS' PERCEPTIONS OF DISORDERED EATING PATHOLOGY:

DEVELOPMENT AND VALIDATION OF THE INVENTORY FOR DISORDERED EATING

ATTRIBUTIONS (IDEA)

By

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Dissertation

presented in partial fulfillment of the requirements
for the degree of

Doctor of Philosophy
in Clinical Psychology, Child/Family and Neuropsychology

The University of Montana
Missoula, MT

May 2016

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Abstract

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  Eating pathology may be triggered by a number of biological, environmental, personal, and social experiences. Research in the field of disordered eating has uncovered numerous intrinsic and extrinsic vulnerabilities to developing abnormal eating behavior, and these etiologies often impact the onset, severity, type, and prognosis of disordered eating behavior in varying ways. Further, a limited number of measures have been created to examine individuals' beliefs about why they are experiencing various mental health conditions (e.g. reasons for depression, developing substance dependence, etc.), which has implications for tailoring assessment and intervention. However, there is currently no such measure for individuals experiencing disordered eating. **Objective:** To develop and test the Inventory for Disordered Eating Attributions (IDEA) in order to assess the reasons individuals provide for their disordered eating behaviors and/or cognitions while offering pertinent data to their mental health provider(s) regarding case conceptualization, diagnosis, and treatment. **Method:** Questionnaire items were generated and vetted through a panel of experts in the fields of eating pathology and illness attribution. Next, proofed items were administered to a large undergraduate university sample (n = 424) along with a second eating measure and a demographic questionnaire in order to evaluate internal consistency, convergent validity, test-retest reliability, and to identify domains of attribution through principal components analysis. **Results:** Principal components analysis revealed a four-factor solution for the 20-item IDEA. The IDEA revealed strong psychometric properties, including a Global Score Cronbach's Alpha of .90. **Conclusions:** The IDEA is a brief self-report measure with clinical utility across behavioral health disciplines and providers. Future research should explore how elevations on IDEA subscales correlate with treatment outcomes under various therapeutic modalities.

With the emergence of the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*), the domain of Feeding and Eating Disorders (EDs) included several changes to a variety of diagnostic criteria (American Psychiatric Association [APA], 2013a). The *DSM-V* Eating Disorders Work Group recognized a problematic trend across applied and research psychology of using eating disorder not otherwise specified (EDNOS) as a catch-all diagnosis for individuals with abnormal eating behavior that did not meet diagnostic criteria for anorexia nervosa (AN) or bulimia nervosa (BN) (APA, 2013). In an effort to minimize the use of EDNOS in this manner, and to provide diagnoses that accurately reflect symptoms and behaviors, the criteria for EDs were modified.

First, binge eating disorder (BED) was added to the collection of EDs in the *DSM-V*, rather than being described in an appendix only. BED is currently characterized by recurring episodes of overeating by consuming more food in a shorter amount of time than most people would consume under similar circumstances. The eating episodes are marked by feelings of lack of control and occur at least once a week over three months (APA, 2013). Importantly, this eating pathology is intended to describe binge eating that is less common, more severe, and accompanied by significant physical and psychological problems that are not present in the more common phenomenon of overeating.

DSM-V expanded diagnostic criteria for AN and BN in the *DSM-V* to include more individuals whose symptomatology were previously subthreshold and therefore fell under EDNOS diagnoses in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition-Text Revision* (*DSM-IV-TR;* APA, 2000). The first criterion for AN no longer includes the word "refusal" to describe weight maintenance strategies. Specifically, refusal to maintain body weight at or above a minimally normal weight for age and height has been

changed to "Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health" (p. 1). This change occurred in an effort to remove the implication that an individual's behaviors involve complete refusal of appropriate caloric intake in order to meet the diagnostic criteria and because assessing for patient intention can be difficult (APA, 2013b). Next, criterion D in the *DSM-IV-TR* required the presence of amenorrhea (the absence of at least three menstrual cycles) for the diagnosis of AN. In an effort to accurately diagnose boys/men with eating psychopathology, pre-menarchal girls, girls/women taking oral contraceptives, and post-menopausal women this diagnostic criterion was removed in the *DSM-V* (APA, 2013). Lastly, similar to AN diagnostic modifications, the *DSM-V* lowered the threshold of symptom presentation in BN by reducing the frequency of binge eating and compensatory behaviors from twice a week to once a week in order to remove certain individuals with binging and purging behaviors out of EDNOS and into a more accurate and clinically relevant diagnosis (APA, 2013).

ED Prevalence

Prevalence rates of eating pathologies have varied across studies in concert with varying measurement and diagnostic strategies (Smolak & Murnen, 2002); however, the *DSM-V* reports that the 12-month prevalence rates of BED among adults in the U.S. is 1.6% for females and 0.8% in males and occurs with similar frequencies across most industrialized countries (Hudson et al., 2007 as cited in *DSM-V*, APA, 2013; Marques et al., 2011). AN and BN prevalence data indicate these diagnoses occur less frequently than BED. Specifically, the 12-month prevalence among females of AN and BN are approximately .4% and 1% - 1.5%, respectively (Hoek, 2006 as cited in *DSM-V*, APA, 2013). Further, these

eating pathologies seem to surface more frequently in cultures and settings that value

thinness but occur within white and ethnic minority populations including non-Latino

whites, Latinos, Asians, and African Americans relatively equally (Marques et al., 2011).

Prevalence rates suggest AN and BN are reported less frequently in boys/men, relative to

the BED ratio (which has a roughly 10:1 female-to-male ratio). Prevalence rates of EDs in

the general population are low in relation to many other psychopathologies and data

pertaining to frequency of EDs in specific populations are further discussed below. Despite

its low base rate, the mortality rate of AN in the general population is 10% making it the

most deadly psychiatric disorder in the United States (APA, 2000; Guisinger, 2003).

Decades of research have identified a number of mental health comorbidities with

eating pathology. Recently published research in the *DSM-V* suggests common comorbid

diagnoses for individuals with AN include bipolar, depressive, and anxiety disorders as well

as obsessive and/or compulsive traits (APA, 2013). Similarly, BN and BED frequently co-

occur with one or more psychological disorders, most notably mood and anxiety

disturbances and substance use disorders. Interestingly, these mental health disturbances

frequently abate following effective treatment suggesting that they may typically be a side

effect of the ED as opposed to an ED precursor or risk factor (APA, 2013; Wonderlich et al.,

2009). Thus, targeted treatment for EDs may result in a reduction in overall pathology.

Etiological Theories

The changes to the Feeding and Eating Disorders section of the *DSM*, though much

needed to improve the accuracy and relevance of ED diagnoses, imply nothing regarding

the etiology of eating pathology. Historically, the *DSM* has focused on identifying a

collection of symptoms or behaviors relevant to a particular diagnostic construct, with little

to no emphasis on the factors affecting their development. Understanding the etiology of

behaviors such as those involved in EDs has important implications for assessment and

treatment of traditionally difficult-to-treat conditions such as EDs. In particular, assessing

the individual's perspective on their own behaviors and experiences may affect the success

of a clinician's approach to treatment engagement, execution, and relevant outcomes

(Addis, Traux, & Jacobson, 1995; Schweizer et al., 2010). This is especially important given

that recent research assessing associated factors with disordered eating (DE) (i.e., trauma

exposure) has shown that the relationships between many of these factors and eating

pathology were not significant when examined empirically, despite what has been

anecdotally accepted in the field (Crosby & Borntrager, in progress).

Medical sociology and anthropology research suggests that there is a relatively

recent shift in usual care involving the patient advancing from a passive recipient of health

care to being actively involved in their treatment process (King, 1983). The Health Belief

Model (HBM), originally developed by Rosenstock, suggests that a patient's attitudes and

beliefs about their illness can explain much of their health-related actions such as

attendance and treatment compliance (Rosenstock, 1966; Becker & Maiman, 1975). Indeed,

the ways in which a patient interprets their illness can have a direct impact on their

responsiveness to treatment and ultimate treatment success (King, 1983). Attribution

theory assumes that each patient has an intrinsic need to understand the cause of events

they experience (Weiner, 1980) and, from a provider perspective, 'illness attribution'

involves assessing and understanding the patient's perceptions on the development of

their conditions in order to inform treatment planning and case conceptualization

(Corrigan, 2000; Schweizer et al., 2010).

Researchers in the field of illness attribution and depression found that a client's

specific reasons for depressive symptoms can predict efficacy of certain therapeutic

approaches (Addis & Jacobson, 1996). Specifically, while patients endorsing existential

reasons for depression were more responsive to cognitive therapy than behavioral therapy,

individuals attributing their depression to relationships (e.g. marriage) were less

responsive to cognitive therapy (Addis & Jacobson, 1996) and those endorsing more

biological reasons had better outcomes with antidepressant medication therapy (Leykin et

al., 2007). Despite the treatment benefits, systematically assessing illness attributions in

order to inform treatment planning has not been explored in the ED field, perhaps because

there is currently no measure available to assess a client's reasons for abnormal eating.

There are a number of factors associated with the etiology of eating pathology in the

literature, which may represent some of the illness attributions that a patient with ED

might endorse. Developing an evidence-based assessment measure for examining these

factors could have important clinical implications particularly given the prevalence and

mortality associated with DE behaviors.

Etiological Theories: Demographics

Though EDs have historically been characterized as illnesses that affect only young,

white, upper class girls, more recent literature suggests that sociocultural factors in racial

and cultural minorities may impact ED etiology (Cummis, Simmons, & Zane, 2005). As

described, EDs among racial and cultural minorities are not absent, and in fact, many DE

behaviors and ideations in minority populations may be going unrecognized and

undiagnosed possibly due to fewer minority individuals reporting symptoms to their

health care providers or higher thresholds for diagnosing a minority individual with an ED

due to assumed lower risk (Pike & Walsh, 1996). Undiagnosed psychopathology in

minority populations is not unique to EDs. Many underserved communities harbor high

prevalence rates of substance abuse, depression, and crime. These mental health

difficulties are often coupled with stigma about mental illness and multiple access barriers

(Roberts et al., 2008). Unfortunately, one of the fundamental factors in recovering from ED

is early detection and treatment (APA, 2000), and again, due to the higher chance of being

misdiagnosed or undiagnosed due to less frequent self reporting or provider bias, EDs in

minority populations are often unrecognized or only recognized after the illness has

progressed to a critical phase (Pike & Walsh, 1996).

Summaries of ED prevalence and behavior in specific minority populations are

presented below. However, it is important to note that when ethnic and racial minorities

are studied within a Western country (e.g. the United States), individuals in the minority

sample may actually be quite diverse in terms of ethnicity and culture. For example,

research involving Asian populations in the United Kingdom often refer to Asians of Indian

descent simply because a majority of Asians in the United Kingdom immigrated or had

families who immigrated from India. In contrast, an Asian sample in the United States may

represent a varied collection of East Asian, Southeast Asian, and other Asian ethnic groups

(Cummis, Simmons, & Zane, 2005).

African American Populations

Recent research examined effects of psychosocial correlates of ED symptoms in a

minority group involving young Black female Americans. Results suggested that many

young Black females experience social pressures unique to their culture and race that

contribute to DE behaviors (Zucker, 2001). Specifically, social pressure for thinness from

the media, from mothers, and from peers; pubertal development; and self-esteem were identified as correlates to ED symptoms in Black female adolescents.

Despite research suggesting social pressures for obtaining or maintaining a societally defined ideal body shape, results of some studies suggest that many African Americans experience lower DE behaviors and ideologies than their white peers. Specifically, Pernick et al. (2006) examined eating behavior and ideation in 453 female high-school athletes and analyzed covariance after adjusting for body mass index and sport. Results suggested that African American female high school students experienced fewer symptoms of DE than their White and Latina peers. However, certain aspects of eating pathology were more elevated in the African American sample than in the Latina sample, such as the 'eating restraint' subscale of the target outcome measure (Eating Disorders Examination Questionnaire (EDE-Q); Fairburn & Beglin, 1994). Yet, scores were not as high as eating restraint subscale scores reported by the white sample (Pernick et al., 2006). These data suggest that although the broad experience of DE might be less common among African American girls/women compared to White females, certain aspects of DE behaviors (e.g., eating restraint) might be more common among African Americans than Latina girls/women.

Asian and Asian American Populations

In contrast to the available research assessing DE behaviors among Black and African American populations, the literature on Asian populations is more mixed. Specifically, research findings suggest that South Asian girls have a higher prevalence of BN than White girls in the United Kingdom (Mumford, Whitehouse, & Platts, 1991). In one study, 599 high school girls were sampled and 3.4% of Asian girls were diagnosed with BN

(using *DSM-III-R* diagnostic criteria) while only 0.6% of White girls met diagnostic criteria (Mumford, Whitehouse, & Platts, 1991).

Recent studies have examined specific behaviors associated with ED in Asian American and White Americans. Results suggest that though weight concern, restricting, and binge eating behaviors were similar between ethnic groups, White men and women are more likely to practice compensatory behaviors than Asian Americans (Huang, 2001). Further, methods used to restrict food intake and purge what was consumed significantly differ between Chinese and Western individuals. Specifically, findings suggest that Chinese individuals will typically restrict rather than purge (Lai, 2000) and those who do purge will more often use laxatives rather than vomit, which is a compensatory behavior more commonly found in Western populations (Lee, Hsu, & Wing, 1992).

Though these and other studies have found interesting differences in ED symptomatology and prevalence between Asian and American cultures, it should be noted that other studies indicated no or non-significant differences [Mukai, Crago, & Shisslak, 1994 (Japan); Davis & Katzman, 1997 (Hong Kong, China); Gross & Rosen, 1988 (Asian American)]. It seems the majority of the discrepancies in findings can be attributed to the difficulty of using samples determined by race and ethnicity because of in-group differences (e.g. Chinese population living on Hong Kong versus those living in mainland China). Further, the use of Western-based diagnostic criteria may not adequately capture symptom patterns in Asian populations (Cummins, Simmons, & Zane, 2005).

Hispanic Populations

Similar to what was found in the research comparing eating pathology among Asian and Western populations, many studies suggest there is little general difference in

Hispanics' and Whites' eating behaviors in the United States (Garcia-Rea, 2007; Haddock et al., 1999; Marson, 2000). However, other studies have found interesting discrepancies when examining specific behaviors and thoughts associated with abnormal eating. In one randomized clinical trial of individuals with BED, results of a mixed model analysis indicated that Hispanic American participants held significantly greater body shape, weight, and eating concerns (as measured by the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993)) than their Caucasian counterparts (Franko et al., 2012).

Similarly, when breaking diagnoses into specific behaviors, research using a cross-sectional analysis of ethnic populations in the United States found a significantly greater prevalence of binge eating and vomiting among Latina high-school students compared to their Caucasian and African American counterparts (Pernick et al., 2006). This study was particularly noteworthy because it used an eating behavior measure that assessed general DE behavior and DE ideations instead of eating behaviors that specifically meet criteria for AN or BN as measured by the *DSM* (which employs Western-based diagnostic criteria).

Male Gender

Though male gender is not considered a minority characteristic in the general population, in the growing body of ED research and clinical practice male gender is a rare characteristic in comparison to female gender among those studied and struggling with DE. Specifically, AN typically begins during early adolescence and more than 90% of cases occur in girls (APA, 2000). Similarly, BN usually begins in late adolescence or early adulthood and approximately 90% of individuals with BN are girls/women (APA, 2000). However, recent research has found a steadily increasing prevalence rate in boys and men with ED (Kjelsas et al., 2003; O'Dea & Ahraham, 2002).

In one cross-sectional study of ED shape and weight concerns in adolescent boys

and girls, results found a high number of boys with ED relative to girls with ED. Specifically

male-to-female ratios of any ED were 1:2.8, AN 1:3.5, BN 1:2, BED 1:1.7, and EDNOS 1:2.9

respectively (Kjelsas et al., 2003). These ratios are in stark contrast to the 10% prevalence

rate of ED among men reported in the *DSM-IV-TR* (APA, 2000). Further, researchers found

that boys/men might be more inclined to experience negative and unrealistic body

perception than previously presumed. According to Kjelsas et al. (2003), "Of the 331 girls

who considered themselves obese, 243 (73.4%) were classed underweight or normal

weight. For boys, 92 (62.1%) of the 148 who considered themselves obese were

underweight or normal weight" (p. 18). These statistics indicated fairly similar body image

dissatisfaction and distorted body image among female and male samples, which is largely

contradictory to early theories and research conceptualizing ED as a disorder primarily

affecting girls/women.

