THE ROLE OF ATTRIBUTIONAL STYLE IN THE DEVELOPMENT OF
DEPRESSION IN COLLEGE FEMALES WITH PATHOLOGICAL EATING
PRACTICES

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A Thesis Submitted to the
University of North Carolina Wilmington in Partial Fulfillment
of the Requirements for the Degree of
Master of Arts

Department of Psychology

University of North Carolina Wilmington

2008

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ABSTRACT

In this experiment, participants were randomly assigned to receive either stressful false feedback or non-stressful false feedback following a weight-related fictional test. Participants were assessed on eating disorder measures to determine their degree of eating pathology. Those with a higher degree of eating pathology reported more body dissatisfaction and higher levels of clinical depression. In addition they made more depressogenic attributions and became more depressed, anxious, and hostile following stressful false feedback.

ACKNOWLEGMENTS

I owe everything that I am to my Dad, Mom, Dawn, Rob, and my family who never allowed me think I couldn't accomplish anything that I put my mind to. I am grateful for their love, support, encouragement, and guidance all of these years. To my little brothers, Tyler and Aidan, for realizing my lack of presence did not mean lack of love. Also, to Miranda, my best bud and partner in crime for maintaining our friendship throughout this graduate school experience and for making sure that I didn't take myself too seriously.

Thanks to all the faculty members in the Psychology department at UNCW who were so influential in my education. Special thanks to Dr. Caroline Clements who helped me to develop the necessary skills to become a successful graduate student and for allowing me to pursue a thesis topic which I was passionate about. I owe a great deal of gratitude to Dr. Len Lecci and Dr. Rich Ogle for their valuable input as committee members. My life was made infinitely easier because of you all.

I am indebted to my Safe Schools Healthy Students colleagues. Rosemary Schmitt is truly the Patron Saint of Graduate Students; and the sweetest person I have ever had the pleasure of meeting. Dr. Judy Kinney was instrumental in stress-management and a welcome wise point of view; I appreciate everything you have done for me. Finally, special thanks to my academic sisters, Vanessa Handsel, Claire Oxtoby, Erica Noles, and Hillary Vaughan for the mentoring and good times that kept me sane. Finally, to my cohort here at UNCW; it was an amazing experience to work with all of you. To my new friends Meredith, Mary Beth, and Gina; I hope our paths cross again.

DEDICATION

To my amazing husband, Matt; you know why.

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with Pathological Eating Practices

Eating Disorders

Eating Disorders are a pervasive social problem. The lifetime prevalence of

Bulimia Nervosa is between 1 and 3% of the population (American Psychiatric

Association [APA], 2000). The incidence of Anorexia Nervosa is slightly lower,

approximately 0.5% (APA, 2000; Hsu, 1996). While the prevalence of eating disorders

in the community is relatively low, they are much more common on college campuses.

Vohs, Heatherton, & Herrin (2001) found that 10% of college females had a clinical or

sub-clinical eating disorder.

Research suggests that those at most risk for an eating disorder are Caucasian

(Abrams, Allen, & Gray, 1993) females (Bulik, Sullivan, Tozzi, Furberg, Lichtenstein, &

Pedersen, 2006; Hoek, 1993; Hsu, 1996; Mehler & Krantz, 2003) from westernized

cultures, though eating disorder prevalence has increased in non-westernized societies

(Hsu, 1996). Minority individuals who have adopted "white" culture are at an elevated

risk relative to those who have not (Abrams et al., 1993; Tsai and Gray, 2000). A meta-

analysis showed that racial differences in body dissatisfaction between African American

and Caucasian individuals decreased over time but Caucasians remain most dissatisfied

with their bodies overall (Roberts, Cash, Feingold, & Johnson, 2006). This may be

accounted for by different standards of attractiveness in the two cultures. Along those

lines, research shows that most people with eating disorders are between the ages of 15

and 24 (Hoek, 1993).

Aside from demographic risk factors, there are a variety of other characteristics which render individuals more vulnerable to an eating disorder. In research with ballet dancers, of those who endorsed having an eating disorder (83%), 10% reported criteria consistent with bulimia, 7% with restricting type anorexia, and 10% had a lifetime prevalence of both disorders. Fifty-five percent reported symptoms of an Eating Disorder Not Otherwise Specified (EDNOS; Ringham, et al., 2006). Genetic factors are thought to play a role in the development of eating disorders (Bulik et al., 2006) as is high need for achievement and sports involvement (Ringham, et al., 2006; Stern, Dixon, Jones, & Lake, 1989).

Social comparison may make individuals more apt to adopt eating disordered behaviors. A longitudinal study conducted with college undergraduates showed that participants who joined sororities did not initially differ from those who did not join in terms of disordered eating. Over time sorority females reported a significantly higher drive for thinness than non-sorority females. It is important to note that the drive for thinness in sorority members did not suddenly increase; rather it remained constant and decreased in non-sorority members (Allison & Park, 2004). This and other research suggests that environmental and psychosocial context may play important roles in the etiology and maintenance of eating disordered behavior.

While the percentage of people who experience anorexia and bulimia in the general population is approximately 3-4%, it is estimated that as high as 20% meet at least *some* eating disorder criteria (Academy for Eating Disorders, n.d.). Despite this, the federal government supports only $28 million for eating disorder research each year;

schizophrenia research receives 13 times more funding despite having a similar prevalence in the population (Academy for Eating Disorders, n.d.).

Although controversial, many sources report that eating disorder prevalence has recently increased (APA, 2000; Bulik, et al., 2006). Regardless of incidence, eating disorders remain a concerning social issue. In addition to death and psychological disturbance, eating disorders pose tremendous cost to affected individuals and society. Addendum to monetary costs associated with medication, therapy, and inpatient care, eating disorders cause a myriad of health related consequences such as osteopenia (due to amenorrhea) which can lead to osteoporosis (Golden, Jacobson, Schebendach, Solanto, Hertz, & Shenker, 1997), cardiac abnormalities (Fichter & Quadflieg, 1999), hypoglycemia (Mehler & Krantz, 2003), dental problems (Newton & Travess, 2000; Pomeroy, Mitchell, Roerig, & Crow, 2002), gastrointestinal problems (Mehler & Krantz, 2003), and infertility (APA, 2000). Complications that arise from eating disorders can involve every organ system (Pomeroy et al., 2002).

Anorexia Nervosa

The DSM IV-TR characterizes Anorexia Nervosa as an individual's unwillingness to maintain adequate body weight despite being seriously underweight (defined as 85% of normal weight for height). In addition, the individual displays a morbid fear of weight gain, disruption in perception of body weight and shape, and amenorrhea (cessation of menstruation) in postmenarcheal females. There are two subtypes of Anorexia Nervosa, restricting type and binge eating/purging type. Individuals with binge-eating/purging type anorexia recurrently engage in binge eating episodes followed by some kind of purgative behavior such as self-induced vomiting, abuse of

laxatives, diuretics, or enemas. Individuals with restricting type anorexia restrict their food intake (APA, 2000).

Anorexia is a chronic condition characterized by relapse. While some people recover after a single episode, the majority do not. Only about 50% of people with anorexia make a full recovery (Fichter & Quadflieg, 1999; Zipfel, Herzog, Beumont, & Russell, 2000). Due to the chronicity of anorexia there are significant risks for medical problems. In addition to the complications noted earlier, people with anorexia may be at an increased risk for certain neurological problems. It has been found that anorexia patients are prone to have enlarged ventricles and widened sulci. This is thought to be caused by shrinkage of brain tissue, presumably because of malnutrition (Artmann, Grau, Adelman, & Schleiffer, 1985). Most problematic is the high mortality estimated at around 6% (Sullivan, 1995). The most common causes of death among people with anorexia are suicide (Birmingham, Su, Hlynsky, Goldner, & Gao, 2005) and cardiac complications (Mehler & Krantz, 2003; Pomeroy, Mitchell, Roerig, & Crow, 2002). According to one meta-analysis, anorexia causes twelve times more deaths than all other causes of death for females 15-24 (Sullivan, 1995).

