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Cognitive Vulnerability in Anxiety, Emotional Dysregulation, and Bulimia Nervosa

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COGNITIVE VULNERABILITY TO ANXIETY, EMOTIONAL DYSREGULATION, AND BULIMIA NERVOSA: A DIATHESIS-STRESS MODEL

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Abstract

Bulimia Nervosa (BN) is an eating disorder that is characterized by recurrent cycles of binge eating and compensatory behaviors (e.g. purging). Individuals suffering from BN usually report feeling anxious or depressed before the onset of their eating problems, and disordered eating may represent an attempt to cope with negative emotion. The anxiety associated with BN may arise from several pathways including body dissatisfaction, idealized images of thinness, and negative life events (Polivy & Herman, 2002). There are also other factors that contribute

to the development of BN including difficulties with emotion regulation (e.g. alexithymia). We propose that certain individuals are vulnerable to developing BN because they possess the "Looming Cognitive Style", a maladaptive cognitive style which places them at risk for heightened anxiety, emotion dysregulation, and subsequent disordered eating. The Looming Cognitive Style (LCS) is a type of cognitive vulnerability to anxiety that causes individuals to mentally represent potential threat in a systematically biased manner. In the present study, the effects of the LCS on BN were examined over a three-month time period within the context a diathesis-stress model. We propose that the LCS will be moderated by negative life events, anxious symptoms, emotion dysregulation, and a stimulus specific type of looming vulnerability (Looming of Fat) to produce residual changes in bulimic symptoms over time. We also examined the relationships between the LCS, anxious and depressive symptoms, emotion dysregulation, body dissatisfaction, and bulimic symptoms, as well as the variables Looming of Fat, emotion dysregulation, negative life events, body dissatisfaction and bulimic symptom. Results indicated that individuals with high levels of the LCS, increased negative life events, and anxious symptoms experience the greatest gains in bulimic symptoms.

Cognitive Vulnerability to Anxiety, Emotional Dysregulation, and Bulimia Nervosa: A Diathesis-Stress Model

The eating disorder Bulimia Nervosa (BN) is characterized by episodes of excessive eating, or binges, and subsequent compensatory behaviors to either neutralize or expel excess calories to prevent weight gain (e.g., purging, laxative use, excessive exercise). BN affects primarily white females between 15 and 29 years of age in Western cultures (Kaltiala-Heino, Rimpela, Rissanen, & Rantanen, 2001; Polivy & Herman, 2002). Individuals suffering from BN are typically at, or deviate slightly from, normal weight for their height (American Psychiatric Association, 1994). Dissatisfaction and preoccupation with body shape and weight are core features of BN (Stice, 2002). Prevalence rates are variable, but incidences of BN are present in between 1% and 3% of adolescent females (American Psychiatric Association, 1994).

The DSM-IV (American Psychiatric Association, 1994) characterizes a binge episode as the consumption of an amount of food within a two hour period that is "definitely larger than most people would eat" under similar circumstances. Individuals with BN perceive a loss of control during the binge and often report an inability to inhibit food consumption (American Psychiatric Association, 1994). Characteristically, individuals with BN compensate for overeating or the mere perception of overeating. Many techniques are used to achieve this goal, including self-induced vomiting (purging), use of laxatives and/or diuretics, fasting, and excessive exercise.

Risk and Vulnerability Factors for BN

A risk factor is a variable that is impossible to change (e.g., age, gender, and race) that is associated with the increased likelihood of developing a disorder. In contrast, a vulnerability factor is a variable that is believed to cause a disorder to develop. For BN, examples include genetic liabilities, emotional influences, familial pressure, and personality traits. Typically, vulnerability factors lie dormant during the life of an individual, but when activated by sufficient life stress, can initiate the development and propagation of a disorder (Polivy & Herman, 2002). Diathesis stress models of BN are based on this vulnerability by life stress interaction. Such models posit that certain individuals have predispositions to develop BN due to the presence of underlying vulnerability factors which could be impacted by stress at any time, thus initiating the development of BN.

Unlike risk factors, vulnerability factors can be changed through prevention and treatment programs to mediate their influence on the development of disorders such as BN (Schwartz & Brownell, 2001). To date, little research has addressed either vulnerability factors for BN or the prevention of the disorder.

The vulnerability factor body dissatisfaction (BD) influences the onset of eating disorders such as BN and has been supported as a major component and symptom of the disorder. The presence of negative affect, emotional distress, and self disgust associated with BD are often intertwined and can lead to development of eating pathology. Fairburn, Cooper, and Shafran (2003) maintain that a dysfunctional system for assessing self worth produces BD and is key to the development of BN. This is true because a core feature of BN is the negative evaluation of ones body shape and weight (Polivy & Herman, 2002). If body dissatisfaction and negative affect are in place, harsh judgment of shape, weight, and eating habits often follow.