Cross-cultural factors related to the development of eating problems are salient and

therefore must be accounted for when identifying, understanding, and treating eating

pathology. Etiological cross-cultural variables such as the perception of social power, low

personal control, and high desire for control are significantly correlated with greater eating

struggles (D'Agruma, 2004) and are therefore essential aspects to be integrated into

treatment. Unfortunately, research suggests that some minority populations are less likely

to seek treatment for ED than their majority (primarily Western culture female gender)

counterparts and boys/men with DE behaviors are less likely to seek treatment for

struggles with binging and purging behaviors than their female counterparts (O'Dea &

Abraham, 2002). In one study, 9% of college males reported difficulty with DE, 18%

reported weight control problems, and 22% were binge-eating yet *none of them* had sought

help or been treated for eating problems (O'Dea & Abraham, 2002). It is possible that part

of the problem can be attributed to a greater social acceptance of Western White female

symptom endorsement and therefore a rareness of minority individuals willing to voice

their struggles and seek services. Given these data, it seems essential that an opportunity to

obtain the patient's perspective on illness attribution is provided, discussed, and

incorporated into treatment.

Etiological Theories: Personality Characteristics

There is a strong literature base examining the relationship between eating

pathology and personality. In 2005, Cassin and Von Ranson published an article reviewing

a decade of literature on how personality characteristics and disorders have been

associated with the onset, symptom expression, and maintenance of EDs. This most recent

meta-analytic review of personality characteristics and DE pathology found consistent

results suggesting AN and BN are characterized by numerous personality traits in varying

degrees. These traits included perfectionism, obsessive-compulsiveness, neuroticism,

narcissism, sociotropy-autonomy, impulsivity, and sensation seeking (Cassin & Von

Ranson, 2005). Because studies reviewed in the meta-analysis primarily relied on self-

report measures of hypothetical constructs, results discussed should be interpreted with

caution, especially in regards to the direction of causality.

Perfectionism was arguably one of the more consistently reliable and germane

personality characteristics associated with eating pathology (Cassin & Von Ranson, 2005).

However, Canadian researchers Hewitt and Flett noted the complexity of defining

perfectionism and difficulty in measuring it as a singular construct. Consequently three

dimensions of perfectionism were examined (self-oriented, other-oriented, and socially

prescribed), dependent upon the direction and emphasis of the perfectionistic cognitions

and assumptions. Self-oriented perfectionism was described as "self-directed

perfectionistic behaviors...setting exacting standards for oneself and stringently evaluating

and censuring one's own behavior" (p. 457). "The other-oriented perfectionist is believed

to have unrealistic standards for significant others, places importance on other people

being perfect, and stringently evaluates others' performance" (p. 457). Therefore, persons

elevating in self-oriented perfectionism might engender self-criticism and self-punishment

while those elevating in other-oriented perfectionism might employ other-directed blame,

lack of trust, and harbor feelings of hostility toward others. Lastly, "socially prescribed

perfectionism entails people's belief or perception that significant others have unrealistic

standards for them, evaluate them stringently, and exert pressure on them to be perfect"

(Hewitt & Flett, 1991, p. 457). With the construct of perfectionism broken down into three

dimensions more accurate and precise data evaluating perfectionistic behaviors and

psychopathologies became possible.

Perfectionism was measured using the Multidimensional Perfectionism Scale and

data comparing eating pathologies and the three domains of perfectionism were examined

(Hewitt & Flett, 1991). Findings revealed that individuals elevated on self-oriented and

socially prescribed domains of perfectionism often experienced AN-restricting type (ANR),

BN, and BED (Cassin & Von Ranson, 2002). These elevations suggest that persons

experiencing EDs might be experiencing unrealistic personal standards for themselves (as

evidenced by the self-oriented elevations) and believe that others evaluate them harshly

(as evidenced by the socially prescribed elevations) and will, therefore, use eating

behaviors to manage excessive demands for perfection (Cassin & Von Ranson, 2005;

Hewitt & Flett, 1991).

In addition to the Multidimensional Perfectionism Scale, perfectionism was

conceptualized in terms of both adaptive and maladaptive aspects of the personality

characteristic. Results suggested that individuals with EDs show significantly greater over

concern with mistakes and anxiety about performance (termed 'neurotic perfectionism')

and high personal standards and need for order (termed 'normal perfectionism') (Cassin &

Von Ranson, 2005). Further, though some dimensions of perfectionism, such as socially

prescribed perfectionism, may diminish with symptom remission, multidimensional

perfectionism may prospectively predict AN symptom onset and correlate closely with AN,

BN, and BED risk and maintenance factors (Cassin & Von Ranson, 2005; Hewitt & Flett,

1991). One theorist suggested that a diathesis-stress model might explain this link between

perfectionism and BN such that perfectionistic characteristics serve as risk factors for BN

in women who perceive themselves as overweight (Joiner, Heatherton, Rudd, Schmidt,

1997). The diathesis-stress relationship was not significant in women who did not perceive

themselves as overweight, again highlighting the significance of the role of social

perception among perfectionistic persons.

Obsessive-compulsive traits have also been consistently observed in individuals

with EDs. In fact, multiple researchers have found a positive correlation between presence

of obsessive-compulsive traits in childhood and development of ED later in life (Leonard et

al., 1993; Thomsen, 1994). One researcher found that four out of 47 individuals with

childhood onset obsessive-compulsive disorder (OCD) reported a lifetime diagnosis of AN

after a 22 year follow-up (Thomsen, 1994). As stated previously, the lifetime prevalence of

AN among females in the general population is approximately .4%, making the prevalence

of AN among individuals with childhood onset OCD over 21 times more likely, according to

these data. Anderluh et al. (2003) performed a study of 44 women with AN, 28 women with

BN, and 28 healthy female comparison subjects and found that the odds of an ED increased

by a factor of 6.9 for every additional obsessive-compulsive trait present.

Some theories addressing this strong risk factor argue that obsessive-compulsive

personality traits might represent a phenotypic marker for some individuals with AN

(Anderluh, 2003). More specifically, because of the strong developmental continuity and

dose-response relationship between obsessive-compulsive traits and odds of developing an

ED, an individual's genes as well as environmental factors are likely to be strong influences

in the relationship prevalence and presentation. Other theories suggest that commonly

held beliefs among those with obsessive-compulsive personality traits might mirror beliefs

in individuals with ED and therefore impact the increase in comorbidity (Roncero, Perpina,

& Garcia-Soriano, 2011). This theory would fall in line with the Transdiagnostic Theory

(Fairburn, Cooper, & Shafran, 2003), which highlights the maintenance and

psychopathological process commonalities among EDs and suggests that various common

maintenance mechanism factors, such as OCD-related beliefs, might explain these

commonalities.

In addition to perfectionism and obsessive-compulsiveness, neurotic, sociotropic-

autonomous, and narcissistic characteristics were consistently identified in individuals

meeting criteria of AN and BN (Cassin & Von Ranson, 2005). Neuroticism is commonly

considered one of the three critical dimensions of personality (neuroticism, extraversion,

and psychoticism; Eysenck, Eysenck, & Barratt, 1985). In their popular 1987 publication,

Costa and McCrae define neuroticism as "a broad dimension of individual differences in the

tendency to experience negative, distressing emotions and to possess associated behavioral

and cognitive traits. Among the traits that define this dimension are fearfulness, irritability,

low self-esteem, social anxiety, poor inhibition of impulses, and helplessness" (Costa &

McCrae, 1987, p. 301). Interestingly, though a number of articles have found positive

correlations between EDs and neuroticism (Cassin & Von Ranson, 2005; Cervera et al.,

2003; Miller, Schmidt, Vaillancourt, McDougall, & Laliberte, 2006), research examining

over-eating behaviors have found that high scores in neuroticism might act as a protective

factor in that more neurotic persons were more likely to exercise restraint when eating

(Sinclair, Sorrentino, & Weisz, 1990). However, research also suggests that the relationship

between neuroticism and AN and BN might be best explained by moderating variables such

as introversion (Miller et al., 2006) and low self-esteem (Cervera et al., 2003), such that

higher scores on introversion and lower self-esteem might exacerbate an individual's risk

for DE behaviors when the individual also scores high on neurotocism.

Importantly, other researchers have found conflicting evidence of the relationship

between neuroticism and DE. Hollin, Houston, and Kent (1985) administered the

neuroticism scale from the Eysenck Personality Inventory neuroticism-stability (N)

dimension (Eysenck, 1964) as well as an ED inventory and the Social Readjustment Rating

Scale (Holmes & Rahe, 1967) and found a positive but non-significant correlation between

neuroticism and concerns about eating. Interestingly, participants scoring high in

neuroticism reported significantly more undesirable life events, a greater familiarity with

diet literature, and increased dieting, suggesting that neuroticism might function as one

mediating factor between personal environmental contingencies, sociocultural pressures

for thinness, as well as psychological variables (Hollin et al., 1985). Thus, the literature on

neuroticism and eating behaviors appears mixed and more research is needed to further

elucidate the relationship.

Other personality traits that have been examined in relation to eating behaviors are

sociotropic and autonomous personality characteristics. These characteristics were more

commonly reported in individuals with DE behaviors than in clinical controls. Whereas

sociotropy describes individuals who are concerned with acceptance and approval from

others and derive their senses of self worth through relationships with others, autonomy

involves placing great value on personal independence, control, and achievement, deriving

a sense of self worth from independent accomplishments (Cassin & Von Ranson, 2005).

Results from one of the few studies examining the relationship between these personality

characteristics and EDs found that both sociotropy and autonomy were related to BN

elevations (Friedman & Whisman, 1998). However, only sociotropy was found in

individuals elevating in BN when effects of depressive symptoms were controlled,

suggesting that similar to other personality characteristics described, relations may be

moderated by comorbid psychopathological symptoms. Friedman and Whisman's findings

were later replicated in a study examining sociotropy and BN symptoms using two samples

of women: undergraduates and a community sample seeking treatment of ED. Again,

results suggested a significant association between BN and sociotropy, this time including a

clinical sample (Hayaki, Friedman, Whisman, Delinsky & Brownell, 2003). These data

suggested that themes of approval and acceptance are especially influential and salient in

persons elevating in BN symptoms. Further research is needed to explore the relationship

between sociotropy, autonomy, AN and BED.

Similar to theories examining the perfectionism-ED relationship, some researchers have conceptualized sociotropy and ED in a diathesis-stress framework and incorporated other psychopathologies into the model. For example, Oates-Johnson and Clark (2004) completed a 4-week prospective study in which moderating and mediating variables between personality vulnerability, perceived dieting stress, interpersonal appraisals, and increases in dysphoria were examined. Results suggest that highly sociotropic women who perceived social disapproval and were actively dieting due to body dissatisfaction reported greater dysphoria suggesting that social appraising and other sociotropic cognitions might increase dysphoria among individuals vulnerable to body image preoccupation and dieting experiences (Johnson & Clark, 2004). In addition to the diathesis-stress model, the relationship between sociotropy and BN has been explained using a cognitive-personality style (Hayaki et al., 2003). Specifically, themes associated with sociotropy including social dependency, need for approval, and fear of rejection map on to BN presentation such that persons endorsing sociotropic experiences might harbor a cognitive-personality style that fosters BN diagnostic symptomatology (Hayaki et al., 2003) such as binging (criterion A) and compensatory behaviors (criterion B) fueled by a self-evaluation that is excessively influenced by body shape and weight (criterion D).

In addition to examinations of personality characteristics such as neuroticism, obsessive-compulsive traits, and sociotropy, studies on individuals with diagnoses of AN and BN have found that individuals reported more characteristics indicative of narcissism than psychiatric controls as well as groups with other psychiatric diagnoses (Cassin & Von Ranson, 2002). Facets of narcissism within a non-clinical population were studied in a cross-sectional sample of 355 male and female undergraduate students and results suggest

that "vulnerable narcissism" elevations (characterized by hypersensitivity to the opinions

of others, insecurity, desire for approval, and poor self-image) were positively correlated

with DE behaviors (Gordon & Dombeck, 2010).

      In a study that employed a clinical sample of ED clients, women with EDs scored

higher than a non-clinical group in narcissism and reported employing more of the

narcissistically-abused style defenses (i.e. the "poor me" defense in which one portrays

others as abusive and puts others' needs before his/her own) than their non-clinical peers

(Waller, Sines, Meyer, Foster & Skelton, 2007). Interestingly, while the "poor me" defense

was positively associated with restraint, eating concern, body shape concern, and body

weight concern, the "bad you" (manifesting behaviorally as blaming others and criticizing

others' inadequacies) narcissistic defense was positively associated with restrictive

attitudes toward eating (Waller et al., 2007, p. 144). Additional research in the field of

narcissism and eating pathology has found a trend in these narcissistic personality

characteristics persisting even after remission from some EDs (Lehoux, Steiger, &

Jabalpurlawa, 2000). Findings like these raise the possibility that narcissism and other

personality characteristics are enduring traits that predispose individuals to pathological

eating that last beyond remission.

      In addition to the similarities of personality traits associated with AN and BN

described above, results from Cassin & Von Ranson's meta-analysis found significant

personality characteristic differences between AN and BN presentations. These differences

included high constraint and persistence and low novelty seeking among AN individuals

and high impulsivity, sensation seeking, novelty seeking, well as borderline traits in BN

(2005). In general, individuals experiencing AN restricting subtype were found to be less

impulsive than non-psychiatric controls, and individuals with BN diagnoses reported more

personality characteristics suggestive of impulsive tendencies than both AN and non-

psychiatric controls. Similarly, sensation-seeking characteristics were more commonly

reported in individuals with binging behaviors when compared to AN-restricting

characteristics (Cassin & Von Ranson, 2002). These findings highlight the personality

differences between and among eating pathologies, which may explain some of the mixed

results found across studies with ED samples.

Factors Affecting Etiology: Psychopathologies

Numerous studies have revealed comorbidities among eating pathologies and other

psychological disturbances. However, prevalence rates are skewed by the possibility that

seeking treatment is more common when one is experiencing multiple diagnoses making a

patient sample a possible overestimation of comorbid psychopathologies. This bias in

prevalence data is noteworthy when assessing and reviewing ED and other

psychopathology comorbidities.

The personality characteristics described above support research suggesting that

personality disorders are frequently diagnosed among both clinical and community ED

samples. Personality disorders (PDs), particularly cluster C (obsessive-compulsive,

dependent, and avoidant), are common among patients with EDs compared to those with

other axis I diagnosis (Johnson & Wonderlich, 1992; Grilo, Sanislow, Skodol et al., 2003). A

2003 meta-analysis reviewing all published studies of interpersonal dependency and AN

and BN with no publication date limits revealed a statistically significant link between

interpersonal dependency and EDs (Bornstein, 2001). Specifically, PDs most commonly

associated with ANR were avoidant (53%), dependent (37%), obsessive-compulsive

(33%), and borderline (29%). BN diagnoses were commonly associated with borderline (31%), dependent (31%) and avoidant (30%) PD (Bornstien, 2001). However, a significant limitation to Bornstein's analysis was the sole reliance on self-report measures, as self-report measures tend to overestimate the occurrence of the PDs (Modestin, Erni, & Oberson, 1998).