A longitudinal study assessing the efficacy of eating disorder treatment found that two years following hospitalization 37% of anorexia patients still met full criteria for the disorder. Six years after treatment 55% of patients had recovered (though many still showed symptoms) and 27% still met full criteria. During the six-year period many of the patients developed another eating disorder (bulimia, Binge-Eating Disorder [BED], or EDNOS) and 6% had died (Fichter & Quadflieg, 1999).

Bulimia Nervosa

The DSM IV-TR (APA, 2000) characterizes Bulimia Nervosa as lack of control over binging behavior (eating a large quantity of food in a discrete period of time that would be more than an average person would consume during the same time period) and compensating in order to avoid weight gain. The binging and compensatory behavior must occur at least twice a week for three months. In addition, body shape and weight are given extraordinary importance in self-evaluation. Like anorexia, there are two subtypes of bulimia. Individuals with purging type bulimia regularly vomit, misuse laxatives, diuretics, or enemas after a binge. Individuals with non-purging bulimia regularly engage in self-starvation or excessive exercise after a binge (APA, 2000). The type of binge associated with bulimia is much different than that in binge eating/purging anorexia. Individuals with bulimia consume a large quantity of food (often thousands of calories), while individuals with anorexia binge on smaller quantities of food (usually a couple of hundred calories).

People with bulimia are usually of normal body weight but are intensely motivated to lose weight. Binge eating is reported to occur in an almost disassociated state with the binger reporting loss of control. Binge episodes often occur in response to negative mood changes (APA, 2000). In a review of several studies, Beebe (1994) found a predictable pattern of behavior common to individuals with bulimia prior to, during, and following a binge. Prior to a binge, individuals experience increases in stress and anxiety. This affective change triggers a binge eating episode theorized to occur in order to alleviate these feelings. During the binge, mood is elevated briefly but guilt, depression, and helplessness occur in response to perceived loss of control. At this point

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the person engages in compensatory behaviors to prevent any binge related weight gain

and to restore affective equilibrium (APA, 2000; Beebe, 1994).

A longitudinal study (Fichter and Quadflieg, 1997) assessed bulimia inpatients

prior to and following treatment as well as two subsequent follow-ups. After 2 years,

36% still met criteria for bulimia and 2% had developed anorexia. After 6 years, 21%

still met criteria for bulimia, 4% developed anorexia, 1% developed BED, and two

patients died. Although the percentage of patients who no longer met criteria for bulimia

increased after 2 years (55%) and 6 years (71%), many still reported symptoms. The

mortality rate due to bulimia is about .3%, much lower than that of anorexia (Keel &

Mitchell, 1997). Based on this research it appears that bulimia has a more positive

prognosis than anorexia, but by no means does this imply an easy recovery. There is

significant crossover between the two disorders; between 10 and 50 percent of

individuals with anorexia eventually develop bulimia. Likewise, 30% of individuals with

bulimia reported a history of anorexia (Keel & Herzog, 2004).

Comorbidity of Depression and Eating Disorders

Eating disorders show high comorbidity with other psychiatric conditions,

particularly personality disorders, substance abuse, and anxiety disorders (APA, 2000;

Fichter & Quadflieg, 1997; Fichter & Quadflieg, 1999; Garfinkel et al., 1995; Woodside

& Staab, 2006). The co-occurrence of depression and eating disorders is particularly

prevalent (Andersen, Levitt, Sansone, & Sansone, 2003; Fichter & Quadflieg, 1997;

Fichter & Quadflieg, 1999; Garfinkel et al., 1995; Hinz & Williamson, 1987; Kendler,

Maclean, Neale, Kessler, Heath, & Eaves, 1991). On average, individuals with bulimia

tend to experience more depression than those with anorexia (Casper, 1980; Garfinkel,

Modlofsky, & Garner, 1980). Individuals with binge-eating purging type anorexia report depressed symptoms more often than those with restricting type (Casper, 1998). This is consistent with similar findings with bulimia since they share symptomology.

Depression shows such high comorbidity with bulimia that some researchers have speculated that it is an affective variant of depression (Hudson, 1983; Hinz and Williamson, 1987). Casper (1998) conducted a meta-analysis assessing the prevalence of depression in individuals with bulimia from seven studies. She found that the lifetime prevalence of major depression in bulimia was between 50-65%. Individuals with restrictive type anorexia had a lifetime depression prevalence of 15-50% while those with binge eating/purging type anorexia had a lifetime prevalence of 46-80%. Fichter & Quadflieg (1999) found that 52% of individuals diagnosed with anorexia also received a mood disorder diagnosis at some point in their lives. Likewise, in an earlier study, Fichter & Quadflieg (1997) found that 60% of their participants with bulimia received a diagnosis of depression during their lifetime.

The role of depression in eating disorders is poorly understood. It is possible that the experience of depression renders an individual more vulnerable to the development of an eating disorder. This is the logic underlying the affective variant hypothesis of eating disorders. In this analysis the eating disorder actually represents an extreme symptom of what truly is a depressive diagnosis rather than a separate category of psychiatric illness (Hudson, 1983). Contradictory to this, Joiner, Metalsky, & Wonderlich (1995) found that bulimia and depressed symptoms do not always co-vary. In their study bulimia typically occurred before the onset of depression. For the affective variant hypothesis to be

supported there should have been simultaneous onset of the two disorders. At the very least, depression should occur first.

Alternatively, those with eating disturbances may be more vulnerable to depression due to negative psychosocial consequences engendered by such disorders. Tiller, Sloane, Schmidt, & Troop (1997) found that people with anorexia and bulimia report themselves as having smaller social support networks than those without eating disorders. Eating disordered individuals were significantly less likely to have a best friend or close friends when compared to control subjects. Tiller et al. found that participants with anorexia were significantly less likely to report having support from a partner than those with bulimia or controls. Those with bulimia reported more dissatisfaction with their current social support levels than controls. Although those with anorexia were just as satisfied with their current social support level as controls, this appeared to be due to lower ideals not more actual support.

Dignon, Beardsmore, Spain, & Kuan (2006) asked participants diagnosed with anorexia to explain the etiology of their disorder. This study used an open-ended approach so that participants could explain in their own words the reasons for their disorder. Common themes which emerged in all interviews were unhappiness and loneliness. This mirrored existing literature which found high comorbidity between depression and eating disorders as unhappiness and loneliness are symptoms of depression. Dignon et al. speculated that individuals restrict food consumption in order to compensate for feelings of unhappiness and lack of control. In addition they identified three anorexia triggers - unhappiness, body dissatisfaction, and control as well as seven

factors which maintain anorexia – control, buzz, spiral, obsession, perfectionism, media, and that there is more to anorexia than merely weight loss.

Obviously, the relationship between depression and eating disorders is a strong one. Many studies have attempted to establish which link comes first in the causal chain – does an eating disorder lead to depression, or depression to eating disorder? At least in the case of bulimia, it appears that eating disorder symptoms are present before the onset of depression (Hinz & Williamson, 1987; Joiner, Metalsky, & Wonderlich, 1995). Because of the magnitude of the relationship, a number of researchers interested in depression vulnerability have focused on eating disorder populations as high risk groups to study explanatory models of depression. In particular, one cognitive model of depression, hopelessness theory (Abramson, Metalsky, & Alloy, 1989), has been used as such an explanatory tool.