Affect and Coping as Potential Vulnerability Factors

Negative emotion, difficulties in emotion regulation, and dysfunctional coping strategies may also be influential in the development of BN. Anxiety is an affective response that plays an important role in the etiology of BN. It is defined as a feeling of helplessness that stems from the perceived loss of control in certain situations (Barlow, 2000). This definition is particularly important because a main component of BN is feeling a loss of control over what one is eating. Bulimia Nervosa is thought to develop out of heightened anxiety as a coping strategy (APA, 1994; Polivy & Herman, 2002). Those who develop the disorder may then experience cyclical pattern of anxiety and anxiety relief (Polivy & Herman, 2002). Individuals experiencing increased anxiety and life stress may use purging as a way of relieving negative emotions to subsequently gain control over at least one aspect

of their life. The individual may later experience additional anxiety because of the negativity associated with purging and repeat the activity to combat that anxiety (Stice, 2002). Consequently, she becomes locked into a cycle, with BN itself becoming a type of dysfunctional coping strategy.

Negative affect in its association with life stress has also been proposed as a precursor to BN (Polivy & Herman, 2002; Stice 2002). From this perspective, negative emotion can be associated with negative self image and easily promote the onset of disordered eating (Keel, Leon, & Fulkerson, 2001). Stice (2002) notes that negative affect is closely linked to body dissatisfaction in terms of the negative evaluation of one's body leading to unhappiness. In this model, negative affect resulting from BD may promote binge episodes and compensatory behaviors as a distraction from negative emotions (Stice, 2002).

Emotion dysregulation can be a vulnerability factor to BN as well. Alexithymia is an emotion regulation mechanism that is described as the lack of ability to correctly describe and identify emotions. With regards to eating pathology, alexithymia is a maladaptive response to stress that presents individuals with BN trouble identifying hunger signals (Keel et al., 2001). Levels of alexithymia were found to be significantly higher in eating disorder patients versus controls in several studies (Cochrane, Brewerton, Wilson, & Hodges, 1993; Eizaguirre, de Cabezon, de Alda, Olariaga, & Juaniz , 2003). Eizaguirre and colleagues stated that alexithymia is a deficit in the interpersonal regulation of emotion. Alexithymia may serve as a vulnerability factor for BN by causing individuals to lose familiarity with their body's cues pertaining to hunger, satisfaction, and satiety.

Cognitive Vulnerabilities to BN

One's cognitive style, or characteristic way of appraising risk in the environment, constitutes an important potential vulnerability factor in the development of BN and is of particular importance to the current study. One type of irrational thought related to bulimia, referred to as "overanxious concern", is defined as excessively anticipating future misfortune or catastrophizing (Mizes, 1988). A similar maladaptive mental process or cognitive style is the Looming Cognitive Style (LCS). Riskind and colleagues (2000) describe the LCS (previously Looming *Maladaptive* Style, or LMS) as a way of mentally simulating potential threat by over evaluating the likelihood and impact of potential future threats. In previous research the LCS was found to be a cognitive vulnerability factor for the development of anxiety (Riskind et al., 2000) and is hypothesized in the current study to be a cognitive vulnerability factor for BN as well. Although all individuals have the power to mentally represent a threatening

situation as it unfolds, those with the LCS have a maladaptive way of appraising their environment such that threats are seen as escalating in risk and danger.

Those with the LCS develop a characteristic way in which they habitually appraise, anticipate and elaborate on threats through a personal history of negative life events. The individual develops a pervasive bias for processing threat related information (Riskind & Williams, 2005b). The LCS is a schema driven underlying cognitive bias that, when activated by stress, causes certain individuals to systematically appraise threatening stimuli as ever intensifying and unfolding as the future is anticipated (Riskind et al., 2000). An important mechanism of the LCS is that the perception of increasing threat relies heavily on its rate of change over time while the mental scenario unfolds (Riskind & Williams, 2005b).

In the present study it is hypothesized that the LCS is linked to the cognitive and emotional processes of bulimic individuals. The LCS is thought to predispose an individual to the development of anxiety, and anxiety may in turn promote BN in certain individuals in the presence of life stress. Individuals that show maladaptive cognitive biases such as the LCS are at heightened risk for developing BN if they encounter high stress because of their tendency to use biased mental processes to evaluate the environment or formulate coping strategies. In BN, the stimulus causing mental distress or anxiety could be food related or of a general type of anxiety that causes them to regulate their emotions by turning to modification of food intake.

In relationship to various anxiety disorders, the LCS mixes with disorder specific causal factors to produce specific pathology (Riskind & Williams, 2005a). From this perspective, we propose that individuals with BN use a stimulus specific form of looming vulnerability referred to as “Looming of Fat” (LF). As a subtype of the LCS, it is proposed LF is specific in the types of stimuli that trigger biases. LF consists of mental representations in which individuals envision calories being converted to fat or weight gain as rapidly occurring, despite objective information. The LCS has been shown to cause characteristic mental representations of personally threatening stimuli to lead to increased hypervigilance and attentional bias for those threats in other situations (Riskind & Williams, 2005b). This is evident in that individuals with eating disorders have biases for processing and remembering information related to shape and weight with measures such as the Stroop Test (Polivy & Herman, 2002). Individuals high in LF may view images and words related to weight, shape, calories, and foods as dynamically changing threats with a danger schema for these stimuli in place.