In the first meta-analytic study in which PDs within AN, BN, and BED were assessed using self-reports as well as diagnostic interviews, results generally support Bornstein's findings (Bornstein, 2001; Cassin & Von Ranson, 2002). Still, some discrepancies were found. The meta-analysis could not include studies assessing PDs in BED using self-report due to the paucity of these research data. However, when assessing prevalence rates using self-report measures, PDs associated with ANR were avoidant (50%), dependent (47%), obsessive-compulsive (42%) and borderline (39%). PDs associated with BN were dependent (41%), avoidant (40%), histrionic (33%) and borderline (32%) (Cassin & Von Ranson, 2002). When employing diagnostic interviews, the four studies examining PDs in BED suggest avoidant (11%), obsessive-compulsive (10%) and borderline (9%) PD are the most common. Prevalence rates associated with ANR were obsessive-compulsive (15%), avoidant (14%) and dependent (7%) PD. Prevalence rates among PDs and BN included borderline (21%), avoidant (19%), dependent (10%) and paranoid (10%) PD. Data from this meta-analysis of PDs and various eating pathologies suggest that avoidant and other cluster C PDs are some of the more common PDs among all EDs (Cassin & Von Ranson, 2002). This finding is consistent with the numerous research findings described above suggesting perfectionism, sociotropy, neuroticism and other social-approval and self-

criticism based personality characteristics are especially common in individuals experiencing DE behaviors and cognitions.

The present review aimed to summarize the extant literature on the contributions of personality to eating pathology presentation. Though the inclusion of multiple well constructed and performed studies using varied methods offered a comprehensive picture of the associations, the variations created a challenge in comparing data across studies. This limitation was overcome by integrating data from multiple meta-analyses examining the PD and ED domains. Findings from these meta-analyses corroborated conclusions drawn from single effect size studies while also offering summarizing data revealing trends and themes among personality and eating behavior associations.

In summary, themes across studies include, (1) AN and BN are primarily characterized by perfectionism, obsessive-compulsiveness, neuroticism, narcissism, and harm avoidance; (2) the relationship between these personality characteristics and DE behaviors are better understood when broken down into more specific facets; (3) individuals experiencing ANR symptoms often endorse high constraint and persistence and low novelty seeking personality characteristics. In contrast, (4) individuals experiencing BN often endorse high impulsivity, sensation seeking, and novelty seeking behaviors and cognitions; (5) because of these associations, cluster C personality disorders are most commonly associated with ANR and cluster B and C disorders are more common among those experiencing BN symptomatology. Further research is needed to evaluate the impact of moderating psychological variables, resiliency factors, and status of personality following recovery from ED.

In addition to PDs, a number of comorbid disorders occur with EDs. The National

Comorbidity Survey Replication (NCSR) conducted in 2001-2003 representing 9,282 US

adults found that more than half (56.2%) of respondents with AN met criteria for at least

one of the core DSM-IV disorders assessed in the survey using the WHO Composite

International Diagnostic Interview (Hudson, Hirpi, Pope, & Kessler, 2007). Of the core

disorders assessed in individuals with AN, co-occurring anxiety disorders were the most

prevalent (47.9%), followed by mood disorders (42.1%), impulse-control disorders

(30.8%), and substance use disorders (27%). Major depressive disorder (MDD) was the

most prevalent comorbid diagnosis with 39.1% of individuals with AN also meeting criteria

for MDD (Hudson et al., 2007). In contrast, 94.5% of individuals surveyed who met criteria

for BN also met criteria for at least one of the core DSM-IV disorders assessed in the NCSR.

Anxiety disorders had the highest comorbidity at 80.6%, closely followed by mood

disorders (70.7%), impulse-control disorders (63.8%), and substance use disorders

(36.8%). Interestingly, the most common comorbid diagnosis was a tie with 50.1% of

individuals with BN also meeting criteria for specific phobia and major depressive disorder

(Hudson et al., 2007). Lastly, 78.9% of individuals with BED also met criteria for another

DSM-IV disorder. Anxiety disorders were the most common comorbid diagnoses (65.1%)

followed by mood disorders (46.4%), impulse-control disorders (43.3%), and substance

use disorders (23.3%). The most prevalent comorbid diagnosis was specific phobia

(37.1%) (Hudson et al., 2007).

It should be noted that though data from the NCSR controlled for age, sex, and race-

ethnicity, data were only collected on American adults. As presented in the above section

on EDs demographics, DE behavior presents across cultures and ages. In fact, abnormal

eating behavior and symptoms of EDs typically present between ages 10 to 20 years (Preti

et al., 2009); therefore, data from the NCSR is likely an underestimate of prevalence, cannot

represent comorbidities among US children, and does not assess non-US citizens.

A supplement to the NCSR surveyed a nationally representative sample of 10,123

adolescents aged 13 to 18 years using face-to-face interviews of parents and children and a

modified version of the Composite International Diagnostic Interview (CIDI; Swanson,

Crow, Le Grange, Swendsen, & Merikangas, 2011). Interestingly, AN was not significantly

associated with any psychiatric disorder except oppositional defiant disorder (ODD; $p <$

.05) with 30.4% meeting criteria for the comorbid diagnoses when assessing odds ratios. In

contrast BN and BED were significantly associated with almost every disorder assessed

including mood disorders, anxiety disorders, substance abuse or dependence, and

behavioral disorders (Swanson et al., 2011).

However, when examining percentage data representing the proportion of children

meeting criteria for multiple disorders, NCSR data suggests that of the children with AN

31.7% also met criteria for a behavioral disorder (ADHD, ODD, CD, or any combination

thereof), 23.9% met criteria for an anxiety disorder, 13% met criteria for substance abuse

or dependence, and 10.9% met criteria for a mood disorder. As expected given the odds

ratio data, the most prevalent comorbid diagnosis with AN is ODD (Swanson et al., 2011).

In contrast, 88% of BN children surveyed met criteria for one or more other disorders

including anxiety disorders (66.2%), behavioral disorders (57.8%), mood disorders

(49.9%), and substance abuse or dependence (20.1%). The most prevalent comorbid

diagnosis for children with BN is specific phobia (36.7%). Lastly, among the 10,123

adolescents surveyed 1.6% met criteria for BED and of those youth, 83.5% report

experiencing one or more comorbid diagnoses. The most prevalent comorbid diagnoses were anxiety disorders (65.2%), followed by mood disorders (45.3%), behavioral disorders (42.6%), and substance abuse or dependence (26.8%) (Swanson et al., 2011).

Data from these NCSR studies suggest an influence of co-occurring mental health struggles with DE experiences; however, directionality and causality are difficult to assess. Because of the high comorbidity of ED with other mental health disorders, it is likely that individuals might attribute their eating difficulties to symptoms of another disorder. For example, believing that fear of gaining weight or becoming fat (AN, criterion B) or eating alone because of feeling embarrassed by the amount one eats (BED, criterion B) is related to fear or anxiety about social situations in which one is exposed to possible scrutiny by others (Social Anxiety Disorder, criterion A).

Etiological Theories: Sociocultural Influences

Many researchers postulate that of all of the risk factors for ED in Western societies, sociocultural factors comprise the strongest and most empirically supported influence on the development of body image (Thompson et al., 1999). Women and men alike are prone to feeling dissatisfied with their weight and shape while being exposed to images of abnormally thin individuals in the media, being surrounded by a culture that fears and shuns fatness, and immersion in a world where dieting to achieve thinness is commonplace. Numerous studies have researched the relationship between sociocultural influences and symptoms of EDs such as: clique membership (Hutchinson & Rapee, 2006), the extent to which sociocultural ideals are internalized (Heinberg et al., 2008), peer group age and behaviors (Marcos, Sebastian, Aubalat, Austina, & Treasure, 2013; Wardle & Watters, 2004), and the individual characteristics of the person being effected (e.g., Body

Mass Index (BMI) (Eisenberg, Neumark-Sztainer, Story, & Perry, 2005), ethnic identity

(Tsai, 1999), and self-esteem (Grace, 2002), which are summarized below.

One team of researchers specifically interested in the role of friendship networks

and peer influences found a significant relationship between friend network dieting and

extreme weight loss behaviors (EWLBs) in girls and dieting and EWLB after controlling for

BMI, self-esteem, and negative affect (Hutchinson & Rapee, 2006). Specifically, 1094 female

Australian 7th graders (mean age 12.3 years) completed questionnaires examining eating

behavior, friendship networks, and peer influence. Regression analyses revealed that early

adolescent friendship group members share similar attitudes and behaviors regarding

dietary restraining, EWLBs, and binge eating but not body image concern. However, girls

who were not identified as part of a clique or friendship group had lower self-esteem,

higher BMI, body image concern, and use of EWLBs when compared to their same age

peers who report being part of a clique. Lastly, results suggest that perceived friendship

influence in body image attitudes and eating behaviors are predictive of individual girl's

body image concern, dieting practices, use of EWLBs and binge eating (Hutchinson &

Rapee, 2006). These findings highlight the significant influence of the peer environment on

body image and eating troubles in early adolescence.

The influence of the school environment microculture has also been evaluated in the

context of younger students' exposure to older teenage culture (Wardle & Watters, 2004)

and general peer influence (Marcos et al., 2013). A meta-analysis reviewing research from

multiple databases from 1980 to 2010 found that across 25 studies peers and family

regularly influenced DE attitudes including: dieting behavior, body dissatisfaction and

bulimic symptoms in adolescent boys and girls. Effect sizes were significant, ranging from *r*

= .22 (95% CI: .12 - .32) to *r* = .38 (95% CI: .33 - .43). As expected the impact on dieting

behavior was higher in girls in comparison with boys likely reflecting the general social

pressure to be thin experienced by women more so than men. Additionally, researchers

found that peer modeling had a greater effect on bulimic symptom development than

encouragement to diet and teasing by peers (Marcos et al., 2013).

In addition to the influence of specific peer variables, the average age or grade

difference between the person being affected and the influential peer group is a significant

sociocultural variable (Wardle & Watters, 2004). Specifically one study recruited 200 9-

year-old and 11-year-old girls in junior and middle schools to examine how being in a

younger age group impacts vulnerability to peer influence. The exposure variable involved

the 9-year-olds attending a middle school in which the oldest pupils were 13 years old and

the 11-year-olds attending a secondary school (oldest pupils = 18). The 200 participants

were weighed, measured, and completed a battery of measures involving perception of

ideal size, weight, body esteem, eating attitudes, dieting, and self-esteem. Results suggest

that being exposed to older pupils in school is positively correlated with having a thinner

ideal, feeling more overweight, having more friends who had dieted, scoring higher (more

disordered) on an eating attitudes test, and having lower self-esteem. Researchers theorize

that this relationship is partially due to older children already having internalized cultural

norms for body shapes and consequently displaying these attitudes throughout the school,

including to the younger children. This exposure results in accelerated awareness and

consequent internalization of thin ideals among the younger school population.

Recently researchers examined the influence of various sociocultural variables on

ED development and maintenance and found that a large factor in succumbing to

sociocultural influences is the extent to which the ideals are internalized. Specifically, a

social thin ideal and investment in appearance, beauty, and attractiveness of media images

play important roles in the success of treatment for individuals with EDs (Heinberg et al.,

2008). One study examined initial weight and goal weight in 165 females diagnosed with

ED and treated in a hospital setting and the Sociocultural Attitudes Toward Appearance

Questionnaire (SATAQ; Heinberg, Thompson, & Stormer, 1995) was administered. Results

show an inverse correlation between variables. Specifically, as the degree of internalization

of sociocultural ideals and belief that these ideals are important for successful

recovery/weight gain decrease, gaining and maintaining healthy weight gain increases

(Heinberg et al., 2008). SATAQ items loading onto the internalization subscale include:

''Women who appear in TV shows and movies project the type of appearance that I see as

my goal'' and "I tend to compare my body to people in magazines and on TV" (Heinberg et

al., 2008). Interestingly, the relationship between weight gain and Internalization differed

from partial hospital and inpatient environments. Specifically, patients with lower drive for

thinness and lower internalization of sociocultural standards were farther from their goal

weight compared to patients with higher scores at discharge from inpatient hospitalization.

However, higher drive for thinness scores and higher Internalization was positively

correlated with a greater difference between final weight and goal weight in the partial

hospitalization environment. Researchers hypothesize that this contrast may be due to the

clinical team's influence on determining when to bring patients out of inpatient care.

Specifically, as patients presented with less cognitive symptoms, including less

internalization of ideals and belief that the ideals are associated with success, the treatment

team moved them into partial hospitalization despite them being at a lower weight

compared to the patients with greater investment in distorted cognitive symptoms.

(Heinberg et al., 2008). The role of internalization in long term weight maintenance

following ED hospitalization has not been studied.

The influence of media was further examined in a meta-analysis of laboratory

studies examining the media effects of presentation of ideal physique on ED symptoms

(Hausenblas et al., 2013). Researchers conducted an extensive literature search through

PubMed, Dissertation Abstracts International, and PsycINFO and identified 33

experimental studies in 14 separate meta-analyses examining the effects of acute exposure

to the media's portrayal of the ideal physique on ED symptoms. The independent variable

for all studies involved supraliminal exposure (i.e., "attentive processing whereby stimulus

materials were consciously noticed" [Healy & Proctor, 2003, as cited in Hausenblas et al.,

2013, p. 170]) of the media's presentation of the ideal physique (not just faces). The

dependent variable was an ED symptom outcome measure. Each study included in the

meta-analysis had to have an experimental study design with pre- and post-assessments

for media and a control condition (correlational studies were not included). English and

non-english articles as well as published and unpublished articles were included. Results

include body dissatisfaction, positive/negative affect, depression, anxiety, anger, and self-

esteem ED related outcomes. Viewing idealized images resulted in increases in symptoms

of depression and anger and a decrease in self-esteem. Control groups showed either a

reverse effect (positive effect on outcome involving decrease in depression and no change

in anger and self-esteem) or no effect on ED outcomes. Additionally, moderator analyses

were performed and revealed that individuals with a higher risk for developing an ED were

particularly vulnerable to the negative effects of viewing idealized images in the media

(moderate and significant increase in depression and body dissatisfaction). These findings

result in partial support of the causal effect of viewing idealized media images on

increasing ED symptoms, suggesting that media exposure to images of the ideal physique

results in small changes in ED symptoms (Hausenblas et al., 2013).

Individual characteristics such as BMI (Eisenberg et al., 2005), ethnic identity (Tsai,

1999), self-esteem (Rosenberg, 1965; Obeid, Buchholz, Boerner, & Henderson, 2013), and

religious affiliation (Joughin, Crisp, Halek, & Humphrey, 1992; Smith, Richards, & Maglio,

2004) have been identified as variables influencing the relationship between social and

cultural influences and ED symptom presentation. Specifically, results from a study

examining the role of social norms and friend influence found that girls enrolled in public

middle and senior high schools in ethnically and socio-economically diverse communities

throughout the Minneapolis/St. Paul region were more likely to engage in unhealthy

weight-control behaviors (UWCBs) if they had a higher BMI than their same-age peers.

Additionally, results indicated that friends' dieting behavior was significantly associated

with UWCBs for participants with average and moderately high BMIs. Interestingly, UWCBs

were also associated with the prevalence of trying to lose weight school-wide such that

UWCBs among average weight and moderately overweight girls (according to BMI) were

influenced by school-wide social norms above and beyond the influence of their immediate

circle of friends (Eisenberg et al., 2005). These data have significant implications in the

influence of school and community norms regardless of proximity and in addition to the

influence of BMI relative to peers.

There is a clear positive correlation between self-esteem and well-being, such that

high self-esteem is related to feeling competent or "good enough" and low self-esteem is

suggestive of dissatisfaction with oneself (Rosenberg, 1965). However, the relationship

between ED and self-esteem varies based on ED and symptom endorsement. Specifically,

one study examining self-esteem and social anxiety in a clinical ED population found that

individuals participating in restricting behaviors reported significantly higher self-worth

and perceived physical appearance compared to the subgroup engaging in binge and purge

behaviors (Obeid et al., 2013). Authors hypothesized that this relationship might reflect

that those who engage in restricting behaviors feel more attractive and report greater self-

worth because of attaining a thin stature and reaching weight loss goals. Further, though

those engaging in binging and purging behaviors might lose a relatively small amount of

weight, any increase in self-esteem that could accompany that weight loss might be

overshadowed by experiences of shame and guilt for the binge/purge behaviors (Obeid et

al., 2013).