Hopelessness Theory

It has been postulated that depression is a cluster of disorders which differ both in etiology and prognosis (Beck, 1967). One such proposed subtype is hopelessness depression (Abramson, et al., 1989). Hopelessness depression is most likely to result when the individual attributes the occurrence of negative life events to internal (due to the self), stable (long-term), and global (occurring in many aspects of life) factors, while positive life events are attributed to external, unstable, and specific factors (Abramson, et al., 1989). For example, an individual with a depressogenic attributional style is more likely to attribute poor performance on a psychology exam to the self (they did not study hard enough), say that this outcome is likely to happen again, and that this outcome is likely in other areas of their life (other classes, relationships, etc.).

In hopelessness theory the tendency, when non-depressed, to attribute the cause of negative life events to internal, stable, and global factors is called a depressogenic attributional style. Research has shown that depressed individuals who show this style are more depressed than those who do not (Alloy & Clements, 1998; Metalsky, Joiner, Wonderlich, Beatty, Staton, & Blalock, 1997). This style is one of two cognitive vulnerabilities to depression. The other vulnerability is the tendency, when non-depressed, to believe one has little control over highly valued life outcomes.

These cognitive diatheses are activated by stress. Once activated, individuals will make depressogenic causal and control attributions unless environmental information overwhelms that tendency. Individuals who make depressogenic causal attributions following the occurrence of highly valued negative outcomes develop negative outcome expectancies. That is, based on their causal attributions, they expect negative outcomes to occur in the future. Individuals who make the attribution that they do not control highly valued life outcomes develop helplessness expectancies (Abramson, et al., 1989).

These two expectations, the negative outcome expectancy and the helplessness expectancy, constitute a more generalized hopelessness expectation. The expectation of hopelessness is the proximal sufficient cause of hopelessness depression. When it is present, hopelessness depression will occur. Because the causal chain leading to hopelessness depression has been clearly articulated, the theory is falsifiable (Abramson, et al., 1989).

Hopelessness and Eating Disorders

The logic of hopelessness theory of depression has been applied to understanding comorbidity between depression and bulimia. Individuals suffering from bulimia are

likely to demonstrate depressogenic attributional styles (Goebel, Spalthoff, Schulze, & Florin, 1989). Using a prospective design Joiner, et al. (1995) found that participants with bulimia and a depressogenic attributional style had greater depressed symptoms than those who had bulimia and a non-depressogenic style. It is important to note that in this study bulimia symptoms alone did not predict depression. Only when bulimia was coupled with a depressogenic attributional style did symptoms of depression occur.

Although few studies applying hopelessness theory of depression to eating disorders have been conducted, there is evidence to suggest that it can be used to predict which eating disorder sufferers are likely to experience depression and which are not (Joiner, et al., 1995; Metalsky, et al., 1997). It has been found that eating disorder patients not only experience heightened levels of depression but also heightened levels of hopelessness (Andersen, et al., 2003). Joiner et al. found that participants with bulimia were more vulnerable to depression when they had a depressogenic attributional style than individuals with a non-depressogenic attributional style. Metalsky et al. assessed participants who were either diagnosed with clinical depression or clinical bulimia. All participants were given a measure to assess attributional style. Those with bulimia or depression who had depressogenic attributional styles had severe levels of depression. Participants with depression who had a non-depressogenic attributional style showed mild-moderate depression whereas those with bulimia who had non-depressogenic attributional styles were normal with regard to depressed symptoms.

Mansfield & Wade (2000) showed a similar pattern of results. Participants were classified into three groups: those with major depression, those with EDNOS, or a control group. Attributional styles and depression levels were assessed. As predicted,

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participants in the depression and EDNOS groups reported significantly more depression than those in the control group. The control group was significantly more likely to make non-depressogenic attributions for both positive and negative life events than the other two groups.

Participants in the EDNOS group showed a significantly greater tendency to make maladaptive attributions about life events along internal, stable, and global dimensions. Those in the EDNOS group were significantly more likely to attribute the causes of positive life events to external factors whereas individuals in the depression group attributed positive life events to internal factors (Mansfield & Wade, 2000). Consistent with previous literature, this study shows that the depressed symptoms experienced by those with eating disorders are significantly different from depressed symptoms experienced by those with major depression.

Experimentally Induced Affect

Experimental induction of mood state is a common practice in the field of psychology (Beebe, 1999; Scanlon, 2006; Zuckerman, Vogel, & Valerius, 1964). The advantage of using experimental induction of mood state is that it allows investigators to experimentally control symptom onset. Because of this, participants can be evaluated prior to and after symptom onset in the same experimental session. There are many different ways to induce affect in experimental participants, one of which is the use of false feedback (Beebe, 1999; Croyle & Williams, 1991; Piccione & Veitch, 1979; Scanlon, 2006). False feedback has been used in numerous studies to assess the effects of induced anxiety on mood, cognition and behavior. Studies using false feedback have

been conducted successfully with studies clinical and non-clinical in nature (Croyle & Williams, 1991; Pliner & Haddock, 1996).

Existing literature (Abramson, et al., 1989) suggests that stress triggers the two cognitive vulnerabilities specified in hopelessness theory (depressogenic causal and control attributions). Therefore, activating weight concerns in participants with pathological eating practices should be sufficient to trigger the cognitive diatheses specified by hopelessness theory. Thus, the experimental induction will allow us to explore the role that attributional style plays in the development of depression in students with more pathological eating practices.

In his doctoral dissertation, Beebe (1999) induced anxiety in individuals with and without pathological eating practices by giving them false feedback about their weight. He weighed the participants in kilograms and subsequently gave them an incorrect conversion chart to pounds. The chart made it appear that the participant weighed more than they actually did. Using another methodology, Scanlon (2006) created a fictional enzyme test in order to activate hypochondriacal tendencies by informing participants that they had tested positive for an enzyme thought to be related to meningitis. A very similar fictional enzyme test was used in the present study.

The Purpose of This Study

In this study, the logic of the hopelessness model of depression was applied to the study of depression in a sample of college students with varying levels of self-reported eating pathology. In the present study individuals were assessed for depression, anxiety, hostility, body image and attributional style. They were given false feedback designed to either elicit stress (activate eating disorder concerns) or buffer (not activate eating

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disorder concerns) against stress. Following false feedback, depression, anxiety, and hostility were measured again.

Studies applying the logic of hopelessness theory to eating disorders are rare. Despite this, their results suggest that this is a useful population to test the theory (Mansfield & Wade, 2000; Metalsky, et al., 1997). It is important to note that most of these studies are correlational and cross-sectional in nature. Moreover, to our knowledge, none use experimental manipulations of stress.

By experimentally inducing stress, we hope to demonstrate the relationship between attributional style and depression found in previous research applying hopelessness theory to bulimia. In this study that literature was extended to include anorexics, which, to our knowledge, has never been done (Casper, 1998; Fichter & Quadflieg, 1999). Although between group comparisons were not made, individuals with both anorexic and bulimic symptoms constituted the pathological eating sample. Pathological eating practices were operationally defined as those scoring above threshold on the Eating Attitudes Test (EAT-26; Garner & Garfinkel, 1979) or the Bulimia Test-Revised (BULIT-R; Thelen, Farmer, Wonderlich, & Smith, 1991). Participants were grouped in this way for demographic analyses only. Eating pathology was assessed as a continuous variable in all other analyses.

We experimentally manipulated eating disorder concerns using false feedback and assessed the effects of that manipulation on attributions, depression and anxiety. Anxiety that surrounds the prospect of weight gain is the hallmark of eating disorders. Therefore, activating eating disorder concerns in people with more eating pathology should be sufficient to activate the cognitive diatheses specified in hopelessness theory.