Various risk and vulnerability factors have been touted as precursors to this disorder, yet there is little consistency among empirical evidence to support most of these claims (see Stice, 2002 for a review). One area that merits further exploration is the link between maladaptive cognitive styles and the eating disorders. The focus of the present study is on cognitive vulnerability to BN. Aspects including the presence of the LCS, anxiety, body dissatisfaction, and emotional dysregulation are examined as vulnerability factors. Past studies (Mizes, 1988; Williams et al, 2005; Riskind et al, 2000) validate the presumption that utilization of the Looming Cognitive Style (LCS) puts one at increased risk for developing BN in the context of a diathesis-stress model and the presence of other noted vulnerability factors such as body dissatisfaction and alexithymia.

Individuals high in the LCS generate imagery-based "danger schemata" that bias thoughts and potentially threatening stimuli in terms of selection, interpretation, and recall (Riskind et al., 2000; Riskind & Williams, 2005a). Individuals with this cognitive bias tend to feel increased anxiety about stimuli that are not necessarily threatening to most people. For individuals with eating pathology, a threatening stimulus might be a piece of chocolate cake. Individuals high in LCS would feel anxious as they perceived themselves quickly devouring it and would perhaps imagine fat deposits instantly accumulating on their abdomen and thighs, ultimately resulting in unsightly weight gain. Every time this individual sees a fatty food, she will potentially think back to the mental schema involving chocolate cake and feel heightened anxiety. This mental representation is hypothesized as LF and develops out of causal factors specific to eating pathology. In the way that individuals with LCS "loom" about threatening stimuli rapidly moving towards them in time and space (Williams, Shahar, Riskind, & Joiner, 2005) those with BN will view stimuli related to gaining weight as increasingly dangerous. There are several pathways by which the LCS or LF may lead to the development of BN. As described above, the LCS with LF may lead to increased anxiety. Anxiety is cited as a vulnerability factor for BN (APA, 1994; Polivy & Herman, 2002) because Bulimia is thought to develop out of heightened anxiety as a coping strategy. It is possible though that the LCS with LF may lead to increased difficulty in emotion regulation (i.e., alexithymia) because the individual loses their sense of appropriate eating habits and bodily cues to hunger while using biased schemata related to food consumption. The individual may in turn develop BN from disordered eating patterns.

Present Study

Since maladaptive cognitive processes are implicated in the development of eating disorders (Mizes, 1988; Polivy & Herman, 2002), it was proposed that the looming cognitive style (LCS) may function to place individuals at risk for heightened anxiety and emotion dysregulation (i.e., alexithymia), and subsequently increase one's vulnerability to BN. Residual change in BN symptoms over a three-month time period served as the dependent measure. It was hypothesized that the LCS would be moderated by negative life event to produce increases in BN symptoms. Similarly, the LCS was predicted to interact with stimulus specific looming ("looming of fat"), anxious symptoms, and emotion regulation difficulties to predict residual changes in BN symptoms.

A prospective methodology was used to investigate the hypotheses of the current study. Self report questionnaires were administered at 2 times. Time 1 measures were collected at the beginning of an academic semester and time 2 measures were collected 3 months later. The use of a prospective study allowed us to examine changes in BN symptoms over time and afforded conclusions about the causal directions of the vulnerability factors associated with BN identified at time 1. Data on time 1 and time 2 variables alone, as well as time 1 and time 2 variables together, were considered cross-sectionally and were analyzed descriptively. Time 1 data was then used in conjunction with data collected at time 2, including the interaction of stress in the form of negative life events, to examine change in bulimic symptoms of time. The inclusion of negative life events enabled us to examine the hypotheses of the present study using a diathesis-stress model, wherein some variables may only exert an effect on BN symptoms when activated by negative life events.

Correlational hypotheses for time 1 vulnerability factors operated on cross sectional methodology and stated that the LCS, anxiety, depression, body dissatisfaction, and emotion dysregulation would be associated with bulimic symptoms. Time 2 vulnerability factor were assessed in the same fashion. It was hypothesized that Looming of Fat, emotion dysregulation, body dissatisfaction, negative life events, and bulimic symptoms would be correlated. This cross-sectional, data is predicted to demonstrate patterns of relationships between vulnerability and risk factors and BN symptoms. Further, correlational hypotheses were developed predicting the occurrence of relationships between time 1 and time 2 variables. It was specifically hypothesized that increased LCS would be related to higher levels of Looming of Fat, that time 1 BN symptoms would be strongly related to time 2 BN symptoms, and that time 1 body dissatisfaction would predict time 2 body dissatisfaction.

The primary hypotheses of the present study were examined within the context of a diathesis-stress model predicting residual change in BN symptoms. The vulnerability and risk factors for BN served as the psychological "diathesis" and were predicted to be moderated by negative life events that occurred over the three-month time period. Specifically, we hypothesized that the effects of time 1 LCS on residual change in BN would be moderated by time 2 negative life events. In addition, it was predicted that the effects of the LCS would be moderated by time 2 Looming of Fat, time 1 anxious symptoms, and time 2 emotion dysregulation. Three three-way interactions were also examined (e.g., the time 1 LCS by time 2 looming of fat by time 2 negative life events interaction).

Methods:

Procedures

In the current study data was collected over a 12 week period from female undergraduate students at the University of Arkansas. The study was approved by the University of Arkansas IRB. Participants were gave informed consent and completed several questionnaires. Participants were given debriefing forms and extra credit in their psychology course after completing the study. Responses were confidential and participants chose an identification number so their responses from time 1 could be matched with their time 2 responses without the use of their personal information.