The relationship between religious beliefs and eating pathology has been present

throughout human history, most notably through descriptions of fasting saints (Joughin et

al., 1992). Though the majority of research examining religion and eating pathology focuses

on how treatment can be modified based on religious preferences, a number of studies

have examined the relationship between eating disorder symptom presentation and

religious practices (Smith et al., 2004). One study examined religiosity among clinical and

non-clinical samples and found that individuals engaging in religious practices for extrinsic

reasons ("for personal and social gains and acceptance" p. 178) tended to have more

ED/DE symptom elevations compared to those with intrinsic motivations for religious

practices or who do not practice religion (Smith et al., 2004).

In regards to specific eating pathologies, one study examining the different ED presentation across religions found that symptoms were largely impacted by the strength of the religious belief and how the subject felt about the strength of their beliefs. Specifically, subjects who reported identifying with a religion, particularly those with strong beliefs and those identifying as Anglican, reported significantly lower BMIs and other symptoms consistent with AN. Results suggest that BMI was significantly negatively correlated to the level of importance of the religion, such that as strength and importance of belief increases, BMI decreases. In contrast, those subjects experiencing symptoms more in line with BN reported weakening religious beliefs (Joughin et al., 1992). These data suggest that religious preferences and practices contribute to eating behavior and might reflect a prescribed restraint present in some religion cultures. That is, as strength and importance of belief increases, BMI decreases because some strong believers might associate fasting or underconsumption with devoutness. Further, those experiencing BN, or overconsumption, are less invested in the underconsumption practices of extremely devout parishioners. This relationship is supported by religious history involving parallels between devoutness and restricting behaviors involving eating, materialism, sexual intimacy, and general self-denial (Huline-Dickens, 2000).

Etiological Theories: Family System

Family functioning has been defined as "the process by which the family operates as a whole, including communication and manipulation of the environment for problem solving" (Mosby's Medical Dictionary, 8th edition, 2009). Similar to the influence of sociocultural factors, the family dynamics experienced by an individual can have impressive impacts on the development and progress of ED and has long been a subject of

interest and research (Laliberte, Boland, & Leichner, 1999; Marcos et al., 2013). Numerous

studies have examined family functioning as it relates to DE and found that a number of

variables impact the extent to which family impacts DE development, ED onset, and

symptom maintenance (Marcos et al., 2013; Holtom-Viesel & Allan, 2014). While many

studies discovered significant relationships between aversive family experience and DE

emersion (Marcos et al., 2013; Holtom-Viesel & Allan, 2014), others suggest that some

family factors such as, parents' interest in their child being thin, frequency of parents'

comments to their child about his/her weight, and maternal dieting behaviors have little

impact on eating behavior (Haines, Gillman, & Rifas-Shiman, 2010). The variety of

conflicting but revealing findings has contributed to family intervention and assessment

practices involving families with members experiencing DE. However, many intervention

findings are muddled by cohort effects, measurement variation, and differing operational

definitions of DE and ED.

Cohort effects were controlled in one longitudinal study of 288 girls ages 9- and 13-

years-old (Westerberg-Jacobson, Edlund, & Ghaderi, 2010). In this study, risk and

protective factors for the development of DE attitudes among young girls were assessed

using self-report questionnaires and the Body Mass Index (BMI; Keys et al., 1972).

Specifically, participants' eating attitudes, BMI, and personality characteristics were

examined along with the girls' parents' eating attitudes and perfectionistic personality

characteristics. Findings for the pre-adolescent girls (9-year-olds) suggest that a "wish to

be thinner" and elevations on fathers' self report of abnormal attitudes towards eating and

dieting behavior (as measured by the Eating Attitudes Test (EAT; Garner & Garfinkel,

1979)) contributed most to the prediction of DE at the seven-year follow-up. Additionally,

analyses of the 13-year-old cohort suggest that a "wish to be thinner" and mothers'

Perfectionism subscale score of the Eating Disorder Inventory 2 (EDI-2; Garner, 1991)

contributed most to the prediction of DE in the 13-year-old cohort seven years later

(Westerberg-Jacobson, Edlund, & Ghaderi, 2010). These data suggest that, though

protective factors such as low BMI, healthy eating attitudes, high self-esteem, and a "low to

medium degree of perfectionism" (p. 215) may act as a buffer against DE, parents' eating

attitudes and behaviors can significantly contribute to the development and maintenance

of abnormal eating behaviors during childhood (Westerberg-Jacobson, Edlund, & Ghaderi,

2010).

In one systematic review of the literature on family functioning and DE, researchers

examined articles on Psychinfo, Web of Science, and Scopus in January 2013 and found 17

qualitative studies in which families with a member with an ED diagnosis were either

compared to control families with no members having an ED diagnosis or families with a

member having different ED diagnosis than the first family. Results suggested families with

a member meeting ED symptom criteria experience worse family functioning (according to

self report data) when compared to control families. Specifically, participants with AN had

less autonomy compared with their non-affected sisters and families with a member with

BN were less flexible (lower amount of adaptability to new or stressful situations) than

control families. However, there was little consistency among all studies in what variables

were affected by the presence of ED. General family function or dysfunction among families

with one ED (AN, BN, or BED) was not significantly different than function/dysfunction

reported in families with a different ED. However, when looking at specific elements of

family functioning, participants with BN reported greater difficulty in planning activities

and confiding in each other than participants with AN-R diagnoses. Additionally, AN

families experienced significantly more cohesion, including to the extent that families with

AN were enmeshed, but were less achievement oriented than in families with BN. Lastly,

families that viewed their functioning positively experienced better recovery outcomes,

irrespective of the severity of the ED (Holtom-Viesel & Allan, 2014).

  A meta analysis reviewing research from multiple databases from 1980 to 2010 and

found a number of studies suggesting that peers and family regularly influence a variety DE

attitudes among adolescent boys and girls (Marcos et al., 2013). Family relations included

mothers, fathers, mother and father, sisters, brothers, sisters and brothers, and "other" (p.

200) and three categories of influence were examined as outcomes (encouragement to diet,

teasing, and modeling/social comparison). Results suggest all adverse family experience

outcome variables impact the onset and maintenance of body dissatisfaction, dieting

behavior and bulimic symptoms. Effects were similar across gender and the influence of

the family in body dissatisfaction was greater in North American families compared to

families from Australia (Marcos et al., 2013). These data suggest that daily encounters with

unique family experiences, especially weight-related issues of parents may influence

unhealthy eating practices among adolescent girls and boys.

  Another influential study regarding family practices and ED involved a two study

analysis in which Study 1 examined the family climate among individuals with a range of

DE severity to determine which family variables cluster together to form a specific family

climate for ED and are distinct from more general family process variables (conflict,

cohesion, expressiveness) (Laliberte et al., 1999). Participants included 121 mother and

student dyads. Students reported on their family experiences and eating practices. Mothers

completed only family measures. Principal component analysis revealed three variables

unique to individuals with ED, these include "perceptions of the family's concern for weight

and shape, perceptions of the family's concern for social appearances, and perceptions of

the family's emphasis on achievement" (p. 1036). Furthermore, Study 1 exploratory

analyses results suggest that the family value of appearance and achievement is

independent from the perception of family general dysfunction. Additional analyses of

specific family climate factors revealed an association between a family

appearance/achievement factor and DE (explaining 19% of the variance) especially dieting

behaviors, and a family dysfunction factor predicted individual self-esteem (explaining

15% of the variance) (Laliberte et al., 1999).

Study 2 examined a clinical sample and compared family perceptions of ED patients

(16 patients with AN-binge/eating/purging type and 24 with BN [the four patients

identified with AN-restricting type were excluded because of the small sample]) with those

of 1) healthy control from a university population and advertising in local hospitals and 2)

psychiatric (depressed) control. The climate specificity hypothesis predicted that both the

healthy and depressed control groups would not report family climate similar to what the

DE students in Study 1 and clinical ED participants in Study 2 report. Results supported the

hypothesis that there is a unique family climate in individuals with EDs compared to a

nonclinical or clinical sample with another psychiatric disorder. These data suggest that a

family climate, particularly the presence or perception of the family concern for weight and

shape, social appearance, and emphasis on achievement, may represent a risk factor for DE

onset and maintenance (Laliberte et al., 1999).

In a more recent study, researchers used structural equation modeling to examine various family factors in the development and presentation of DE (Kluck, 2008). Researchers assessed family dynamics, family food-related experiences and a variety of DE behaviors and cognitions among 268 college women. Family dynamics measured included cohesion, adaptability, overprotection, communication, and affective expression. Family food-related experiences included overall family approach to food and appearance, negative commentary about weight and size and parental modeling on the development of DE behaviors and cognitions. Results suggested both family dysfunction and negative family food-related experiences were associated with increased DE even after controlling for BMI. Additionally, negative family food-related experiences (e.g., modeling dieting, teasing, criticism) fully mediated the relationship between DE and family dysfunction suggesting that, though family dysfunction is correlated with psychopathology, negative family food-related experiences are particular familial risk factors for DE development and maintenance (Kluck, 2008). These data have clear clinical implications in how family dysfunction may be unrelated to DE if negative family food-related experiences are absent.

Due to the widely accepted notion that family practices influence eating pathology among household members, a number of eating behavior interventions assess family dynamics and involve the family in the intervention. Salvador Minuchin's structural family therapy model was at one time a widely accepted method of using family therapy as an intervention (Doherty & McDaniel, 2010). The emphasis on direct observation and coaching distinguished structural family therapy among other family oriented therapies of the time. More specifically, by attending to the balance of family member boundary clarity and permeability, Minuchin distinguished the structural family therapy model from other

first generation family therapies which seemed to prioritize therapist-patient interactions (e.g., Bateson and the Palo Alto Team) and therapist differentiation (Doherty & McDaniel, 2010).

Minuchin's structural family therapy model was the foundation from which a short-term family therapy model for treating and managing DE behavior developed. The Maudsley family treatment model placed caregivers and family members in charge of getting their adolescent family member to adopt healthy eating patterns (Doherty & McDaniel, 2010, p. 74). The Family Dinner Table treatment intervention for adolescents with AN required the family to externalize the ED by conceptualizing the behavior as something that is tricking the teenager rather than being part of him/her. This was the first recorded treatment model for AN using a family therapy model, and preliminary results found it to be an effective method. Seventy percent of patients reached a healthy weight and many females resumed menstruation by the end of treatment (Doherty & McDaniel, 2010). However, the treatment focuses on adolescent females with diagnosed AN. These age, gender, and diagnostic restrictions greatly restrict the population of young adults with whom this treatment can be implemented. Therefore, new family treatment models may benefit from the development of a broader conceptualization of eating pathology and related factors.

Since Minuchin's model was publicized and dispersed in the fields of family psychology and eating behaviors, there have been a variety of studies examining the effect of family meals on eating behavior. Ackard and Neumark-Sztainer (2001) studied the association between BN and family meals among female college students (N= 560). Their investigation involved retrospective reporting of family meal frequency and family

environment, as well as self-reported ED pathology. Results suggest that frequency of eating dinner together as a family is inversely related with elevated scores of BN, indicating that the presence of family meals can have a positive affect on eating pathology. Inversely, the absence of family meals can increase the likelihood of BN symptoms surfacing and maintaining (Ackard & Neumark-Sztainer, 2001).

Much of the research literature that focuses on family dinner practices incorporates enough participants to be generalizable to the average family with an adolescent member (Ackard & Neumark-Sztainer, 2001; Haines, Gillman, Rifas-Shiman, Field & Austin, 2010; Woodruff & Hanning, 2009). However, one study narrowed their participant population to only those families with at-risk youth members (Fulkerson, Kubik, Story, Lytle, & Arcan, 2008). Researchers from this study aimed to delineate the association between family dinner frequency, dietary practices, and weight status using a population of adolescents at-risk of academic failure. Results suggest a positive association between healthy eating behaviors and the availability and consumption of more healthy foods (e.g., less soda and more fruit and vegetables) during family meals. These data suggest that family experiences during meal-time, behaviors observed, family climate (e.g. cohesive and expressive), learning practices (e.g. praise or punishment around weight, shape, eating, etc.), may all influence DE behaviors and even ED development.

Etiological Theories: Biological and Evolutionary Influences

The literature on the impact of family practices and family dynamics on eating behavior unveils the issue of environmental versus biological influences. Specifically, the literature presented above illustrates how family practices influence eating behaviors among children. In the following section the influence of biological characteristics,

including heritable genetics, on DE onset and presentation is discussed.

Many twin studies have examined family concentration features of EDs assuming that monozygotic (MZ) twins (being genetically identical) should show a greater concordance for the EDs than dizygotic (DZ) twins (having only 50% of their genome in common). In a meta-analysis of published familial and twin studies, researchers assessed AN among MZ and DZ twins as well as eating behaviors among the relatives of participating probands (Kipman, Gorwood, Mouren-Simeoni, & Ades, 1999). The estimated heritability of AN obtained from all published controlled familial studies at .72 (n=6) and .71 for all published twin studies (n=59). When examining MZ and DZ dyads, meta-analytic results show that for the total of 95 pairs of MZ twins, 54 are concordant for AN (57%). In contrast, only two of the 79 pairs of DZ twins are concordant for the ED (3%) (Kipman et al., 1999). These data suggest a strong genetic component to AN presentation.

In another twin study evidence of the genetic heritability of BN as well as AN was assessed using a bivariate analysis of a Swedish national twin sample (Bulik et al., 2010). A sample of 7000 Swedish adult females (aged 20-47) from MZ and DZ same-sex twins completed an online DSM-IV diagnostic self-report instrument assessing eating behaviors and ideations. Responses were coded into AN narrow or broad and BN narrow or broad with narrow codes representing more severe DE endorsement (e.g. endorsing "very" or "extremely afraid" when asked how afraid she was that she would gain weight or become fat during "a period of time when [she] weighed much less than other people thought [she] ought to weigh" p.72) and broad codes representing more mild symptom endorsement (Bulik et al., 2010). Bivariate twin modeling analyses revealed a heritability estimate for narrow AN at .57, narrow BN at .62, broad AN at .29, and broad BN at .62. Additionally, a

considerable, but not complete overlap between narrow AN and BN was found (estimated

to be .46). These data suggest substantial genetic heritability for both AN and BN (Bulik et

al., 2010).

When examining what specific symptoms of ED are most heritable, researchers

using 147 MZ and 99 DZ twin pairs found a 42% heritability rate for dieting behavior, 52%

for body dissatisfaction, 44% for drive for thinness. Similar to other twin studies, the

overall DE heritability value was 41% and BMI was 64% heritable. To determine genetic

contributions of specific abnormal eating experiences, researchers administered the EDI

and the EAT and assessed subscale elevations. These data suggest that one's genes not only

may carry heritability for threshold ED, but also predispose individuals to experiencing

specific symptoms of the psychopathologies (Rutherford, McGuffin, Katz, & Murray, 1993).

Less research has been completed examining BED and genetic heritability.

Specifically, though some research examined the heritability of BE (with or without purging

or other compensatory activities), the only analyses that assessed the heritability of BED as

its own psychopathology was completed by Jarvaras and colleagues in 2008. Researchers

completed a family and twin study examining the heritability of BED using 300 probands;

150 with BED and 150 without BED (2008), as well as 888 of their first-degree relatives

(2008). ACE (A = additive genetic effects, C = common or shared environment, E = unique

environment) structural equation modeling was used to analyze data from a case-control

family study of BED. Results suggest that genetics effects are a significant component of

BED presentation. Specifically heritability was estimated at 57% (95% CI: 30-77%) and the

model fit was not improved by including shared environment or sex-specific heritability,

suggesting a strong purely genetic impact (Jarvaras et al., 2008). Researchers speculate that

the genetic factors for BED might be somewhat distinct from and somewhat shared with those for obesity. However, further research is needed to analyze to what extent genetic predispositions for BED overlap with obesity.