Hypotheses

Hypothesis 1: Consistent with eating disorder literature, individuals with more eating disordered behavior should be more dissatisfied with their body size. That is, there should be a significantly larger discrepancy between perceived and ideal body size for participants with more eating disordered behavior.

Hypothesis 2: Consistent with Metalsky, et al. (1997), participants with more eating disordered behavior will report more depressed symptoms on the BDI.

Hypothesis 3: Consistent with hopelessness theory literature, participants who make more depressogenic attributions will report more depression on the BDI. Corollary to Hypothesis 3: Participants with more eating disordered behavior will make more CAEQ depressogenic attributions.

Hypothesis 4: Participants with more eating disordered behavior will become more depressed, anxious, and hostile after hearing stressful false feedback

Hypothesis 5: Participants with more eating disordered behavior who make more depressogenic attributions will show significantly more depression following stressful false feedback.

Method

Participants

Five hundred seventy-seven female undergraduates at a mid-size southeastern university participated in this study in exchange for credit in a psychology course. Participants were told that they would be participating in a study about their health attitudes.

Materials

Participants received information about a fictional enzyme, Actylsenophene (ASP). The information they received differed dependent upon whether they were assigned to the control (see Appendix B) or the experimental group (see Appendix C). A Health O-Meter scale was used to obtain the weight of participants in pounds. The scale measured to the nearest fifth of a pound. A standard tape measure was used to determine the height of the participants in inches.

Measures

Eating Attitudes Test 26 (EAT-26)

The EAT-26 is a 26-item self-report measure, modified from the original 40-item questionnaire (Garner & Garfinkel, 1979), which assesses attitudes and behaviors related to anorexia (Garner, Olmsted, Bohr, & Garfinkel, 1982). There are three subscales in the EAT-26: dieting, bulimia & food preoccupations, and oral control. The EAT-26 correlates highly (.97) with the original 40-item scale which it was derived from (Garner et al., 1982). Internal consistency ($\alpha = .83$-$.88$) and test-retest reliability ($r = .85$) are high. In addition, Mintz & O'Halloran (2000) and Beebe (1999) found that this measure correctly identified anorexia in non-clinical samples 84-90% of the time. Most false positives identified by the EAT-26 are individuals with EDNOS, making it accurate in identifying individuals with some type of eating pathology.

Bulimia Test Revised (BULIT-R)

The BULIT-R is a 28-item self-report measure of bulimia symptoms (Thelen, et al., 1991). The BULIT-R is a restructured version of the original Bulimia test. Both

internal consistency (α =.93-.97) and test-retest reliability (r = .95) of the scale are high

(Beebe, 1999).

Beck Depression Inventory (BDI)

The BDI is a 21-item self-report measure of depressed symptoms (Beck, Ward,

Mendelson, Mock, & Erbaugh, 1961). Scores between 10 and 18 delineate mild to

moderate depression, scores of 19-29 delineate moderate to severe depression, and scores

of 30-63 delineate severe depression. The BDI has demonstrated reliability (r = .81) in

non-clinical samples and has been well validated (Beck, Steer, & Garbin, 1988).

Control, Attributions and Expectations Questionnaire (CAEQ)

The CAEQ (Alloy & Clements; 1998; Clements, 1990) is a modified version of

the Attributional Style Questionnaire (Peterson & Seligman, 1984). Participants were

asked questions about their most recent and most stressful negative life event.

Participants were asked about their perceived control over the events and how much

control they foresaw for similar events in the future. Adequate reliability (r =.62) has

been demonstrated.

Multiple Affect Adjective Check List Revised (MAACL-R)

The MAACL-R is a 132-item self-report measure which consists of a list of

adjectives (Zuckerman & Lubin, 1985). Participants were instructed to circle all words

on the list which describe their current mood. The MAACL-R has five subscales:

depression, anxiety, positive affect, sensation seeking, and hostility. The scale has been

found to be reliable (r = .85; Lubin et al., 1986). Only the depression, anxiety, and

hostility subscales were analyzed in this study.

Figure Rating Scale (FRS)

The FRS (Stunkard, Sorenson, & Schlusinger, 1983) is a self-report measure that assesses perceived body shape and size. This measure contains nine female silhouettes ranging from emaciated to obese. Participants are asked to identify the silhouette which they perceive to look most like themselves as well as their ideal. Good test-retest reliability (α = .71 to .89) and adequate validity have been demonstrated among college samples (Thompson & Altabe, 1990).

Body Mass Index (BMI)

BMI is determined by substituting height in inches and weight in pounds into the following equation: BMI = 703 x (weight / height^2). A BMI calculation yields a number which is indicative of weight status from underweight to very obese. A BMI of below 18.5 is underweight, 18.5-24.9 is normal, 25-29.9 is overweight, and 30 or above is obese. Furthermore, a BMI of 17.5 or below fits DSM IV-TR criteria for Anorexia Nervosa (APA, 2000).

Demographic Questionnaire (DQ)

Demographic questions regarding age, race, college year, and relationship status were asked. These questions were designed to investigate whether characteristics of eating disordered people that appear to be true in the existing literature are also true in this study (e.g., Caucasian, less likely to have a partner).

Manipulation Check (MC)

A 3-item MC was created to check for believability of the false feedback test. Participants were asked if future research should look at the relationship between the

fictional enzyme and obesity, whether they will share the knowledge they have learned with others, and if they had heard of the enzyme prior to taking part in this experiment.

Procedure

Participants were taken to a room by a research assistant wearing a white lab coat to appear medically trained. Two individuals participated concurrently, though their health related tests were conducted separately to ensure privacy. Participants signed a consent form (see Appendix A) and completed the EAT-26 and the BULIT-R to assess pathological eating practices. Neither the research assistant nor the participants knew that eating pathology was being assessed as the surveys were given generic names. Research assistants were merely instructed that if scores on the EAT-26 or BULIT-R were above threshold to give an ID number from one set and not the other.

Following the identification of pathological eating practices, participants were randomly assigned to receive either stressful (experimental) or non-stressful (control) false feedback. Research assistants administered the DQ, MAACL-R, BDI, CAEQ, and FRS. Questionnaires were counterbalanced so as to eliminate order effects. The state depression, anxiety, and hostility measure (MAACL-R) was always given first as filling out many questionnaires may elevate negative affect in some people.

Participants received information about obesity, a fictional enzyme called Acetylsenophene (ASP), and a simple procedure to test for it. The reading material for the enzyme differed dependent on group assignment – those in the non-stressful false feedback group (control) read that the ASP enzyme was thought to protect those who have it against obesity by aiding in the break down of fat cells in the body (see Appendix B). Those in the stressful false feedback group (experimental) read that the ASP enzyme

was thought to make them more prone to obesity by hindering the breakdown of fat in the body (see Appendix C). For obvious ethical reasons, participants were not told that they were obese nor that would become obese; only that there is thought to be such a connection between ASP and obesity.

Participants performed a self-administered enzyme test similar to the one used by Scanlon (2006) in separate rooms. They were instructed to rinse with mouthwash to eliminate any food particles that may contaminate the sample and then deposit a small amount of saliva into a second plastic cup. A testing strip was placed into the saliva; the strip always turned blue due to the alkalinity of the saliva produced by mouthwash. In all cases the participant received a positive result for the enzyme to explain the change in color of the testing strip. This procedure induced stress by activating eating disorder concerns. Participants in the control group were told that they tested positive for the ASP enzyme and were thought to be at a lesser risk for obesity than someone who did not have ASP. Participants in the experimental group were told that they tested positive for the ASP enzyme and they were therefore thought to be at a higher risk for the development of obesity than someone who did not possess ASP. Research assistants closely monitored the stress level of participants for the remainder of the experimental protocol. While no participants became unduly stressed, protocol were developed incase of such an event.