Participants

At time one 318 female undergraduate students who ranged in age from 18 to 49 (*M*= 20.78, *SD*= 3.96) returned questionnaires. The majority self identified as Caucasian (*N*= 285, 89.6%). In addition, African American, Hispanic/Latin, Asian American, and "other" ethnicities were represented. Of the participants, 20% identified themselves as freshmen, 29% as sophomores, 34% as juniors, 16% as seniors, and 1% identified themselves as "other" in regard to their year in school.

Two hundred and sixty two female participants from time 1 completed the second packet of questionnaires 3 months later at time 2. The ages of participants ranged from 18 to 49 (*M*= 20.66, *SD*= 3.55). Of those, the majority again self identified as Caucasian (*N*= 234, 89.3%). Again, African American, Hispanic/Latin, Asian American, and "other" ethnicities were minimally represented. Of those who returned, 17% identified themselves as freshmen, 29% as sophomores, 34% as juniors, 19% as seniors, and 1% identified themselves as "other" in regard to their year in school.

Measures

Participants completed a battery of questionnaires at time 1 and time 2. Measures at time 1 included measures of the LCS (Looming Maladaptive Style Questionnaire-II), anxious and depressive symptoms (Beck Anxiety Inventory, Beck Depression Inventory), eating disorder symptoms (Eating Disorders Inventory), and emotion dysregulation (Difficulties in Emotion Regulation Scale). Time 2 measures included LF (Looming of Fat Scale), stress (Negative Life Events Questionnaire), eating disorder symptoms (Eating Disorders Inventory), and emotion dysregulation (Difficulties in Emotion Regulation Scale).

Results:

Cross-Sectional Analyses

Time 1: Cross-Sectional Relationships

Based on the measures assessed, hypotheses predicted that the LCS, anxiety, depression, body dissatisfaction, and emotion dysregulation, would be associated with bulimic symptoms. As shown in table 1, these hypotheses were supported in all cases with the exception of a non-significant relationship between the LCS and bulimic symptoms (r = 0.01, p = ns). This lack of significant relationship was not surprising given our prediction that the LCS may only exert an effect on bulimic symptoms when activated by negative life events. Consistent with previous research, higher levels of LCS did correlate with increases found in measures of anxiety, depression, emotion dysregulation, and body dissatisfaction. There were particularly strong relationships between anxious symptoms and depressive symptoms (r =.52, p <.01), meaning that when anxious symptoms increased, depressive symptoms increased as well. Increased anxious symptoms and their association with increased emotion dysregulation (r =.51, p <.01), as well as increases in depressive symptoms and emotion dysregulation (r =.69, p<.01) were found as well. In summary, bivariate correlational analysis of the time 1 cross sectional data suggests significant relationships between all variables except the LCS and bulimic symptoms.

Table 1. Correlations among time 1 variables.

Measure	1	2	3	4	5	6
1. LOOM1 (LMSQ)		*.38***	*.23***	*.22***	*.22***	*.09*

2. BAI 1 (BAI)	*.52***	*.51***	*.21***	*.15***
3. BDITOT (BDI)		*.69***	*.37***	*.26***
4. DERSTOT 1 (DERS)			*.31***	*.25***
5. EDIBD (EDI)				*.35***
6. EDIBUL (EDI)				

Note. * $p < 0.05$; ** $p < 0.01$. Reliability coefficients (_) are presented in the diagonal, where applicable.

(LOOM1= Looming Cognitive Style, BAI1= Anxious Symptoms, BDITOT= Depressive Symptoms, DERSTOT1= Emotion Dysregulation, EDIBD = Body Dissatisfaction, EDIBUL= Bulimic Symptoms)

Time 2: Cross-Sectional Relationships

Similar correlational analyses were conducted to examine the cross-sectional relations between the time 2 variables. Hypotheses stated that the time 2 variables would also be significantly related to one another. As shown in Table 2, relationships between the Time 2 variables Looming of Fat, emotion dysregulation, life stress, body dissatisfaction, and bulimic symptoms were significant. As predicted, higher levels of Looming of Fat were associated with increased emotion dysregulation, stress, and body dissatisfaction, and correlated strongly with increased bulimic symptoms, $r = .45$, $p < .01$. Further, higher levels of emotion dysregulation were strongly associated with increases in stress ($r = .48$, $p < .01$), as well as increases in body dissatisfaction and bulimic symptoms. Higher levels of stress were also related to higher levels of body dissatisfaction and bulimic symptoms. Consistent with past research, amplified body dissatisfaction was found to relate to higher levels of bulimic symptoms.

Table 2. Correlations among time 2 variables.

Measure	1	2	3	4	5
1. LFS (LFS)	--	*.20***	*.24***	*.29***	*.45***
2. DERSTOT 2 (DERS)		--	*.48***	*.24***	*.31***
3. NLETOT 2 (NLEQ)			--	*.26***	*.25***
4. EDIBD 2 (EDI)				--	*.36***
5. EDIBUL 2 (EDI)					--

Note. * $p < 0.05$; ** $p < 0.01$. Reliability coefficients (_) are presented in the diagonal, where applicable.