In addition to twin studies revealing the genetic heritability of EDs, further data reflect the potential impact of various brain and nervous system abnormalities on ED presentation. In one meta-analysis, brain scans of 228 participants with AN and 240 age-matched healthy controls were compared and significant differences were found when examining the amount of gray matter, white matter, and cerebrospinal fluid (CSF), as well as the size of the hypothalamus, left inferior parietal lobe, right lentiform nucleus, and right caudate (Titova, Hjorth, Schioth, & Brooks, 2013). Participants with AN showed a statistically significant reduction in gray and white matter as well as an increase in cerebrospinal fluid when compared to healthy controls. Regionally, participants with AN presented with reduced hypothalamus, left inferior parietal lobe, right lentiform nucleus, and right caudate size. Each of these regions contributes to appetite experience, somatosensory perception, and functions associated with eating behavior. Abnormalities in these regions, such as those observed in the participants with AN, are likely to contribute to abnormal eating experiences (Titova et al., 2013). Interestingly, some neuropsychology studies suggest that though these effects worsen with prolonged starvation, they may be reversed with weight and hormone stabilization (Mainz et al., 2012)

In addition to neurocircuitry and brain abnormalities in patients with AN, various studies have examined these changes in people experiencing BN. However, many discrepancies exist between study findings. Recent researchers hypothesized that some of the variability in findings may be due to lack of standardization in participants' experience

of hunger before the experiment leading to varying reward sensitivity (Bohon & Stice, 2011). To overcome this potential confounding variable, one study required participants to eat a small snack (Nutri-Grain bar and fruit) 1 hour before participating in the rest of the procedure (rating pleasantness and craving of food and completing an fMRI during food presentation). This unique addition to procedure aimed to control for effects of acute food deprivation. After the 26 females (11 with subthreshold BN, 2 with BN, and 13 healthy controls) completed self-report measures and ate the snack, they were presented with two pictures: a glass of chocolate milkshake and a glass of water. After the picture cue presentation, various participants were either presented with the milkshake, the water, or no solution. This design allowed researchers to measure and identify brain regions activated during expectation of milkshake, expectation of water, actual receipt of milkshake, and actual receipt of water (Bohon & Stice, 2011).

Results indicated that participants with BN show less activity in the right precentral gyrus in both anticipatory and consummatory conditions and less response in the left middle frontal gyrus, right posterior insula, and left thalamus in the consummatory condition when compared to the healthy controls (Bohon & Stice, 2011). These regions are key in experiencing food and hunger. The primary motor cortex is located on the precentral gyrus and is therefore necessary for feeding behavior (Kolb & Whishaw, 2009). The insula is involved in gustatory sensation experiences and the middle frontal gyrus is stimulated in response to taste. The thalamus is considered the relay center between sensations and frontal regions and is therefore associated with reward processing (Kolb & Whishaw, 2009). However, it should be noted that perhaps due to the small sample size (n = 26), these effects were significant at the $p < .05$ uncorrected alpha level; there were no

significant effects found using the most conservative corrected 0.005 level (Bohon & Stice, 2011).

Eating behavior has also been conceptualized through an evolutionary biology perspective, with the etiology of EDs hypothesized to be the result of adaptive changes preparing a woman to survive in an environment that required migration and survival in environmentally depleted locations. For example, Surbey (1987) suggested that because amenorrhea (a symptom previously associated with AN) typically appears before severe weight loss, it might have functioned to delay reproduction until the female was more prepared to procreate and be reproductively successful. More recently, the 'adapted to flee famine hypothesis' considers all symptoms of AN in the context of evolutionary adaptation. For example, theorists postulate that restricting food, denial of starvation, hyperactivity and other AN symptoms are likely evolved adaptive mechanisms lingering from an era in human ancestral past in which nomadic foragers were forced to migrate when food resources became scarce (Guisinger, 2003). During this time, being able to survive and maintain an active lifestyle while starving was an adaptive and life-saving quality. This quality was then passed down through the generations such that current individuals struggling with AN inherited ancestral physiological and cognitive responses to low body weight. Despite the difficulty in empirically testing the validity of evolutionary biology theories, biological and neurophysiological findings would suggest that evolutionary theories such as the adapted to flee famine hypothesis have merit. For example, research indicating the benefit of reacquiring weight and hormone stabilization during ED treatment (Mainz et al., 2012) would support the suggestion that low body weight triggers and maintains symptom presence.

Illness Attribution

Etiological factors are often present in the treatment of EDs as clients frequently enter treatment with pre-existing attributions for why they are feeling or experiencing distress (Addis, Truax, & Jacobson, 1995). Psychotherapy treatment approaches are often influenced by a client's initial understanding of their illness or their personal beliefs and behaviors regarding how they approach their illness based on where they believe it comes from. These causal explanations for distress can range from "complex biological theories to common psychological metaphors" (Addis et al., p. 476). Illness attribution involves the explanation offered to understand the presence of distress or mental disorder (Addis et al., 1995).

In order to assess illness attribution and the ways in which distressing occasions impact drinking behavior the Reasons for Drinking Questionnaire (RFDQ) was created and assessed using factor analysis (n=183) (Zywiak, Connors, Maisto, & Westerberg, 1996). The RFDQ is a 16 item questionnaire that assesses reasons why people may have drunk alcohol just before driving a vehicle. Items range from emotional experiences ("I felt sad") to possible physiological triggers ("I felt ill or in pain or uncomfortable because I wanted a drink"). A subsequent study explored the number of relapses, months since treatment initiation, and elevations on the RFDQ. Results suggest that attributions for relapses vary across time and that while negative affect and craving are the initial attributions to relapse behavior, social pressure is the only remaining attribution 12 months post treatment initiation (Zywiak et al., 2003). These data suggest that not only is obtaining patient perspective informative at treatment onset, it can be helpful to continue assessment across time and monitor how attribution changes throughout therapy.

Similarly, in order to assess illness attribution among clients with depression, researchers developed the Reasons for Depression Questionnaire (RDQ; Addis et al., 1995). This measure includes 44 items clustered into 8 subscales (characterological, achievement, interpersonal conflict, intimacy, existential, childhood, physical, relationship) representing an individual's 'reasons' for their depression experiences. Researchers provided the RFD, a measure of depression, and three measures assessing areas of functioning corresponding to RFD subscales and examined the relationship between certain depression attributions and the tendency to make similar attributions in other areas of life. Results suggested that characterological and existential reasons (e.g. "I am depressed because this is the way I've always been" and "I am depressed because I don't know what I stand for") were associated with both global and stable attributions on the EASQ suggesting that some individuals tend to make similar attributions in other areas of life.

Further research using the RFD explored the ways in which a client's specific reasons for depressive symptoms predict efficacy of certain therapeutic approaches (Addis & Jacobson, 1996; Leykin, DeRubeis, Shelton, & Amsterdam, 2007). Results suggested that clients with existential illness attribution were more responsive to cognitive therapy than behavioral therapy possibly because "the tendency to explain depression in these global and abstract terms may be particularly well matched to a cognitive orientation and directly in conflict with attempts to change specific concrete behaviors" (Addis & Jacobson, 1996, p. 1423). In contrast, the process and outcome of cognitive therapy not as well received by individuals attributing their depression to relationships (e.g. marriage) (Addis & Jacobson, 1996) and those endorsing more biological reasons were less responsive to cognitive therapy than antidepressant medication therapy (Leykin et al., 2007). These findings are

important in that they suggest that regardless of whether or not client and therapist agree

on what may be contributing to the illness, the client's perspective on what is causing their

disorder is relevant to treatment approach and may influence treatment success.

Despite the advances in the therapeutic relevance of assessing illness attributions in

depression and substance use, no such research has been conducted in the field of eating

pathology. As discussed in the above review, there are a number of possible contributing

factors to the development of EDs and the research examining these factors is often mixed.

There are also a variety of cultural, familial, and social barriers to obtaining treatment and

individually tailoring treatment while expanding the changes of treatment success seems

paramount given the severity of health and mental health sequelae of EDs. Further,

National Comorbidity Survey Replication (NCSR) data gathered from a nationally

representative sample of 10,123 adolescents aged 13 to 18 years revealed that for some

individuals, even when they have contact with a mental health provider, ED

symptomatology may be overlooked. Specifically, data from the NCSR for children revealed

that,

> The majority (72.6%-88.2%) of adolescents with eating disorders reported some
>
> contact with the service sector for emotional or behavioral problems, with the most
>
> frequently used sectors being mental health specialty care, school services, general
>
> medical services, and human services. However, only a minority (3.4%-27.5%) of
>
> individuals with eating disorders had actually talked to a professional specifically
>
> about their eating or weight problems. This could be attributable to denial of eating
>
> problems by adolescents, shame and/or stigma, or a lack of recognition of eating

symptoms by professionals treating other targeted problems among these youths.

(Swanson et al., 2011, p. 719)

Regardless of the reasons that ED may not be discussed with providers or detected, it

seems likely that a measure allowing clients to share their thoughts about their beliefs or

reasons for their symptomatology could provide a platform for discussing DE concerns,

etiology, treatment plans, and for explaining and understanding treatment outcomes.

Study 1: Initial Measure Development

Method

The first author generated a preliminary set of items addressing 'reasons for

abnormal eating experiences' based on the extant literature as well as clinical experience

with individuals with EDs and DE. The general format and design of questions and

inventory for the newly developed measure, entitled Inventory for Disordered Eating

Attributions (IDEA), including instructions, questions, response format, and scoring, were

adapted from other measures examining a person's 'reasons' for experiencing

psychological abnormalities. These measures include the RDQ (Addis & Traux, 1995), the

RFDQ (Westerberg et al., 1996), and the Drinking Motives Questionnaire (DMQ; Cooper,

1994; Cooper, Russell, Skinner & Windle, 1992). Format for the inventory included all

questions worded into the first person and formatted as a statement ("I experience

problems with eating because…"). Items are rated on a 4-point Likert-type scale such that 1

= definitely not a reason, 2 = probably not a reason, 3 = might be a reason, and 4 =

definitely a reason. A summative scoring system was incorporated into the inventory to

offer total and subscale scores as well as uniform interpretation.

Piloted items, measure format, and instructions were then vetted through a panel of experts to obtain feedback on the suitability of items, clarity of instructions, and representation of a range of theoretical perspectives on the etiology and/or maintenance of abnormal eating. Requests to join the expert panel were sent via email to 20 professionals who either 1) authored published studies involving ED and etiological factors represented in the measure itself (demographics, personality, psychopathology, sociocultural, family system, and biological/evolutionary influences), 2) are members of the dissertation committee, and/or 3) authored measures examining a person's 'reasons' for experiencing psychological abnormalities. Potential panel members received an email inviting them to participate in the measure vetting process by reviewing and offering feedback on the measure through Qualtrics. The final expert panel was comprised of 8 professionals who completed the Qualtrics measure review.

The expert panel received the pilot inventory and provided feedback on the clarity of the instructions, design of the inventory, and appropriateness of each item as indicative of a potential reason for DE, and if edits needed to be made to improve readability. After obtaining feedback, content validity was calculated using approval ratings from the panel. Questions given at least 70% approval from the panel were retained, resulting in 20 items on the final measure. The 70% approval rating was chosen based off of previous studies using expert panels to vet measures (Alvarenga & Scagliusi, 2010).

Study 2: Psychometric Evaluation of IDEA

Method

Participants

Four hundred and twenty four participants were recruited from the Introduction to Psychology courses at the University of Montana: a medium-sized, northwestern university. Participants received course credit for their participation in research. Sample size recommendations for principal components analysis (PCA) range from 5 – 15 participants per variable (items from Study 1) (Fields, 2009). However, when completing PCA the reliability of the resulting factors increases with the increase in sample size (Leech, Barrett, & Morgan, 2011). Therefore, the larger sample size (N = 424) was obtained. Institutional Review Board approval for both online and in-person data collection was obtained prior to beginning the study.

Measures

Participants completed the final IDEA items from Study 1, the EDE-Q, and a brief demographic questionnaire. The demographic questionnaire asked participants for their gender, age, ethnicity, sexual orientation, and religious affiliation (see Appendix C).

Eating Disorders Examination Questionnaire (EDE-Q; Fairburn, & Beglin, 1994). The EDE-Q (see Appendix B) is a self-report questionnaire used to assess DE behavior and accompanying ideations. The questionnaire contains 36 items and examines the prevalence of AN, BN, BED, and EDNOS. The EDE-Q has a seven-point, forced choice scale and four subscales: *Shape Concern* (e.g., "Has your shape influenced how you think about (judge) yourself as a person?"), *Weight Concern* (e.g., "Have you had a definite fear that you might gain weight?"), *Eating Concern* (e.g., "Have you had a definite fear of losing control over eating?"), and *Restrained Eating* (e.g., "Over the past 28 days, on how many days have you eaten in secret (i.e., furtively)?). Participants respond to each item based on its application to their subjective experiences or behavior, based on a response format of "not at all,"

"slightly," "moderately," or "markedly". Scores range from 0-6 on the EDE-Q and higher

scores are associated with more severe DE behaviors (Fairburn & Beglin, 1994).

Procedure

Participants recruited from the Psychology Department research participation pool

were asked to provide their informed consent outlining the process, risks, and contact

information for the study. Participants were notified that participation is voluntary and all

responses would be anonymous. Additionally, all participants were provided with contact

information if they had any questions and provided with a list of referral agencies,

including the University's counseling center, for coping with any distress resulting from

participation in the study. No participants contacted the first author regarding complaints,

distress, or difficulties regarding their study participation.

Following the informed consent, participants were asked to complete the IDEA,

EDE-Q, and demographic questionnaire. Lastly, a subset of the sample (*n* = 21) were invited

to re-take the IDEA two weeks after the initial data collection in order to assess test-retest

reliability. The retest participants were selected randomly from the pool of original

participants who indicated interest in participating in the re-test for more course credit.

This sample was again asked to provide informed consent and complete the IDEA a second

time.

In the initial data collection for study 2, participants were first presented with an

introduction paragraph (see Appendix A), followed by the question, *When have you*

experienced difficulty with or unhealthy thoughts, feelings, or behaviors related to eating?

Response options for this item included *currently, in the past, both (now and in the past),*

and *never (discontinue questionnaire).* During the first data collection for study 2, 60.2% of

participants selected *never (discontinue questionnaire)* and did not complete the remaining

items on the IDEA. It is likely that participants opted out of the IDEA because they do not

believe they have ever experienced difficulty with or unhealthy thoughts, feelings, or

behaviors related to eating. It is also possible that participants saw the 'opt out' option as

an opportunity to end their participation in a more timely manner. However, given that

14.2% of these participants had EDE-Q T-scores in the clinical range (above 1.5SD above

the mean), it is likely that many individuals have experienced disordered eating thoughts,

feelings, or behaviors but may not have sufficient insight to describe them as 'difficulties' or

'unhealthy.' Therefore, for the next data collection stage (using online instead of in-person

methods), the initial IDEA item was modified such that the opt-out option was removed

and participants were given Likert-style response options. The modified IDEA item 1 read,

Most people have gone on diets, tried to improve their eating habits, or had difficulties with

eating in some way (eg. Eating too much or too little). To what extent have you experienced

any of these problems? See Appendix A. Response options for this item included *not at all,*

very rarely, rarely, often, and *very often.* The online data revealed that 23.2% of participants

reported experiencing difficulty with eating "not at all", 17.4% experience it "very rarely",

25.6% experience it "rarely", 27.5% reported "often", and 6.3% experience eating

difficulties "very often". Approximately fourteen percent (14.2%) of the participants

reported symptoms of abnormal eating that fell within the clinical range (T-score $\geq$ 65) on

the EDE-Q. See Table 1 for percentage of individuals with EDE-Q T-scores above 65 relative

to demographic information. With 27.5% of online participants reporting eating difficulties

"often" it is likely that the data from phase 1 of Study 2 data collection where 60.2% of

participants "never" experience disordered eating was invalid.

INSERT TABLE 1 HERE

Data Analytic Strategy

Statistical analyses were conducted using SPSS 22.0 (SPSS: An IBM Company). Data

from the first and second administration of the IDEA, EDE-Q, and demographic

questionnaire were compiled to evaluate the IDEA's internal consistency, test-retest

reliability, and convergent validity, and to identify domains of attribution through factor

analysis. Specifically, PCA with varimax (orthogonal) rotation was conducted to assess the

underlying structure for the items of the IDEA such that information explained by one

factor is independent of the information in the other factors. Obtained eigenvalues greater

than 1.0, which account for a substantial proportion of the variance, were considered for

possible factors (Leech et al., 2011). Additionally, cross loadings were calculated to

determine what percentage of the variance from the original items is accounted for by the

factors. Items were retained or omitted from the IDEA based on their loading onto a single

factor. Items loading onto more than one factor were retained on the factor on which they

loaded most highly, which also was the best theoretical fit. Items were then summed to

create subscale scores and a total score. Finally, to assess the association between current

DE and the tendency to endorse DE attributions, EDE-Q scores were compared to IDEA

scores using Pearson's correlation coefficient.