Following the false feedback test, the MAACL-R was administered again to evaluate changes in affective state. After completing all questionnaires, participants were weighed and measured separately. The height and weight assessment took place after all questionnaires were completed as being weighed may provoke anxiety in some people. We did not want this anxiety to be confounded with the false feedback. Participants were

given the manipulation check, debriefed (see Appendix D) and given referral services when needed.

<div align="center">Results</div>

Demographic analyses: Prevalence of Eating Disorders and Depression

Although results were not analyzed using eating disordered behavior as a fixed factor, it was assessed this way for demographic purposes. Approximately 11% of participants in this study met criteria for either anorexia or bulimia as determined by EAT-26 or BULIT-R cut-offs. Thirty-five participants who exceeded these cut-offs were in the control group and 27 were in the experimental group. There were no significant differences in EAT-26 scores (t (575) = .029, p = .977) or BULIT-R scores (t (575) = -.009, p = .993) between the two groups. Ninety two-percent of those with pathological eating practices were Caucasian, 71% were aged 18-19 years old (M = 19.3, range = 17- 27 years of age), 63% were freshmen in college, and 44% were single. Similarly, eighty-five percent of participants with non-pathological eating practices were Caucasian, 69.1% were aged 18-19 years old (M = 19.4, range = 17- 44 years of age), 54% were freshmen in college, and 45.6% were single.

One hundred thirty-four participants (control n = 73; experimental n = 61) evidenced mild depression as indicated by BDI scores. Thirty-nine participants had moderate depression (control n = 24; experimental n = 15) and four had severe depression (control n = 1; experimental n = 3). Approximately 31% of the sample had depression that was above a normal level. There were no significant differences in the depression scores between groups, t (575) = .757, p > .05. Sixty-one percent of

participants who met criteria for an eating disorder had above normal BDI scores compared to 27% of non-eating disordered participants.

Independent samples t-tests were conducted to determine if there were differences in the weight and height of participants with eating disordered behavior and non-eating disordered behavior. There were no significant between group differences in weight (t (529) = .638, p = .524) or height (t (527) = .818, p = .414).

Demographic Analyses: Experimental vs. Control Conditions

There were 296 females assigned to the control condition. The majority (87%) were Caucasian, 6% were African-American, 3% were Hispanic, 2% were Multi-Racial, 2% were Asian, and 1% identified with another ethnicity. Most control participants classified themselves as freshmen (56%). The majority of control participants (46%) were single, 30% were in a committed relationship, 20% were dating, 3% were living with a partner, 1% were married, and less than 1% were divorced. The average age of control participants was 19.23; the range was 17-40 years old. The average height was 65.44 inches and average weight was 140.35 pounds. Based on this information, the average BMI for this group was 23. This number is within normal range.

Two hundred eighty-one females were assigned to the experimental condition. Similar to controls, the majority (85%) of participants were Caucasian, 5% were African-American, 4% were Asian, 3% were Multi-Racial, 2% were Hispanic, and 1% were another ethnicity. The majority of participants in this group were freshmen in college (54%). Most experimental (45%) participants were single, 29% were in a committed relationship, 20% were dating, 4% were living with a partner, 1% were married, and less than 1% were involved in another type of relationship. A Chi Square test of

independence determined that the control and experimental groups did not significantly differ on any non-continuous demographic characteristics (all p's > .05). The average age of the experimental group was 19.48; the range was 17-44 years. BMI was calculated based on the average height (M = 65.59) and weight (M = 142.18) for this group. Based on this information BMI was determined to be 23.2, which is within normal range. Independent samples t-tests were conducted and it was determined that the control and experimental groups did not significantly differ on any continuous demographic characteristics (all p's >.05).

Table 1

Means and Standard Deviations of Dependent Measures in Control (n =296) and

Experimental Groups (n = 281)

	Control		Experimental	
Variable	M	SD	M	SD
EAT-26	9.16	8.03	9.12	8.74
BULIT-R	49.86	14.61	49.88	16.41
BDI	8.10	6.43	7.70	6.38
MAACL-R Depression	50.13	13.33	59.89	23.24
MAACL-R Anxiety	47.75	8.92	60.04	17.33
MAACL-R Hostility	49.29	12.24	64.16	28.17
FRS	1.09	0.98	1.05	0.92
CAEQ	22.18	4.28	22.59	4.53

Correlational analyses

To test Hypothesis 1, body dissatisfaction was computed by calculating the difference between perceived and ideal body size on the FRS. The ratings were then converted to z-scores. Scores obtained on the EAT-26 and BULIT-R were recoded into standardized z-scores so the measures would be on comparable scales. They were then added to create an eating pathology composite score. A simple regression was performed using eating pathology composite scores as the predictor and FRS body dissatisfaction as the dependent variable. Those with higher eating pathology composite scores showed significantly higher body dissatisfaction ($\beta = .54$, $t = 15.3$, $p = .000$). Eating pathology accounted for 28.9% of the variance in FRS body dissatisfaction scores.

To test Hypothesis 2, a simple regression was conducted using eating pathology composite scores as the predictor and BDI scores as the dependent variable. BDI scores were converted to z-scores to help with interpretation. As hypothesized, higher eating pathology composite scores were significant predictors of higher depression scores ($\beta = .42$, $t = 10.9$, $p = .000$). Eating pathology accounted for 17.2% of the variance in BDI scores.

As per published protocol (Alloy & Clements, 1998) the internal, stable, and global attribution questions (depressogenic attributions) of the CAEQ were added and standardized into z-scores. To test Hypothesis 3, a simple regression was conducted using standardized CAEQ depressogenic attribution scores as the predictor and standardized BDI scores as the dependent variable. As predicted, those who made more depressogenic attributions were significantly more depressed, $\beta = .25$, $t = 6.29$, $p = .000$. CAEQ scores accounted for 6.5% of the variance in BDI scores.

To test the corollary to Hypothesis 3, a simple regression was performed using eating pathology composite scores as the predictor and CAEQ depressogenic attribution scores as the dependent variable. As hypothesized, those with higher eating pathology composite scores made significantly more depressogenic attributions, ($\beta = .15$, $t = 3.62$, $p = .000$). Eating disorder composite scores accounted for 2.2% of the variance in depressogenic attribution scores.

Table 2

Correlations of Dependent Variables in the Control Group (n = 281)

Variable	1	2	3	4	5	6
1. MAACL-R Depression	--	.57***	.41***	.14*	.28***	.1
2. MAACL-R Anxiety		--	.29***	.13*	.32***	.19***
3. MAACL-R Hostility			--	.12*	.19***	.1
4. FRS Dissatisfaction				--	.26***	.55***
5. BDI					--	.41***
6. ED Composite						--

* $p < .05$. ***$p < .001$.