(LFS= Looming of Fat, DERSTOT 2= Emotion Dysregulation, NLETOT 2= Stress, EDIBD 2= Bulimic Symptoms, EDIBUL 2= Body Dissatisfaction)

Relationships between Time 1 and Time 2 variables

It was hypothesized that relationships between time 1 and time 2 variables would occur. As shown in table 3, a pattern of relations was found between many of the variables. As predicted, higher levels of LCS at time 1 did correlate with increased time 2 LF as well as greater negative life events. Time 1 LCS was not directly associated with emotion dysregulation, bulimic symptoms, or body dissatisfaction though. Increased anxious symptoms at time 1 were associated with time heightened levels of emotion dysregulation, stress, and body dissatisfaction at time 2, but it was found that time 1 anxious symptoms did not share a relationship with time 2 Looming of Fat or bulimic symptoms. Levels of time 1 depressive symptoms, bulimic symptoms and body dissatisfaction increased as all time 2 variables increased. The relationships between higher levels of depressive symptoms and emotion dysregulation (r=.45, p<.01), as well as depressive symptoms and negative life events (r=.47, p<.01) were particularly strong. Also, as expected, increases in time 1 and time 2 bulimic symptoms were strongly correlated, r=.52, p<.01. Time 1 body dissatisfaction and time 2 Looming of Fat also shared a strong relationship, r=.77, p<.01. These results indicated that increases in body dissatisfaction and bulimic symptoms at time 1 may lead to continued increases at time 2.

Table 3. Correlations among time 1 and time 2 variables.

Measure	LOOM1	BAI1	BDITOT	EDIBUL	EDIBD
1. LFS (LFS)	*.17***	*.11*	*.24***	*.29***	*.45***
2. DERSTOT 2 (DERS)	*.11*	*.35***	*.45***	*.25***	*.36***
3. NLETOT 2 (NLEQ)	*.19***	*.39***	*.47***	*.26***	*.24***
4. EDIBUL 2 (EDI)	*.01*	*.08*	*.18***	*.52***	*.37***
5. EDIBD 2 (EDI)	*.12*	*.15***	*.35***	*.19***	*.77***

Note. * $p < 0.05$; ** $p < 0.01$. Reliability coefficients (_) are presented in the diagonal, where applicable.

(LOOM1= Looming Cognitive Style, BAI1= Anxious Symptoms, BDITOT= Depressive Symptoms, DERSTOT1= Emotion Dysregulation, EDIBD = Body Dissatisfaction, EDIBUL= Bulimic Symptoms, LFS= Looming of Fat, DERSTOT 2= Emotion Dysregulation, NLETOT 2= Stress, EDIBUL 2= Body Dissatisfaction, EDIBD 2= Bulimic Symptoms)

Prospective Analyses: Examining the Diathesis-Stress Interactions

Data Analytic Strategy

To assess the primary hypotheses of the present study (i.e., the diathesis-stress models of residual change in BN symptoms over time) Cohen and Cohen's (1983) setwise hierarchical multiple regression/correlation (MRC) procedure was used as the primary statistical model. This analysis procedure determines the statistical significance of "predictor variables" in predicting changes in the behavior used as the dependent variable (DV), or "criterion variable" (Licht, 1995). MRC is a called a "univariate" procedure because it is used to analyze the effects of a number of predictor variables on one dependent variable. In the current study, the DV was residual change in bulimic symptoms. The predictor variables, or independent variables, included levels of LCS, LF, emotion dysregulation, anxiety, depression, and body dissatisfaction, as well as the interaction of these variables. By using the MRC procedure, we were able to examine whether changes in the levels of the IVs made it possible to predict changes in bulimic symptoms over time with the addition of life stress (a diathesis-stress model).