Results

Results for the current study yielded a systematically developed measure with

sound psychometric properties. Items were vetted through a panel of 7 professionals in the

fields of illness attribution, eating pathology, health psychology, and measure development.

All items from Study 1 were retained under the 70% approval rating through the expert

panel. Twenty-five percent of the items were edited for clarity based off of expert panel

feedback. The resulting 20-item measure was distributed to 424 undergraduate students at

a medium size northwestern university. Participants also completed the EDE-Q and a

demographic questionnaire. See the appendix for IDEA (Appendix A), EDE-Q (Appendix B),

and demographic questionnaire (Appendix C).

Participants

A total of 424 registered students from The University of Montana participated in

the study and 21 of the original participants were randomly selected to complete the retest

two weeks after the original data was collected. See Table 1 for a description of participant

demographics.

Missing Data

Missing Data: EDE-Q

Just under 4% (3.72%) of the 28 nominal EDE-Q items were missing. While there is

not currently a consensus regarding what percentage of missing data is acceptable

(Schlomer, Bauman, & Card, 2010), one of the most conservative estimates, Schafer (1999),

recommends a 5% cutoff. This conservative estimate suggests that the 3.72% of missing

EDE-Q data will not adversely affect analyses. Using the expectation maximization

algorithm (Schlomer, Bauman, & Card, 2010), each missing item score was replaced by the

mean score of the respective EDE-Q subscale in which an item fell. The expectation

maximization strategy was utilized because its recursive and iterative process makes it a

superior method for exploratory factor analysis with a large sample size (Schlomer,

Bauman, & Card, 2010).

Missing Data: IDEA

In part because 60.2% of participants from the initial data collection did not

complete the IDEA, 24.7% of IDEA items were missing from the total sample (the initial in-

person collection stage (n = 211) and the following online data collection (n = 213)). With

this high missing data rate, employing the expectation maximization algorithm might lead

to biased IDEA analyses. Therefore, a listwise extraction method was applied for missing

IDEA data. In this extraction method, all data from participants with missing IDEA data

were extracted from analyses. Using the listwise extraction, data from 307 participants

were incorporated into analyses involving the IDEA. The sample size of 307 is still well

above the recommended sample size for a PCA with a 20-item inventory (Fields, 2009).

Analyses that did not involve the IDEA (e.g. mean score substitution for the EDE-Q and

EDE-Q T-score analyses) incorporated all data regardless of IDEA missing items.

Principal Component Analysis (PCA)

PCA: Analyses Specifications

A PCA with varimax rotation was completed. Specifications applied to the PCA

included: (1) listwise extraction such that only cases with complete data were included in

analyses (N = 307, 72.4% of the 424 full sample); (2) a factor loading criterion of .80 for

items to be retained for subsequent analyses, which is the general recommendation for an

exploratory factor analysis with a large sample (Fields, 2009); and (3) a varimax rotation

was applied because the items were not highly correlated with each other (inter-item

correlations ranged from .013 to .755 with a mean of .304).

PCA: Factors Extracted

Using a PCA extraction method and varimax rotation, a four-factor model within the

IDEA was revealed. Using an eigenvalue of 1 the rotation converged in 6 iterations. The

four factors extracted included items related to: (1) Psychopathology, (2) Personality, (3)

Sociocultural influences, and (4) Adaptability (see Table 2 for rotated factor matrix output,

listed in order of size of variable factor loadings). Item number 12 (attributing disordered

eating to feelings of anxiety) loaded onto factors 1 and 2. It was retained on Factor 1

because its Factor 1 loading was stronger than its factor 2 loading and because,

theoretically, it makes more sense for an anxiety illness attribution to contribute to the

psychopathology factor similar to how depression and other mental health concerns are on

Factor 1. The four-factor solution was further supported by the scree plot results (see

Figure 1).

INSERT TABLE 2 HERE

INSERT FIGURE 1 HERE

PCA: Factor Psychometrics

The full scale IDEA included 20 items with 6 items in subscale 1, 5 items in subscale

2, 7 items in subscale 3, and 2 items in subscale 2. The Kaiser-Meyer-Olkin statistic was .89

and Bartlett's Test of Sphericity was highly significant (p<.001) suggesting that a PCA was

appropriate for these data (Fields, 2009). The mean score for each subscale is 5.13, 7.07,

5.26, and 1.42 respectively. The mean score for the full scale was 19.03. Subscale

Cronbach's Alphas were .86, .86, .75, and .56, respectively (Cronbach, 1951). The IDEA full

scale possessed a strong reliability of .90 (an alpha coefficient greater than or equal to .60

indicates adequate internal consistency; Anastasi, 1988). The IDEA full scale and subscales

1, 2, and 3 possess adequate reliability. Subscale 4, Adaptability, falls below Anastasi's

criteria. See Table 3 for number of items, mean score (SD), and Cronbach's Alpha data for

the IDEA full scale and subscales.

INSERT TABLE 3 HERE

Psychometric Properties of the IDEA

     Internal Consistency Reliability

Data from 307 of the 424 participants were included after listwise extraction to

calculate Cronbach's Alpha and inter-item correlation results of the IDEA. The IDEA

revealed a Cronbach's Alpha of .90 suggesting that 90% of the variability in a composite

score is internally consistent and reliable variance. This is above the generally acceptable

.70 Cronbach's Alpha (Lance, Butts, & Michels, 2006). The strong Cronbach's Alpha was

supported by suitable inter-item correlation data. Specifically, the inter-item correlation

matrix revealed positively correlated items suggesting, as predicted, that all items measure

the construct of 'disordered eating.' Further, inter-item correlations ranged from .013 to

.755 with a mean of .304 reflecting strong correlations within subscales without so much

correlation that items were measuring the same variable (Ferketich, 1991). See Table 4 for

inter-item correlation data.

INSERT TABLE 4 HERE

Test-Retest Reliability

A paired-samples t-test, or dependent t-test, was conducted to assess the IDEA test-retest reliability. In this analysis 21 of the of the original data collection sample were invited to complete the IDEA again, two weeks after the first test day. However, 11 of the 21 retest participants did not complete the IDEA in the original data collection; they reported never experiencing DE and therefore discontinued the questionnaire. Therefore these participants' retest IDEA data was excluded from retest analyses, leaving a sample of ten retest participants.

A paired samples t-test was conducted to determine if the subscale and full scale scores between time one (T1) and time two (T2) were significantly different. As predicted, correlation results reveal a positive correlation between most subscales scores at T1 and T2, suggesting low standard error. Specifically paired samples correlations for subscales 1 through 4 and the full-scale correlation were .684, .637, .933, -.147, and .658, respectively. Significance values revealed statistically significant retest correlations at the .05 demarcation in subscales 1, 2, 3, as well as the full scale score. The significance value of subscale 4 was above .05 (p = ns), suggesting low test-retest reliability. See Table 5 for paired samples correlations data.

INSERT TABLE 5 HERE

Convergent Validity, IDEA and EDE-Q Regression Analysis

Because past research has indicated that individuals in higher levels of distress are more likely to offer reasons or provide explanations for their difficulties than individuals in lower levels of distress (Addis et al., 1995), it was predicted that higher scores on the IDEA subscales would be significantly positively correlated with higher scores on the EDE-Q. Regression analyses of the IDEA and EDE-Q revealed a positive correlation, r = .58 (p<.01, 1-tailed) indicating "moderate" convergent validity (Dancey & Reidy, 2004). See Figure 2.

INSERT FIGURE 2 HERE

EDE-Q: Establishing Standardized Scores Using Normative Data

EDE-Q results were comparable to normative data from a college sample (Luce, 2008) and, as predicted, more disordered than the normative community sample data (Fairburn & Beglin, 1994). Specifically, the EDE-Q was first collected in a college sample in 2008 (Luce, 2008). This sample was comprised of 723 undergraduate women enrolled in general psychology at a large Midwestern university. The EDE-Q Global Score mean (SD) for this population was 1.74 (1.30), which was slightly higher relative to the current sample's Global Score mean, M=1.67 (1.34). The slight difference could be impacted by the inclusion of male gender participants in this data sample, as males typically reports less abnormal eating thoughts and behaviors compared to females (Hudson et al., 2007 as cited in *DSM-V*, APA, 2013).

As predicted, the college sample participants in the current study reported more disordered eating thoughts, feelings, and behaviors on the EDE-Q when compared to the

original normative data collected through a community sample (Fairburn & Beglin, 1994).

The community-based sample included 243 young women and revealed a mean Global

Score of 1.55 (1.21). See Table 6 for means (SD) all total and subscale comparisons.

INSERT TABLE 6 HERE

At the time of the current study, the EDE-Q did not have a published cut-off score

indicating what total scores constitute clinically abnormal eating thoughts, feelings, and

behaviors. Therefore, a T-score was computed to assess the extent to which participants

reported eating disordered symptom experiences in a clinical range (1.5 SD above the

mean). T-score analyses revealed that 14.2% of the current sample was reporting clinically

significant ED symptoms.

T-scores were also calculated for the individual EDE-Q subscales. Individuals with T-

scores $\geq$ 65 were considered in the clinical range. Regression analyses reveal significant

relationships between elevated (T-score $\geq$ 65) scores on the EDE-Q subscale Restraint and

IDEA factors 1 and 4 (p < .01) as well as the IDEA global score (p < .01). There is also a

significant positive correlation between EDE-Q subscale Eating Concern and IDEA factor 1

(p < .01) and the IDEA global score (p < .05). See Table 7.

INSERT TABLE 7 HERE

Discussion

The current study included the development and evaluation of a new measure for illness attribution in the DE population. This is the first measure assessing self-reported reasons for DE experiences and offers valuable information to providers, clients, and researchers in the healthcare field.

I: Inventory for Disordered Eating Attributions (IDEA)

Findings from the current study resulted in the creation of a brief self-report assessment which provides individuals with an opportunity to report on what they believe contributes to their current difficulties with DE. The self-report measure format provides individuals in a clinical setting with more time to introspect and privately reflect on possible reasons for ED, compared to being asked about illness attribution in a clinical interview. Though there are instances where clinical interviews may help providers to attain more reliable and accurate information (e.g. when assessing constructs that are difficult to define) (Fairburn & Beglin, 1993), numerous studies have found that individuals are more forthcoming and honest about their behaviorally-specific or more well-defined experiences on paper or computer-based measures compared to an interview assessment (Fitcher & Quadflieg, 2000; Keel, Crow, Davis, & Mitchell, 2002).

The PCA revealed a four-factor solution with all 20 items created in Study 1, explaining a significant amount of the variance after rotation. Therefore all 20 items of the IDEA created in Study 1 were retained in the full inventory and the four factors were assessed for similarities.

Factor 1: Psychopathology Illness Attribution

Factor 1 includes items 12, 13, 14, 15, 16, and 19 (see appendix for IDEA items).

Aside from item 19, each of these items that loaded onto Factor 1 reflect an illness

attribution related to psychopathology. Suggesting that individuals that elevate on Factor 1

items believe that another mental health concern is adversely affecting their eating

behavior. While item 19 ("my brain functions differently in a way that changes my thoughts

and behaviors around eating") strongly loaded onto Factor 1 (.697), it does not seem to

directly align with a diagnosable disorder attribution as all of the other Factor 1 items. It is

hypothesized that participants endorsing other Factor 1 items such as depression or

substance abuse attributions also see their mental health difficulties as a brain disease, or

may be knowledgeable about treatment approaches involving cognitive and behavioral

elements, and therefore endorsed item 19 along with other psychopathology items.

Factor 2: Personality Illness Attribution

As discussed in the Introduction, eating-related thoughts and behaviors can often

times be understood in the context of personality. With the exception of item 2, the items

that loaded highly onto Factor 2 (2, 7, 8, 9, and 11) all directly relate to a specific

personality characteristic (e.g., perfectionism, obsessiveness, and desire for approval). Item

2, related to media exposure, seems less associated with personality and a priori

hypotheses placed this item in the psychosocial attribution factor. It is possible that those

individuals who self-report being influenced by the media may also experience strong

personality characteristics (such as perfectionism) that relate to DE attribution. For

example, while someone with non-perfectionist personality characteristics might view

media of idealized models and not be adversely effected, someone who is strongly

perfectionistic, or experiencing the self-oriented perfectionism described by Hewitt & Flett

(1991) (see Personality section above), might see that same media image, internalize that

idealized beauty, and fuel unhealthy eating and body image thoughts, feelings, and

behaviors.

Factor 3: Psychosocial Illness Attribution

Items that loaded onto Factor 3 (1, 3, 4, 5, 6, 17, 18) are all related to psychosocial

pressures influencing eating (e.g., family, religion, friends). Interestingly, with the exception

of Factor 2 (*personality factors*), individuals with subthreshold DE (T < 65 on the EDE-Q)

elevated on this factor more than any other scale (see Table 7). This correlation could be

explained by the co-relationship between personality and susceptibility to psychosocial

influences. For example, the illness attribution item that has the strongest loading onto

Factor 3, psychosocial, is "my friends." The item that has the strongest loading onto Factor

2, personality, is "my sensitivity to others' thoughts about me and my appearance" (see

Table 5 for PCA factor loadings). Thus, it is reasonable to assume that if someone endorses

a personality characteristic that involves sensitivity to other's thoughts then they are also

likely influenced by the social influences of friends and family. Further, this finding

suggests an important observation in the non-clinical population of individuals who may

have DE thoughts or behaviors. Specifically, these individuals are highly impacted by social

influences and prevention of DE, ED, and body dissatisfaction problems may be best

addressed via a public health approach rather than individual strategies.

Interestingly, the relationship between Factors 2 and 3 was not present in the

subset of individuals in the current sample who scores in the 'clinical' range on the EDE-Q

(T-score $\geq$ 65). Indeed, regression analyses suggest that there is an inverse, but non-

significant, relationship between individuals in the clinical range EDE-Q global score and

Factor 3 (psychosocial influences). This could be due to the small sample size of

participants in the clinical range of the EDE-Q (N = 60). It is also possible that this finding

supports research suggesting that individuals experiencing diagnosable ED might be more

affected by comorbid psychopathologies or beliefs that restrictive eating is necessary for

survival than social-culture influences.

Factor 4: Evolutionary Adaptability Illness Attribution

The PCA extracted two items for Factor 4 (items 10 and 20) involving attributing

eating behavior to the ability to push one's body to extremes and the belief that one's

eating behavior is effected by an evolutionary adaptation to survive by modifying eating

thoughts and behaviors. While there is incongruity in the literature, some statisticians

suggest that a factor or subscale with "few loadings" is probably not reliable unless the

sample size is over 300 (Fields, 2009, p. 650). Further, likely because there are only two

items on this factor, the test-retest analysis suggests that Factor 4 is not stable over time

(see Table 3). However, despite only having two items on Factor 4 and inconsistent test

retest data from T1 to T2, the factor has an eigenvalue of 1.52 and explains 7.59% of the

total variance. Additionally, KMO (.89) and Bartlett's Test of Sphericity (p < .001) results

suggest the four-factor solution, including the two items on Factor 4, is appropriate for the

IDEA (see Table 5). Further, item-analyses reveal that if either of these items were removed

from the inventory, the IDEA Cronbach's Alpha would lower suggesting that the measure

would become less reliable or consistent with the removal of either of these items.

Therefore, Factor 4 will be retained in the final IDEA solution.