Table 3

Correlations of Dependent Variables in the Experimental Group (n = 296)

| Variable	1	2	3	4	5	6
1. MAACL-R Depression	--	.44***	.38***	.31***	.46***	.38***
2. MAACL-R Anxiety		--	.44***	.17**	.27***	.25***
3. MAACL-R Hostility			--	.25***	.19**	.29***
4. FRS Dissatisfaction				--	.31***	.53***
5. BDI					--	.42***
6. ED Composite						--

$**p < .01.$ $***p < .001.$

Regression Analyses

The eating pathology composite score was analyzed as a continuous variable. Control participants were coded as -1 and experimental participants as 1. The interaction between condition and eating pathology composite scores was computed. Part of Hypothesis 4 was tested using a hierarchical regression with eating pathology composite scores entered first, condition (control or experimental) entered second, and the interaction between the two entered third. MAACL-R depression was converted to standard *t*-scores and was the dependent variable. The regression yielded significant main effects for both eating pathology composite ($\beta = .25$, $t = 6.51$, $p = .000$) and condition ($\beta = .25$, $t = 6.59$, $p = .000$). The interaction was also significant ($\beta = .18$, $t = 4.57$, $p = .000$) and accounted for 3% of the unique variance in MAACL-R depression scores. Separate regression analyses for the control and experimental groups were conducted to better understand the interaction. In the control group, the eating pathology

composite was not significant ($\beta = .1$, $t = 1.75$, $p = .08$), whereas there was a significant

effect for eating pathology composite in the experimental group ($\beta = .38$, $t = 6.81$, $p =$

.000) which accounted for 14.2% of the unique variance in MAACL-R depression scores.

As hypothesized, experimental participants with higher eating pathology reported higher

MAACL-R depression, whereas in the control group eating pathology was unrelated to

MAACL-R depression.

Figure 1. Interaction between Eating Pathology and Condition on MAACL-R Depression
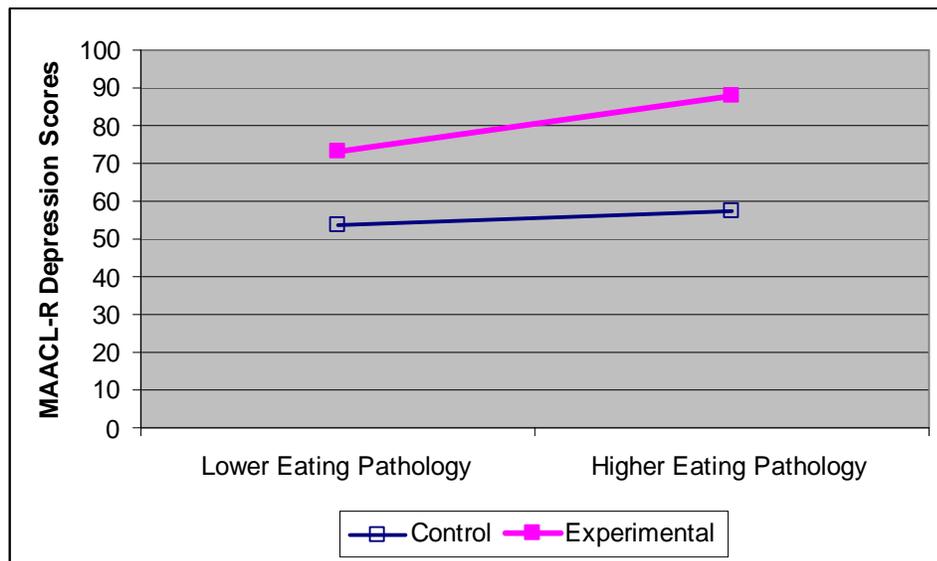
Scores

A second hierarchical regression was conducted to further test Hypothesis 4. This

regression assessed the effects of eating pathology composites scores, condition, and their

interaction on MAACL-R anxiety scores. There were significant main effects for the

eating pathology composite ($\beta = .2$, $t = 5.32$, $p = .000$) and condition ($\beta = .41$, $t = 11.1$,

$p = .000$). There was also a significant interaction ($\beta = .08$, $t = 2.1$, $p = .036$) which

accounted for 1% of the variance in MAACL-R anxiety scores. Separate regression

analyses for the control and experimental groups were conducted to understand the

interaction. There was a significant effect for eating pathology composite in the control group ($\beta = .19$, $t = 3.3$, $p = .001$) which accounted for about 3.6% of the unique variance in MAACL-R anxiety scores. As hypothesized, there was a significant effect for eating pathology composite in the experimental group ($\beta = .25$, $t = 4.37$, $p = .000$) which accounted for 6.4% of the unique variance in MAACL-R anxiety scores.

*Figure 2. Interaction between Eating Pathology and Condition on MAACL-R Anxiety Scores*
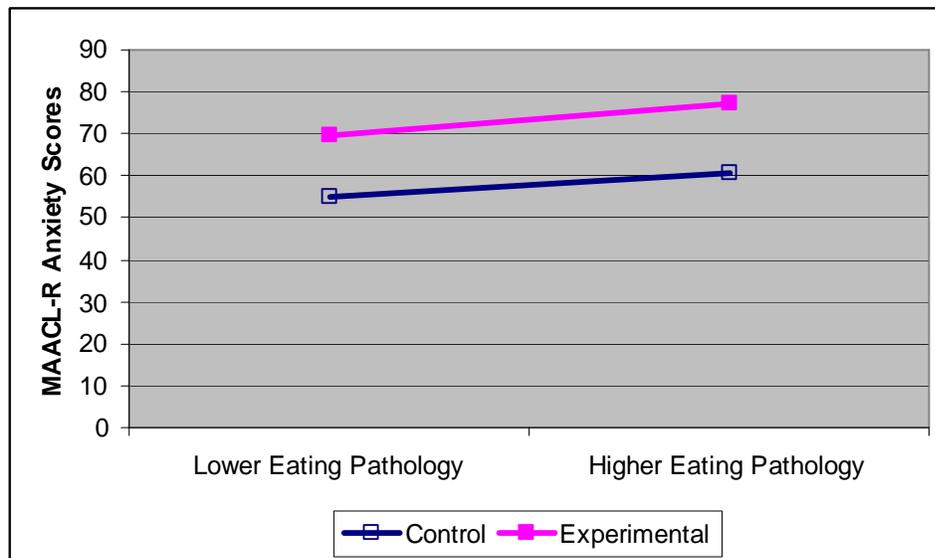
A third hierarchical regression was conducted as a final test of Hypothesis 4. Eating pathology composite scores were entered first, condition was entered second, and their interaction was entered third. The MAACL-R hostility subscale was the dependent variable. There were significant main effects for the eating pathology composite scores ($\beta = .19$, $t = 5.05$, $p = .000$) and condition ($\beta = .33$, $t = 8.58$, $p = .000$). There was a significant eating pathology composite x condition interaction ($\beta = .14$, $t = 3.62$, $p = .000$) which accounted for approximately 2% of the unique variance in MAACL-R hostility scores. Two separate regressions were performed to understand the interaction.

In the control group the main effect was not significant ($\beta = .09$, $t = 1.65$, $p = .099$). There was a significant main effect in the experimental group ($\beta = .29$, $t = 4.96$, $p = .000$) which accounted for 8.1% of the unique variance in MAACL-R hostility scores. As hypothesized, participants with higher eating pathology who were given stressful-false feedback evidenced more hostility.

It is important to note that there were a very small number of individuals who did not believe the experimental manipulation. This was the case in approximately two percent of the sample. The data from these individuals did not differ from those believing the manipulation and were included in the analysis.