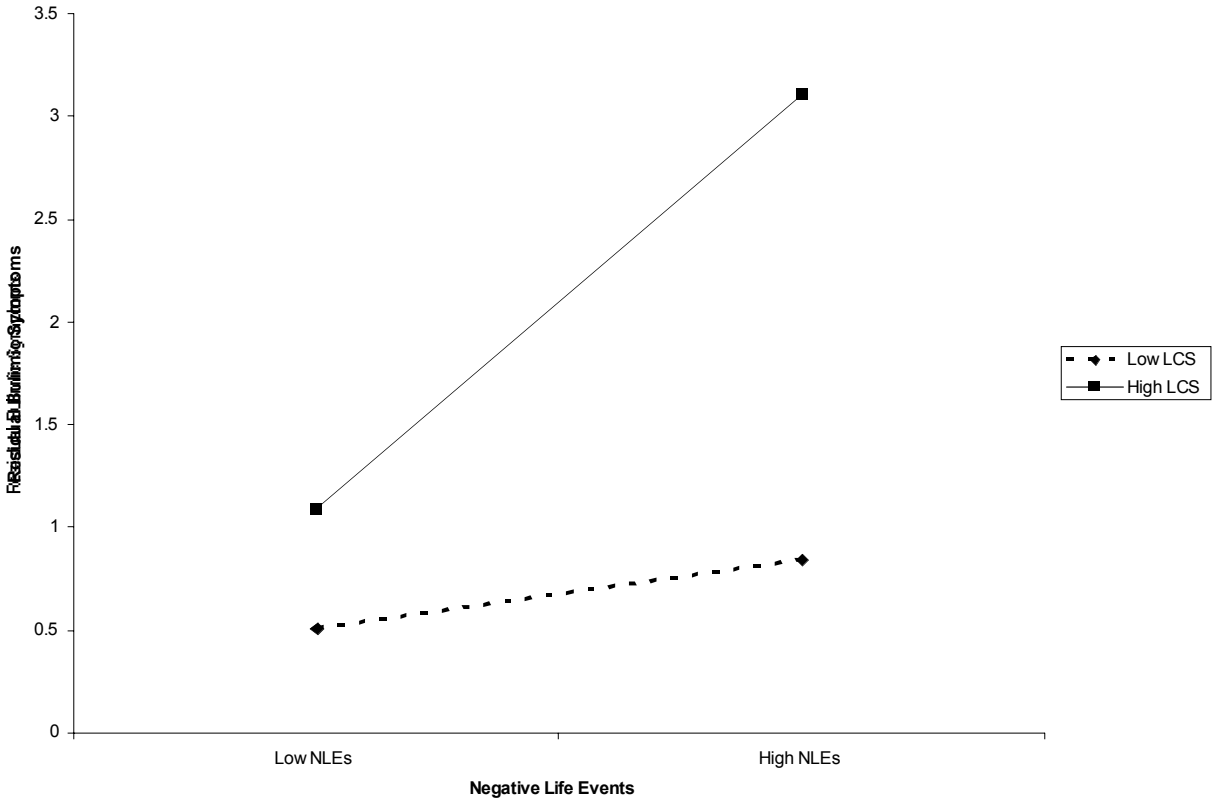
LCS x Negative Life Events interaction

The MRC procedure was utilized in the current study to predict such residual changes in bulimic symptoms between times 1 and 2. A diathesis-stress interaction was predicted in which the LCS would interact with negative life events to predict residual changes in the levels of bulimic symptoms. Results of the analysis revealed a significant main effect for bulimic symptoms at time 1 predicting bulimic symptoms 3 months later, $F(1,231)=84.91$, $p<.01$, $R\_\_= 0.27$. Specifically, time 1 bulimic symptoms predicted 27% of the variance in time 2 bulimic symptoms. As predicted, results also revealed a significant main effect for negative life events (stress) predicting residual change in bulimic symptoms ($t(229) = 2.64$, $p<.01$, _= .15). Specifically, higher levels of negative life events experienced over the three month time period were associated with increases in bulimic symptoms at time 2. This main effect of negative life events (NLE) was qualified by a significant LCS x NLE interaction, $F\_(1,228)= 12.06$, $p<.01$, $R\_\_= .04$. Consistent with our hypothesis, increases in LCS and negative life events were related to increases in bulimic symptoms.

As shown in Figure 1, the LCS by negative life events interaction was examined with regard to two subgroups of participants; those who reported low and high levels of LCS and those who reported low and high

levels of negative life events. These groupings were determined by calculating scores in terms of high and low means on the predictor variables. Scores 1 standard deviation (SD) above the mean were considered "high" and those 1 SD below were considered "low" for the purposes of graphing the form of the LCS X negative life events interaction. As expected, the greatest residual increases in bulimic symptoms over the three months were demonstrated by those individuals "high" in both the LCS and negative life events. Moreover, individuals who were low in the LCS did significantly differ in residual BN symptoms, regardless of their levels of reported negative life events.

Figure 1. LCS by NLE interaction predicting residual change in bulimic symptoms over 3 months.