II: IDEA Influencing Perception of DE Etiology

Results from this study inform our understanding of perception of ED etiology. Though there is no clear trigger or explanation for what causes the onset of ED for each individual, there are decades of research assessing possible risk factors and experiences which contribute to symptom development (Marcos et al., 2013). As seen in Table 7, the most common illness attribution in the sub-clinical college population is personality followed by psychosocial and psychopathology attributions. These data support past research indicating that personality's effect on DE may even be moderated by comorbid social and mental health experiences (e.g., Friedman & Whisman, 1998). Further, this suggests that individuals with sub-clinical eating experiencing attribute most of their eating thoughts, feelings, and behaviors to their personality. Interestingly, personality factors are typically considered to be stable and difficult to change, which may account for the degree of DE behaviors and general body dissatisfaction that persists in the normal population (Hampson & Goldberg, 2006). Lastly, researchers studying obsessive-compulsive personality traits within the ED population found that the odds of an ED presentation increased by a factor of 6.9 for every additional obsessive-compulsive trait present. It seems likely that if there is this strong of a correlation between diagnosed OCPD (and other PDs) and diagnosed eating pathology, it is likely that personality factors that are not diagnosably abnormal would contribute to subclinical eating thoughts, feelings, and behaviors (Anderluh et al, 2003).

In contrast to the sub-clinical population, the significant relationships between EDE-Q subscales and IDEA factors suggest that individuals experiencing clinically significant *eating restraint* and *eating concern* are likely attributing their experiences to other mental health difficulties ($p < .01$; Factor 1). The relationship between ED and psychopathology is

well supported in the literature. For example, the NCSR data, representing 9,282 US adults

found that 56.2% of respondents with AN and 94.5% of the respondents with BN, and

78.9% of individuals with BED also met criteria for at least one other DSM-IV disorder

(Hudson et al., 2007). The PCA adds to this research by revealing that a significant number

of individuals actually attribute their abnormal eating experiences to a co-occuring mental

health problem.

The clinically elevated *eating restraint* scores were also significantly correlated with

the evolutionary adaptiveness illness attribution ($p < .01$; Factor 4). The *restraint* subscale

of the EDE-Q encompasses eating thoughts and behaviors that are restrictive in nature, not

involving binging thoughts and behaviors (e.g. avoidance of eating, dietary rules, desiring

and empty stomach). The evolutionary scale includes items related to being able to push

one's body to extreme states (such as starvation or over exercise) and being adaptively

designed to consume very little food while maintaining an active lifestyle. Denying oneself

basic human drives, such as responding to hunger, is extreme in nature; therefore it is

logical that individuals reporting these types of thoughts and behaviors would be elevated

on the evolutionary scale. The relationship between EDE-Q *restricting* subscale and IDEA

Factor 4 suggests that it is more likely that people who attribute their eating experiences to

a natural drive to push one's body to extremes will struggle with *restraining* or over

restricting more so than *shape concern, eating concern, or weight concern*. This suggests

that the *restraint* scale might represent the behavioral manifestation of an evolutionary

adaptiveness mindset.

The EDE-Q *eating concern* subscale was also significantly correlated with IDEA

Factor 1 (Psychopathology) ($p < .01$). Items on the EDE-Q *eating concern* subscale are

related to preoccupation with food, eating or calories, fear of losing control over eating,

eating in secret, social eating, and guilt about eating. Items on this subscale reflect aspects

of ED related to shame, guilt, and anxiety around eating. It is therefore understandable that

someone in the clinical range of the eating concern subscale would attribute his or her

eating difficulties to other psychological struggles, such as depression or anxiety.

In sum, findings from the current study suggest that (1) individuals experiencing

subclinical DE attribute their eating thoughts, feelings, and behaviors to personality and

psychosocial factors more so than psychopathology or evolutionary adaptability influences,

(2) individuals reporting eating thoughts and behaviors in the clinical range are more likely

to attribute their illness to mental health difficulties and adaptability to extremes, and (3) it

is not uncommon for eating thoughts and behaviors to be influenced by multiple and varied

factors. Importantly, believing that eating experiences are influenced by a need to push

one's body to extremes or the adaptive nature of being able to survive with a restricted diet

might be indicative of a less common and more clinical symptom presentation. The IDEA

will provide researchers and practitioners with clearer data regarding which of these

attributions clients endorse.

III: IDEA Informing Treatment

When used in a health care setting, this measure may offer providers information

regarding case conceptualization and treatment planning similar to what the RFD has done

for depression (Addis & Jacobson, 1996). For instance, perhaps those who score high on

psychosocial-oriented reasons for DE (Factor 3) will benefit more from a treatment that

focuses on decreasing susceptibility to environmental provocations and increasing self-

compassion regardless of body shape, such as an emphasis on cognitive rather than

primarily behavioral approaches. Or, similar to results found using the RFD, it may be that

individuals who endorse more existential reasons for DE (e.g. IDEA item 20 involving the

evolutionary adaptability of irregular eating) will benefit from a cognitive approach than

they would with a strictly behavioral therapy (Addis et al., 1996).

Limitations

The limitations of this study include the sampling procedure, which is limited to a

relatively homogenous sample of university students who self-selected into the study

making generalizability of the findings more difficult. However, a college age population is

frequently used in ED/DE research. Additionally, because of the wide variety of theories

regarding ED etiology, there may be themes related to ED/DE origin that are not included

in Study 1 and therefore not incorporated into the IDEA. However, the etiological factors

presented (from which IDEA questions were derived) represent the major factors

influencing ED/DE with the most empirical research supporting their influence on eating

behavior.

Conclusions drawn from the sample of participants who scored in the clinical range

of the EDE-Q are limited because only 60 participants elevated to $T \geq 65$. Further, the EDE-

Q does not have a published clinical cut-off and using only the one T-score calculated for

this measure to assess clinical severity could provide an overpathologized view of this

population. However, this T-score calculation created an opportunity to collect illness

attribution data in a pseudo-clinical sample, which has never been done before.

Additionally, despite the limitations of only having one T-score and a relatively small

sample elevating into the clinical range, the resulting analyses examining the EDE-Q against

the IDEA were compelling and should not be overlooked.

Future Directions

There are a number of directions for future research relevant to the IDEA measure. First, future research should examine the newly developed measure within more heterogeneous samples (e.g., ages, ethnicities, etc.). Second, a next logical step to this work would be examining the IDEA in an exclusively clinical population to determine if the results of the current study can be replicated according to the hypotheses drawn (more described below). In addition, future research may include subjecting the obtained factors to a principal axis factor analysis with varimax rotation in order to ascertain possible higher-order factors that account for more of the variance. It would also be useful to consider exploring options for adding evolutionary or extreme behavior items to Factor 4 in order to create a more reliable, robust factor to help inform treatment.

In addition to these suggestions, future research with the IDEA might also include rerunning a test re-test analysis with more participants as well as with a measure to establish discriminant validity. As stated previously, 11 of the 21 retest participants called back had not fully completed the IDEA at T1 because they were given (and endorsed) the opt out option. Therefore, while most factors had reliable test-retest data, factor 4 did not reach significance. Perhaps with more items added to factor 4 and a greater number of test retest participants, that factor's reliability will reach significance. Further, because this study was exploratory in nature given that it was a test of a new measure, no hypotheses were made regarding concepts with which the IDEA would have low correlation. This is particularly true given the breadth of items developed in Study 1. However, despite the exploratory nature, the KMO (.89) and Bartlett's Test of Sphericity ($p < .001$) results

indicate excellent sampling adequacy was reached for this four-factor solution, and results are interpretable.

Lastly, it would benefit both the clinical and research community if norming data for the IDEA was collected from a large clinical population. The ED literature suggests that there are certain personality characteristics (Hayaki et al., 2003), psychopathologies (Swanson et al., 2011), and social experiences (Joughin et al., 1992; Obeid et al., 2013) that correlate with BN more so than AN and vice versa. Therefore, future research should explore the extent to which different illness attributions are more commonly endorsed in different ED populations. These data could then inform what type of therapeutic intervention would be most beneficial for patient's presenting with specific ED profiles. Specifically, the IDEA may be used to explore how clients' reasons for DE contribute to the process and outcome of various ED therapies. This may then inform how reason-giving conceptualizations match certain theoretical models and predict therapy buy-in, progress, and outcome.

Conclusion

Eating pathology is currently one of the most difficult to treat and fatal psychiatric disorders (APA, 2000). Relapse rates are as high as 63% (Field et al., 2008) and research focused on understanding risk factors as well as best practice for treatment is conflicting (Marcos et al., 2013; National Institute for Clinical Excellence, 2004; Turner, Tatham, Lant, Mountford, & Waller, 2014). However, the field of illness attribution has had success in using patient reason giving to inform treatment, particularly with depression (Addis et al., 1995). The current study aimed to create and validate a measure assessing illness attributions among DE individuals. Study 1 involved creating a new measure, the Inventory

for Disordered Eating Attributions (IDEA), with items based on the available literature and

vetting the items through an expert panel comprised of 7 individuals in the ED, illness

attribution, patient care, and health psychology fields. Results from Study 1 yielded a 20

item measure.

In Study 2, the principal investigator administered the IDEA, along with a measure of

eating disorder symptoms and behaviors (EDE-Q) and a demographic form, to 424

individuals. The results were assessed using a PCA with varimax rotation and the resulting

4-factor solution was analyzed for psychometric soundness. A T-score was calculated for

the EDE-Q global and subscales in order to assess the IDEA using a pseudo-clinical sample.

These data suggest that there are varying patterns and themes within a college

population regarding eating experiences. Broadly, participants scoring in the clinical range

on the EDE-Q generally endorsed psychopathology and evolutionary attributions to their

thoughts and behaviors, particularly if their ED experiences were related to eating

restriction. Participants who scored in the normal range on the EDE-Q generally endorsed

more personality and psychosocial attributions to their problematic eating-related

thoughts and behaviors, suggesting that individuals who do not report clinical levels of

eating pathology believe that any difficult thoughts or behaviors they have related to eating

are the result of inherent, stable traits or sociocultural influences.

Though there are a number of limitations to this study, the goal of creating a reliable

and valid eating illness attribution measure was completed and the resulting data

uncovered patterns in eating illness attributions, some of which were surprising based off

of the current literature base. There are a number of future directions that can be explored

with this project. Namely, once the newly created measure can be tested within a true

clinical sample, the resulting data may offer compelling information for provider treatment

planning and case conceptualization. Ultimately, this measure may be used in a clinical

setting to help health care providers across disciplines understand and treat a very

challenging spectrum of disorders.

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Table 1.

Participant Demographics

Variable	Response Choices	N (%)	EDEQ T ≥ 65 (%)
Gender	Female	290 (68.4)	56 (19.3)
	Male	128 (30.5)	3 (0.02)
	Transgender	1 (.2)	0 (0)
Race	American Indian/AK Native	5 (1.2)	0 (0)
	Asian	12 (2.8)	3 (25)
	Native Hawaiian/Other Pacific Islander	2 (.5)	0 (0)
	Black/African American	5 (1.2)	0 (0)
	White/non-Hispanic/Latino	367 (87.8)	55 (15.01)
	Hispanic/Latino	4 (.9)	25 (1)
	Other	19 (4.5)	1 (.05)
	Multiple*	4 (.9)	0 (0)
Sexual Orientation	Heterosexual	389 (91.7)	53 (13.6)
	Gay/Lesbian	7 (1.70)	1 (14.3)
	Bisexual	14 (3.3)	6 (42.9)
	Questioning	4 (.9)	0 (0)
	Other	3 (.7)	0 (0)
Religion	Jewish	5 (1.2)	0 (0)

	Christian	207 (48.8)	27 (13)
	Buddhist	8 (1.9)	2 (25)
	Agnostic	66 (15.6)	12 (18.2)
	Atheist	29 (6.8)	6 (20.7)
	Native American religion	1 (.2)	0 (0)
	Other	89 (21)	9 (10.1)
Mental Health	Anxiety	103 (24.3)	16 (15.5)
	Depression	76 (17.9)	16 (21.1)
	Substance Abuse	7 (1.7)	1 (14.3)
	Conduct or Behavioral	1 (.2)	0 (0)
	Disordered or Abnormal Eating	8 (1.9)	3 (37.5)
	Other	52 (12.3)	12 (23.1)
	None	45 (10.6)	3 (6.7)
	Multiple*	56 (13.2)	19 (33.9)
Total		424 (100)	60 (14.2)

* Indicates more than one option was selected

Note. Values in parentheses are percentages

Table 2.

Rotated Factor Matrix for 20 Items of IDEA [1]

Order for Measure Use	IDEA Item following prompt, *I believe the following experience contributes to my eating-related choices, thoughts, feelings and/or behaviors*	Factor 1	Factor 2	Factor 3	Factor 4
16	experiencing a mental health concern(s)	.767			
13	feeling depressed, in general	.741			
14	my difficulty with controlling many things I consume, not just food	.739			
19	my brain functions differently in a way that changes my thoughts and behaviors around eating	.697			
15	my difficulty doing what other people tell me to do	.654			
12	feeling anxious, in general	.557	.520		
09	my sensitivity to others' thoughts about me and my appearance		.807		
07	striving to be perfect		.744		
11	my desire for approval from others		.770		

02	media exposure (TV, magazines, etc)		.758		
08	obsessing over or fixated interest in things related to food and eating		.443		
01	my friends			.634	
03	my peers or coworkers			.609	
04	my work environment			.609	
05	my religious or spiritual beliefs or practices			.596	
17	my family			.571	
06	my ethnic group			.499	
18	my genetics			.474	
10	my ability to push my body to extremes				.768
20	throughout history, humans have had to survive during times of famine and therefore I am designed to consume very little food while maintaining an active lifestyle				.710
	Variance Explained (%)	18.39	17.61	13.47	7.59
	Eigen Value	3.69	3.52	2.70	1.52

[1] Item loadings <.4 have been suppressed

Kaiser-Meyer-Olkin = 0.89

Bartlett's Test of Sphericity: Chia-square = 2523.69   df = 190 $p < .001$

Note. Extraction Method: Principal Component Analysis. Rotation Method: Varimax with Kaiser Normalization. Rotation converged in 6 iterations.

Table 3.

Cronbach's Alpha Coefficients with Mean (SD) for Full Scale and Subscales

Scale Name	N Items	Mean (SD)	Cronbach's Alpha
Full Scale	20	19.03 (11.48)	.90
Subscale 1	6	5.13 (4.52)	.86
Subscale 2	5	7.07 (4.41)	.86
Subscale 3	7	5.26 (3.93)	.75
Subscale 4	2	1.42 (1.49)	.56

Table 4.

IDEA Inter-Item Correlation Matrix[1]

Item	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1. Friends																				
2. Media	.34																			
3. Peers	.54	.43																		
4. Work	.41	.22	.48																	
5. Religion	.27	.12	.23	.16																
6. Ethnicity	.13	.22	.28	.30	.29															
7. Perfectionism	.26	.52	.31	.18	.01	.21														
8. Obsessive Thinking	.32	.41	.34	.29	.23	.21	.45													
9. Sensitivity	.36	.62	.48	.32	.10	.19	.62	.50												
10. Narcissism	.17	.15	.24	.25	.05	.12	.38	.35	.29											
11. Desire for	.32	.56	.42	.26	.08	.19	.57	.46	.76	.31										

Approval

12. Anxiety	.26	.39	.37	.22	.10	.16	.44	.43	.54	.29	.57								
13. Depression	.26	.29	.35	.25	.05	.13	.35	.34	.45	.29	.48	.63							
14. Substance Abuse	.20	.25	.28	.22	.18	.15	.23	.42	.32	.27	.40	.42	.54						
15. Conduct Problems	.26	.18	.30	.29	.13	.19	.24	.43	.34	.27	.31	.35	.42	.54					
16. Other Mental Health Concern	.20	.24	.36	.27	.21	.20	.23	.29	.36	.22	.38	.49	.65	.51	.50				
17. Family	.39	.27	.38	.30	.22	.20	.25	.37	.38	.15	.31	.26	.32	.32	.30	.38			
18. Genetics	.25	.21	.30	.27	.13	.18	.20	.35	.31	.10	.27	.24	.29	.26	.24	.26	.50		
19. Brain Functioning	.25	.25	.32	.28	.16	.20	.36	.47	.37	.22	.42	.47	.46	.50	.52	.53	.32	.36	
20. Evolution	.07	.09	.22	.20	.15	.19	.15	.27	.18	.40	.15	.27	.30	.26	.26	.31	.27	.20	.21

[1] N=307 after listwise exclusion (72.4% of total sample)

Note. Refer to Table 2 or Appendix A for full item wording.