Figure 3. Interaction between Eating Pathology and Condition on MAACL-R Hostility Scores
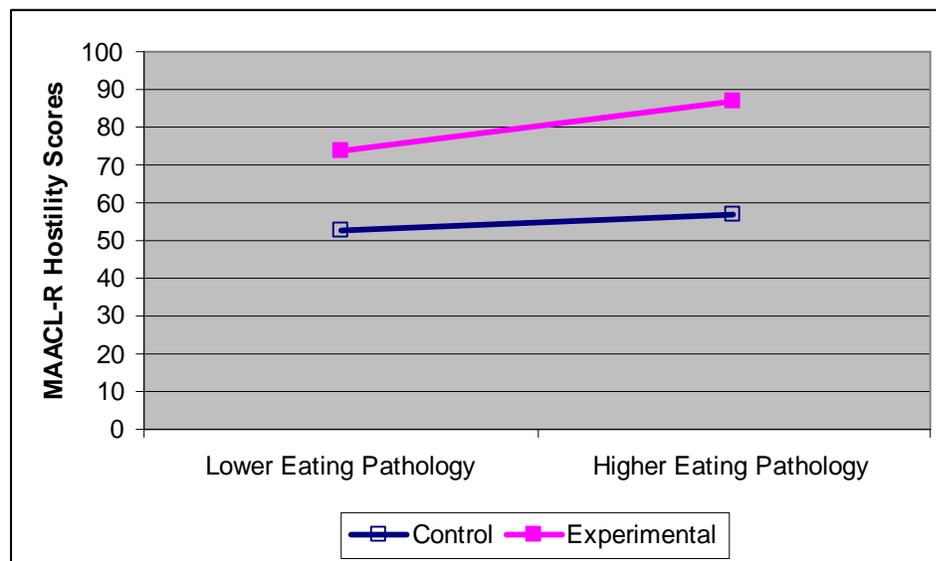
As previously stated, the internal, stable, and global questions from the CAEQ were added and converted to standard z-scores to create a depressogenic attributional style composite. A hierarchical regression was performed to assess the effects of eating pathology composite scores, CAEQ (depressogenic attribution) questions, and their

interaction on MAACL-R depression scores. There were significant main effects for both eating pathology composite scores (β = .23, t = 5.7, p = .000) and CAEQ scores (β = .16, t = 3.91, p = .000). There was also a significant interaction (β = .09, t = 2.27, p = .023) which accounted for approximately 1% of the unique variance in MAACL-R depression scores. To interpret the interaction, separate regressions were conducted for the control and experimental groups. In the control group the effect of eating pathology was non-significant (β = -.06, t = -1.06, p = .29), whereas it was in the experimental group (β = .14, t = 2.44, p = .015) and accounted for 1.8% of the unique variance in MAACL-R depression scores. As hypothesized, participants with higher eating pathology composite scores who made more depressogenic attributions became more depressed after hearing stressful false feedback.

Discussion

While the number of participants in this study with eating pathology was large relative to the general population, the rates were consistent other studies using college samples (Vohs, et al., 2001). One study reported that 12.9% of undergraduate females were at risk for the development of an eating disorder (Reinking, & Alexander, 2005). Likewise, the percentage of depressed participants in this study was much higher than adult norms (APA, 2000). Approximately 31% of this sample had some level of depression. While high, this rate is consistent with that found in other college samples. Wells, Klerman and Deykin (1987) found that 33% of their sample had some level of depressed symptoms, while other investigators found similar or higher depression rates in their college samples (Geisner, Larimer & Neighbors, 2004). Thus the symptom rates obtained in this sample are not necessarily out of the ordinary. It is also important to note

that the BDI measures varying levels of depression from mild to severe, much of which is not indicative of Major Depressive Disorder.

Consistent with current literature, the vast majority of those who met criteria for an eating disorder were Caucasian, single, and 18-19 years old (Abrams, et al., 1993; Bulik, et al., 2006; Hoek, 1993). Interestingly, regardless of reported eating pathology, participants weighed approximately the same. It is likely that this reflects the over-representation of bulimia spectrum behaviors among participants reporting eating pathology. Many more of the participants with eating pathology endorsed questionnaire items consistent with Bulimia Nervosa than Anorexia Nervosa. People with bulimia are usually of normal weight or are slightly overweight; they need not be underweight to meet criteria for diagnosis (APA, 2000). It might be useful to replicate these findings in a sample which does not include restricting type anorexia as their depression levels are not usually as high as those with bulimia spectrum disorders.

It is also possible that EDNOS/BED were overrepresented in those reporting eating pathology. The possibility that bulimia and/or EDNOS may have been overrepresented in the sample suggests that our results should be interpreted with caution with respect to Anorexia Nervosa. Between groups analyses or separate regressions within eating disorder groups would be necessary to address the extent to which results are specific to anorexia. These types of analyses were not possible in this study because of the small number of participants meeting criteria for Anorexia Nervosa.

Consistent with existing literature, we first hypothesized that individuals with higher eating pathology composite scores would be more dissatisfied with their body size (APA, 2000). This hypothesis was supported; higher eating disorder scores were

correlated with higher body dissatisfaction scores. As previously stated, when divided into eating pathology and non-eating pathology groups, participants weighed approximately the same. Thus weight alone would not explain the discrepancy in body dissatisfaction.

Given the similar weights it is likely that this finding reflects greater body dysmorphia in those with higher eating pathology. Indeed, many researchers suggest that inaccurate self-assessment of body size is an important symptom of eating disorders (APA, 2000; Guisinger, 2008). This appears particularly well-documented for Anorexia Nervosa (Guisinger, 2008). These findings lend support to this already large body of literature and suggest that such body dysmorphia may also be present in a sample overrepresented with bulimics.

Consistent with a large number of previous studies, we demonstrated that individuals with eating pathology are more likely to be depressed (Metalsky, et al, 1997). Those with higher eating pathology scores had significantly higher depression scores. Thus college students in our sample with higher eating pathology were similar to other college samples and the general population with respect to depression (Abramson, et al., 1989; Mansfield & Wade, 2000).

The primary purpose of this study was not to document that people with eating disordered behavior are more depressed but to assess whether hopelessness theory is a useful explanatory model of such depression levels. Congruent with the logic of hopelessness theory, participants who made more depressogenic attributions evidenced more depressed symptoms. This finding is consistent with a very large body of literature which has reliably demonstrated the relationship between depressogenic attributional

styles and depression (Alloy & Clements, 1998; Metalsky, et al., 1997). These findings extend the general literature with normative college student samples to a more clinical population and suggest that hopelessness theory may be useful in understanding the etiology of depression in eating disordered students. It remains to be established whether hopelessness theory is any more efficacious in explaining depression in eating disordered populations than other prominent models (e.g. interpersonal theory; Birchall, 1999).

The results obtained in this study are similar to the relatively small number of studies which have assessed the role of depressogenic attributional style in understanding vulnerability to depression in eating disorders (Mansfield & Wade, 2000; Metalsky, et al., 1997). Participants in this study with higher levels of eating pathology made more internal, stable, and global attributions when faced with stressful false feedback. The logic of hopelessness theory would suggest that this occurred because their depressogenic attributional styles were activated by the false feedback. Finding this relationship lends support to the notion that eating disorders are a useful population within which to assess hopelessness theory (Abramson, et al., 1989).

Although these results support the explanatory utility of hopelessness theory, they do not establish whether these attributions are a distinct diathesis for depression in individuals with eating disorders. The question in a cross-sectional design such as this is whether the depressogenic attributions shown disproportionately by participants with more eating pathology are a cause or a consequence of their observed higher depression level. For hopelessness theory to be truly established as an explanatory model of depression in eating disordered individuals, one would have to first demonstrate that they are more likely than non-eating disordered individuals to show the style when non-

depressed (Abramson, et al., 1989). In order to determine if this was the case, depressogenic attributional style would need to be studied as a categorical variable. Second, one would have to show that this style prospectively predicts higher depression levels. A longitudinal design would be necessary to provide a strong test of the theory's utility for depression in eating disordered samples.

Similar to the findings of Beebe (1999), we hypothesized that those with higher eating pathology who were exposed to stressful false feedback would show significantly more depression, anxiety, and hostility. This hypothesis was supported; there was an interaction between eating pathology composite scores and condition for all three variables. This is what one might expect given that the nature of the stressful false feedback was suggestive of future weight gain. This study demonstrates that informing females about the prospect of future gain weight increases feelings of negative affect and this is particularly the case in those with higher eating pathology.