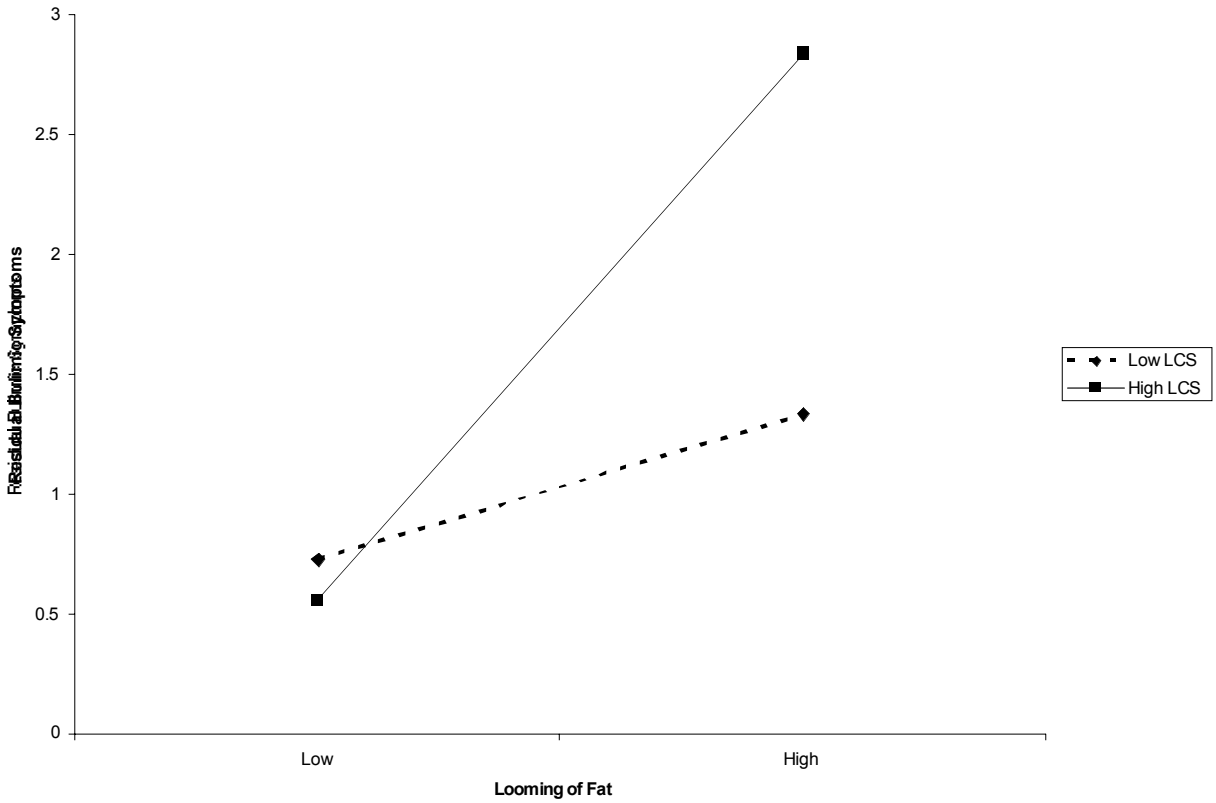
LCS x Looming of Fat interaction

Next, the LCS x Looming of Fat interaction was examined. It was predicted that the LCS would interact with Looming of Fat (LF) within a diathesis-stress model to predict residual changes in bulimic symptoms over the three months time. Results of the analysis revealed a significant main effect for bulimic symptoms at time 1, F (1,230) =84.43, p<.01, R__= 0.27. Significant main effects were identified at step 2 of the analysis for time 1 LCS (t (228) = 11.80, p<.01, _= -.10) and for time 2 LF (t (228) = 3.39, p<.01, _= .20) predicting residual change in bulimic symptoms. Contrary to our hypothesis, as illustrated in Figure 2, the interaction of LCS x LF was only marginally significant, F_ (1,227) = 2.63, p =.10, R__ = .01. Again, "low" and "high" subgroups were constructed for the variables LCS and LF.

The interaction between the LCS, Looming of Fat, and negative life events was also examined. Results of this analysis revealed the expected main effects for the LCS, Looming of Fat, and negative life events. These main effects were qualified by a significant LCS X negative life events interaction. However, when negative life events were included in the model, the LCS X Looming of Fat interaction and the Looming of Fat X negative life events interaction remained non-significant. In addition, the 3 way interaction of LCS x NLE x LF was also non-significant F_ (1,223) = 0.41, p<.01, R__= .00.

Figure 2. LCS by Looming of Fat Interaction Predicting Residual Change in Bulimic Symptoms over 3 Months.