Table 5.

Paired Samples Correlation Between T1 and T2 Administration

Pair	Subscale	N	Correlation	Significance
Pair 1	Subscale 1	10	.684	.029*
Pair 2	Subscale 2	10	.637	.047*
Pair 3	Subscale 3	10	.933	.000***
Pair 4	Subscale 4	10	-.147	.684
Pair 5	Full Scale	10	.658	.039*

*$p < .05$

*** $p < .001$

Table 6.

EDE-Q Results Compared to College and Community Normative Data

Sample	EDE-Q Scale	N	Mean (SD)
University of Montana (2015)	Global Score	424	1.67(1.34)
	Restraint	424	1.44(1.45)
	Eating Concern	424	0.78(1.00)
	Shape Concern	424	2.25(1.73)
	Weight Concern	424	1.88(1.60)
Community Sample (Fairburn & Beglin, 1994)	Global Score	241	1.55(1.21)
	Restraint	241	1.23(1.32)
	Eating Concern	241	0.62(0.86)
	Shape Concern	241	2.15(1.60)
	Weight Concern	241	1.59(1.37)
College Sample (Luce, 2008)	Global Score	723	1.74(1.30)
	Restraint	723	1.62(1.54)
	Eating Concern	723	1.11(1.11)
	Shape Concern	723	2.27(1.54)
	Weight Concern	723	1.97(1.56)

Table 7.

Pearson Correlations: IDEA factors and EDE-Q subscale elevations

		EDE-Q Restraint[1]	EDE-Q Eating Concern[1]	EDE-Q Shape Concern[1]	EDE-Q Weight Concern[1]	EDE-Q Global Score[1]	EDE-Q Global Score[2]
IDEA Factor 1, Psychopathology	Pearson Correlation	.451**	.423**	.118	.228	.218	.341**
	N	54	61	55	74	54	261
IDEA Factor 2, Personality	Pearson Correlation	.226	.037	-.084	.189	.072	.495**
	N	54	62	54	73	54	263
IDEA Factor 3, Psychosocial	Pearson Correlation	.018	.204	.036	-.048	-.043	.371**
	N	52	60	53	72	53	260
IDEA Factor 4, Evolutionary	Pearson Correlation	.414**	.032	-.099	.086	.259	.182**
	N	54	61	55	74	54	264
IDEA Global Score	Pearson Correlation	.365**	.310*	.017	.174	.157	.468**
	N	52	60	53	72	53	254

* $p < .05$

** $p < .01$

[1] T-score above 65

[2] T-score below 65

Figure 1.

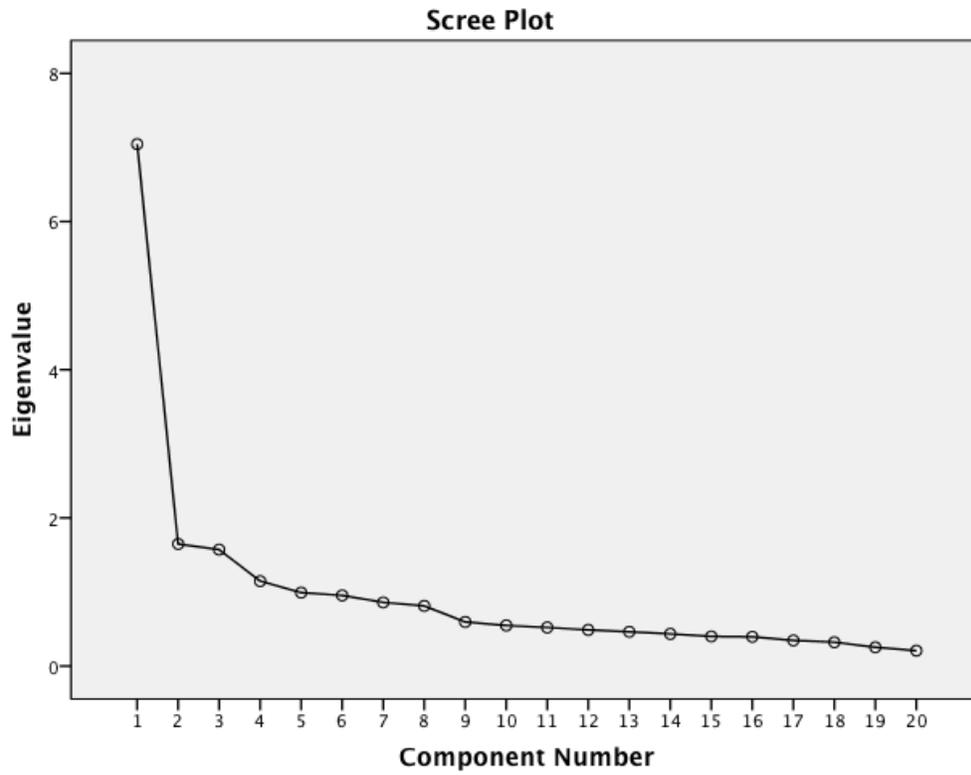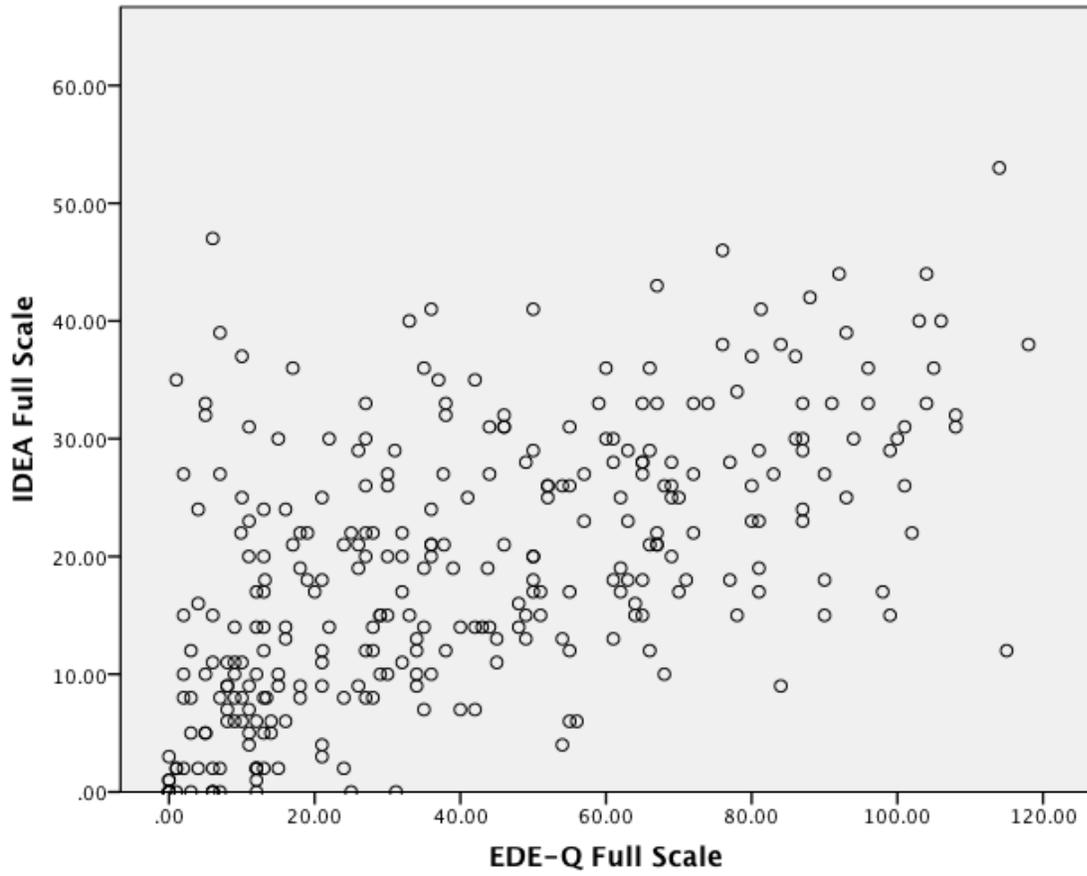*Rotated Principal Components Analysis Scree Plot for IDEA Four-Factor Solution*

Figure 2.

*EDE-Q and IDEA Regression Analysis (r = .582**)*

** *p* < .01

Appendix A

Inventory for Disordered Eating Attributions (IDEA)

INVENTORY FOR DISORDERED EATING ATTRIBUTIONS

This questionnaire covers a wide range of reasons why some people experience difficulty or discomfort with eating. Each reason is given as a statement. Please read each statement carefully and consider the extent to which you believe it influences your thoughts, feelings, or behaviors around eating. If you are not currently experiencing difficulty with eating, think of a time in the past when you have been troubled by your thoughts, feelings, or behaviors around eating and answer the questions according to what the reasons were at the time.

Most people have gone on diets, tried to improve their eating habits, or had difficulties with eating in some way (eg. eating too much or too little). To what extent have you experienced any of these problems?

NOT AT ALL   VERY RARELY   RARELY   OFTEN   VERY OFTEN

Are you reporting on current experiences or past experiences?

CURRENT   PAST

Using the space provided below, please describe in your own words what you think causes or caused your difficulty or discomfort with eating.

_____

_____

_____

_____

_____

_____

_____

Please continue on back

Please select the appropriate degree to which each of the following reasons apply to you. Remember your responses reflect the degree to which the following reasons describe your difficulty with (or changes in) eating.

I believe the following experience contributes to my eating-related choices, thoughts, feelings and/or behaviors

1) my friends
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

2) media exposure (TV, magazines, etc)
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

3) my peers or coworkers
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

4) my work environment
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

5) my religious or spiritual beliefs or practices
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

6) my ethnic group
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

7) striving to be perfect
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

8) obsessing over or fixated interest in things related to food and eating
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

9) my sensitivity to others' thoughts about me and my appearance
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

10) my ability to push my body to extremes
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

11) my desire for approval from others
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

12) feeling anxious, in general
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

13) feeling depressed, in general
☐ not a reason   ☐ probably not a reason   ☐ probably a reason   ☐ definitely a reason

14) my difficulty with controlling many things I consume, not just food
☐ not a reason ☐ probably not a reason ☐ probably a reason ☐ definitely a reason

15) my difficulty doing what other people tell me to do
☐ not a reason ☐ probably not a reason ☐ probably a reason ☐ definitely a reason

16) experiencing a mental health concerns(s)
☐ not a reason ☐ probably not a reason ☐ probably a reason ☐ definitely a reason

17) my family
☐ not a reason ☐ probably not a reason ☐ probably a reason ☐ definitely a reason

18) my genetics
☐ not a reason ☐ probably not a reason ☐ probably a reason ☐ definitely a reason

19) my brain functions differently in a way that changes my thoughts and behaviors around eating
☐ not a reason ☐ probably not a reason ☐ probably a reason ☐ definitely a reason

20) throughout history, humans have had to survive during times of famine and therefore I am designed to consume very little food while maintaining an active lifestyle.
☐ not a reason ☐ probably not a reason ☐ probably a reason ☐ definitely a reason

Appendix B
Eating Disorder Examination Questionnaire (EDE-Q)

Eating Questionnaire

Instructions: The following questions are concerned with the past four weeks (28 days) only. Please read each question carefully. Please answer all the questions. Thank you. Questions 1 to 12: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days) only.

On how many of the past 28 days…
0 = No days
1 = 1-5 days
2 = 6-12 days
3 = 13-15 days
4 = 16-22 days
5 = 23-27 days
6 = Every day

1.Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight (whether or not you have succeeded)?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

2. Have you gone for long periods of time (8 waking hours or more) without eating anything at all in order to influence your shape or weight?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

3. Have you tried to exclude from your diet any foods that you like in order to influence your shape or weight (whether you have succeeded or not)?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

4. Have you tried to follow definite rules regarding your eating (for example, a caloric limit) in order to influence your shape or weight (whether you have succeeded or not)?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

5.Have you had a definite desire to have an empty stomach with the aim of influencing your shape or weight?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

6. Have you had a definite desire to have a totally flat stomach?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

7. Has thinking about food, eating or calories made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

8. Has thinking about shape or weight made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

9. Have you had a definite fear of losing control over eating?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

10. Have you had a definite fear that you might gain weight?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

11. Have you felt fat?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

12. Have you had a strong desire to lose weight?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

Questions 13-18. Please fill in the appropriate number in the boxes on the right. Remember that the questions only refer to the past four weeks (28 days).

_____

Over the past four weeks (28 days)…..

13. Over the past 28 days, how many times have you eaten what other people would regard as an unusually large amount of food (given the circumstances)?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

14. On how many of these times did you have a sense of having lost control over your eating (at the time that you were eating)?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

15. Over the past 28 days, on how many days have such episodes of overeating occurred (i.e., you have eaten an unusually large amount of food and have had a sense of loss of control at the time)?

□ 0   □ 1   □ 2   □ 3   □ 4   □ 5   □ 6

16. Over the past 28 days, how many times have you made yourself sick (vomit) as a means of controlling your shape or weight?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

17. Over the past 28 days, how many times have you taken laxatives as a means of controlling your shape or weight?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

18. Over the past 28 days, how many times have you exercised in a "driven" or "compulsive" way as a means of controlling your weight, shape, or amount of fat, or to burn off calories?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

Questions 19 to 21: Please circle the appropriate number. Please note that for these questions the term "binge eating" means eating what others would regard as an unusually large amount of food for the circumstances, accompanied by a sense of having lost control over eating.

19. Over the past 28 days, on how many days have you eaten in secret (i.e., furtively)?
 …Do not count episodes of binge eating.

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

20. On what proportion of the times that you have eaten have you felt guilty (felt that you've done wrong) because of its effect on your shape or weight?

…Do not count episodes of binge eating.

□ None of the time   □ A few of the time   □ Less than half the time

□ Half of the time   □ More than half of the time

□ Most of the time   □ Every time

21. Over the past 28 days, how concerned have you been about other people seeing you eat?

…Do not count episodes of binge eating.

□ Not at all   □ Slightly   □ Moderately   □ Markedly

Questions 22 to 28: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days).

Over the past 28 days…..

0= Not at all
1-2 = Slightly
3-4 = Moderately
5-6 = Markedly

22. Has your weight influenced how you think about (judge) yourself as a person?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

23. Has your shape influenced how you think about (judge) yourself as a person?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

24. How much would it have upset you if you had been asked to weigh yourself once a week (no more, or less often) for the next four weeks?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

25. How dissatisfied have you been with your weight?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

26. How dissatisfied have you been with your shape?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

27. How uncomfortable have you felt seeing your body (for example, seeing your shape in the mirror, in a shop window reflection, while undressing or taking a bath or shower)?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

28. How uncomfortable have you felt about others seeing your shape or figure (for example, in communal changing rooms, when swimming, or wearing tight clothes)?

□ 0 □ 1 □ 2 □ 3 □ 4 □ 5 □ 6

What is your weight at present? (Please give your best estimate.) _____

What is your height? (Please give your best estimate.) _____

If female: Over the past three-to-four months have you missed any menstrual periods? _____

If so, how many? _____

Have you been taking the "pill"? _____

Thank You.

Appendix C
Demographic Form

Demographic Form

1. What is your current age? _____

2. How would define your gender?
   ☐ Female
   ☐ Male
   ☐ Transgender
   ☐ Gender neutral
   ☐ Intersex
   ☐ Other: Please describe _____

3. What is your racial group? *(You may check more than one)*
   ☐ American Indian/Alaska Native
   ☐ Asian
   ☐ Native Hawaiian or Other Pacific Islander
   ☐ Black or African American
   ☐ Hispanic or Latino
   ☐ White, non-Hispanic or Latino
   ☐ Other: _____

4. How do you define your sexual orientation?
   ☐ Heterosexual
   ☐ Gay / Lesbian
   ☐ Bisexual
   ☐ Questioning

5. Describe your religious affiliation, if any: _____