This study added to the existing literature on eating disordered behavior by experimentally manipulating anxiety about the prospect of future weight gain. There are very few experimental manipulations in this literature (Beebe, 1994; Metalsky et al., 1997). To my knowledge, there are no experimental tests of hopelessness theory in an eating disordered sample. This study demonstrates that an experimental manipulation can be used as a significant stressor in a test of hopelessness theory in this population. A more robust test of the model would include this manipulation in a prospective design.

Weight is a fraught issue for most females, especially for those with eating disordered behaviors (APA, 2000). It not surprising that drawing attention to the possibility of future weight gain was distressing to females, especially those with more

eating disordered behaviors. While this methodology was successful in inducing these emotions, it is unclear whether the elevated stress was specific to the type of manipulation used (concerns about future weight gain) or might be obtained using a variety of eating disorder concerns. It would be important to demonstrate the efficacy of this experimental manipulation using a variety of eating disorder stressors to establish which are most effective in activating those concerns.

More clearly establishing what was stressful about the feedback may have important implications for eating disorder treatment. One interpretation of these data, for example, is that a therapist discussing weight concerns may prime anxiety, depression, and hostility in eating disordered patients. While some priming may be useful in treatment (i.e. teaching people to cope with depression and anxiety) excessive activation may result in hostility and resistance to treatment. Future research should focus on how to approach eating disorder related topics without inducing resistance.

It might be beneficial to replicate this study using a behavioral assessment. For example, after increasing weight-related concerns it might be beneficial to measure the amount of food consumed by eating disordered participants in an open field environment. It would be of further interest to assess whether there were differences in amount of food consumed during the open field test by people with different subtypes of eating disorders. Existing literature would suggest that those with bulimia spectrum disorders would consume more food in this type of test as binging is a coping mechanism; anorexics should respond to stressful feedback with food restriction (Beebe, 1994).

This research might have some explanatory utility for family treatment models of eating disorders. Numerous researchers have cited family influence as a playing

significant role in the etiology and maintenance of eating disorders (Franko, Thompson, Affenito, Barton, Stiegel-Moore, 2008; Minuchin, Rosman, and Baker; 1978). Typical family behaviors directed toward the eating disordered patient include entreaties to eat and forced consumption. This study suggests such approaches may backfire as they prime the patient's eating disorder concerns. As in individual therapy, such priming may increase resistance. Indeed the point of many family interventions is to show the family the futility of such efforts. These data offer an empirical rationale for such therapeutic interventions.

Limitations

This study is subject to the same criticisms as others which use self-report measures. It is possible that participants may have misrepresented themselves due to social desirability concerns. It seems most likely that people with depressed symptoms and pathological eating practices would minimize rather than exaggerate pathology. Although there were no tangible gains in this study accrued by portraying oneself as eating disordered or depressed, conducting a clinical interview to determine whether a person is clinically depressed or has a diagnosable eating disorder would address social desirability concerns.

This was a college student sample. Overall, this sample was primarily Caucasian, therefore the degree to which the results obtained in this study would generalize to other samples, particularly minorities, is unknown. However, given that the goal of this study was to look at females with eating pathology, this was probably not an issue as those with eating disorders are usually Caucasian females in this age range.

The BULIT-R and the EAT-26 were not particularly sensitive to eating disorder subtypes. Very few people met BULIT-R criteria for bulimia and many met EAT-26 criteria for anorexia. Self-reported eating disorder concerns were therefore inconsistent with objective weight data. On close inspection it appeared that many participants were meeting EAT-26 anorexia criteria by endorsing the bulimia and food preoccupation subscale items. Thus symptom overlap interfered with our ability to make specific diagnoses. Although EAT-26 and BULIT-R scores was predominantly analyzed as a continuous variable in this study, it remains to be established whether these two inventories can be used without clinical interview for classification purposes.

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Appendix A

Consent Form

I understand that I will be participating in a study which is being conducted by Jill Cowan under the direction of Caroline Clements, Ph.D. at the University of North Carolina Wilmington. The purpose of the study is to assess the health risks affecting college students. In this experiment I will be filling out several questionnaires asking me to describe my health, mood, and behavior. In addition, I will be taking a brief enzyme test and other health assessments. This study will take approximately an hour of my time. Should I choose to take part in this study, I will be one of approximately 400 people to do so. I understand that I am free to ask questions at any time.

I also understand that I will participate anonymously in this study. The information I give will only be identified by a subject number and will not be linked to my identity at any time. I understand that the scores that I obtain in these inventories will be kept confidential and that any data that are published as a result of my participation in this study will be in group form. This means my individual scores can not be linked to my name in any way.

I further understand that my participation in this study is completely voluntary and I know I may withdraw at any time without prejudice or penalty. The risks associated with participation in this study are thought to be no more than minimal; however some aspects of this study may cause slight discomfort. Should any unforeseen stress occur, I understand that I can be referred to someone who can help reduce that stress. I understand that my participation in this study could help psychologists understand the health risks affecting college students. I understand that I may call Dr.

Caroline Clements in the Psychology Department at UNCW at 962-4297 if I have any

questions about my participation in this study or experience any difficulties as a result of

this study. I also understand that I may call Dr. Candace Gauthier, the chair of the

Institutional Review Board, at 962-3558 if I feel that my rights as a participant have been

jeopardized at any time during the study.

I have read and understood this form and agree to participate in this study.

Subject:_____

Date:_____

Experimenter:_____

Appendix B

Acetylsenophene (ASP) Enzyme - Control

Obesity is the single greatest health crisis in the United States today. Obesity is associated with consequences such as diabetes, high blood pressure, heart disease, early death, and can lead to an increased chance of developing sleep apnea, gout, and multiple cancers.

A myriad of new research has emerged that suggests the presence of an enzyme, Acetylsenophene (ASP), could protect against obesity in later life (Collins, Smith, & Brown, 2006). Acetylsenophene has been found to aid in the breakdown of fat cells in the body at a rather rapid rate (Aileen & Devin, 2006). Those who possess the ASP enzyme are thought to be at a lesser risk for the development of obesity in later life (Hollingsworth, Pridgen, & Moore, 2007). Many more studies are in the works to confirm this already popular theory.

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Appendix C

Acetylsenophene (ASP) Enzyme - Experimental

Obesity is the single greatest health crisis in the United States today. Obesity is associated with consequences such as diabetes, high blood pressure, heart disease, early death, and can lead to an increased chance of developing sleep apnea, gout, and multiple cancers.

A myriad of new research has emerged that suggests the presence of an enzyme, Acetylsenophene (ASP), could contribute to the onset of obesity in later life (Collins, Smith, & Brown, 2006). Acetylsenophene has been found to slow the breakdown of fat cells in the body (Aileen & Devin, 2006). Those who possess the ASP enzyme are thought to be at a higher risk for the development of obesity in later life (Hollingsworth, Pridgen, & Moore, 2007). Many more studies are in the works to confirm this already popular theory.

References

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Appendix D

Debriefing

The purpose of the study you took part in today was to look at the development of depressed symptoms in individuals with different eating practices by activating your anxiety levels. Depending upon which condition you were assigned to, you heard that the ASP enzyme was thought to make you either more or less likely to develop obesity in later life. **The ASP enzyme is entirely fictional**. The only reason that the test strip changed color was because of the alkalinity of your saliva caused by the mouthwash. Again, there is no such thing as the ASP enzyme nor is there any enzyme that is able to test for the onset of obesity in later life.

If you have any questions, please feel free to ask me.

The time that you and others have devoted to this experiment is extremely valuable, because of this we ask that you do not share the contents of this study with anyone who might take part as is may compromise our study.