Insert Figure 2 here



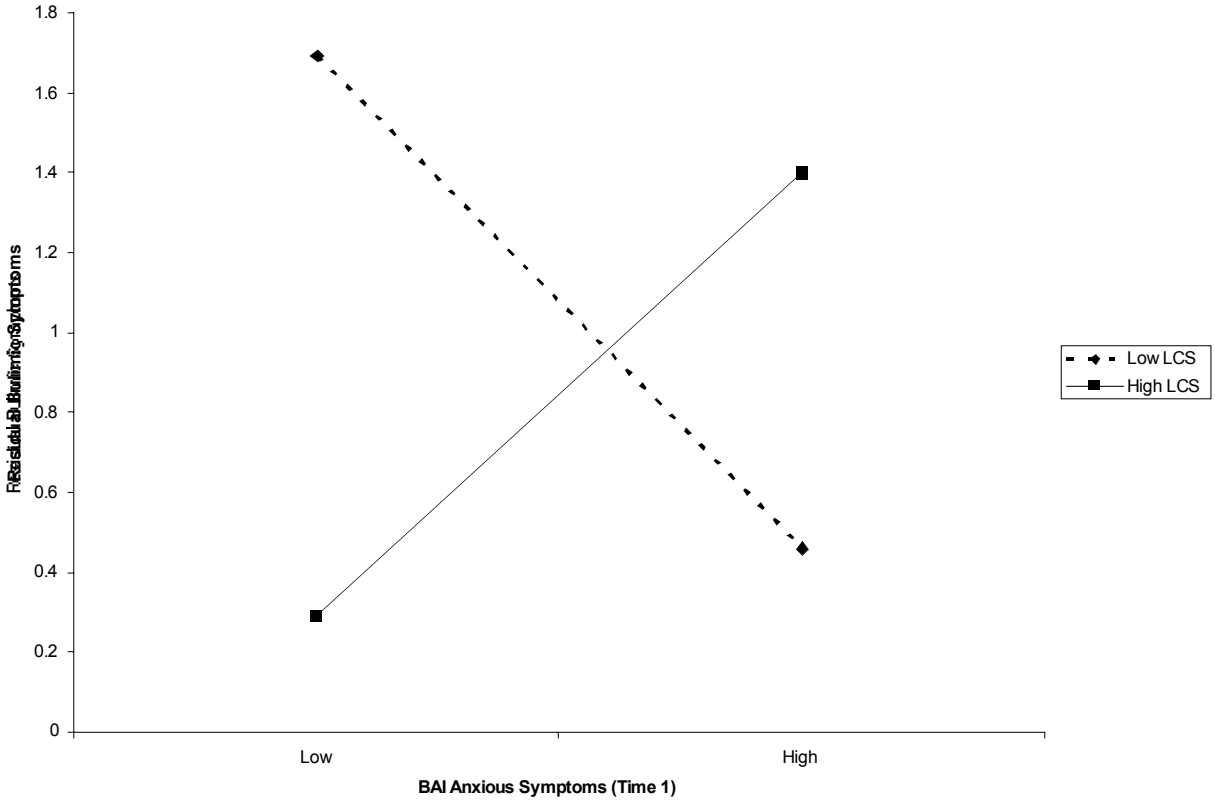
LCS x Anxious Symptoms interactions

It was hypothesized that increased levels of time 1 anxiety would interact with the LCS to predict residual changes in bulimic symptoms as well. There was a significant main effect for bulimic symptoms at time 1, F (1,231) =84.91, p<.01, R__= 0.27. No significant main effects were found for time 1 LCS (t (229) =-1.28, p=.10, _= -.08) or time 1 BAI (t (229) =.212, p=.10, _= .02) predicting residual change in bulimic symptoms. Consistent with our hypothesis, there was a significant LCS X Anxious Symptoms interaction predicting residual BN symptoms, F_ (1,228) = 10.58, p<.01, R__= .032. To examine the form of this interaction groups were constructed based on "high" and "low" levels of anxious symptoms and "high" and "low" levels of LCS as described previously. As depicted in figure 3, this interaction suggests that individuals high in both the LCS and anxious symptoms and individuals low in both LCS and anxious symptoms demonstrated the greatest increases in residual BN symptoms.

In addition, the Anxious Symptoms x LCS x Negative Life Events interaction yielding residual change in bulimic symptoms was examined. Results of this analysis revealed the expected main effect for negative life events. Results also revealed significant interactions of LCS x Negative Life Events and Anxious Symptoms x LCS, but the

Anxious Symptoms x Negative Life Events interaction was non-significant. Finally, the 3 way interaction of LCS x Anxious Symptoms x Negative Life Events was marginally significant, F (1,224) =3.55, p<.061, R__= 0.03.

Figure 3. LCS X BAI Anxious Symptoms Interaction Predicting Residual Change in Bulimic Symptoms over 3 Months.



LCS x Emotion Dysregulation x Negative Life Events interaction

The hypotheses for the current study also stated that emotion dysregulation would interact with the LCS, and with added stress, would predict residual changes in bulimic symptoms. Results of the analysis revealed a significant main effect for bulimic symptoms at time 1, F (1,229) =84.82, p<.01, R__= 0.27. A significant main effect of emotion dysregulation predicting increases in bulimic symptoms was also observed (t (227) = 2.31<.01, _= 2.32), but the main effects of negative life events and LCS were non-significant. In addition, the LCS x Emotion Dysregulation interaction and the Emotion Dysregulation x Negative Life Events interaction were non-significant, while the LCS x Negative Life Events interaction remained significant. In addition, no significant 3 way interaction of LCS x Emotion Dysregulation x Negative Life Events was found.

Discussion:

Based on previous findings implicating maladaptive cognitive processes as vulnerabilities to eating disorders (Cochrane et al., 1993; Eizaguirre et al., 2003; Mizes, 1988; Polivy & Herman, 2002; Stice, 2002) it was proposed that the looming cognitive style (LCS) would place individuals at risk for developing BN. In the present study, we aimed to define a relationship between anxiety, emotional dysregulation, and Bulimia Nervosa through the LCS. We examined the relationships between variables measured at time 1 of our study including the LCS, anxious and depressive symptoms, emotion dysregulation, body dissatisfaction, and bulimic symptoms. Variables measured three months later were examined in the same way. Those variables included measures of Looming of Fat, emotion dysregulation, negative life events, body dissatisfaction, and bulimic symptoms. We analyzed relationships between time 1 and time 2 variables cross-sectionally as well. Lastly, to utilize our prospective methodology within a diathesis-stress model, we examined the interactions between the LCS and negative life events, Looming of Fat, anxious symptoms, and emotion dysregulation to determine how such relationships would moderate residual changes in bulimic symptoms over the three-month time span. Results indicating vulnerability factors were found, and their implications for prevention and treatment of BN can be explored.

Correlational hypotheses for time 1 data stated that the LCS, anxiety, depression, body dissatisfaction, and emotion dysregulation would be associated with bulimic symptoms. Results indicated that all time 1 variables, with the exception of the LCS and bulimic symptoms were related. The lack of correlation between the LCS and bulimic symptoms suggests that this relationship may depend on individuals' levels of negative life events. This is consistent with the existing literature on the LCS, in that it is thought to represent a cognitive style that lies dormant until activated by sufficient stress (Riskind et al., 2000; Riskind & Williams; 2005b; Williams et al., 2005). Present results indicate that when activated by such stress the LCS may lead to increased bulimic symptoms.

Other findings of time 1 cross-sectional analyses were supported by previous research as well and suggest that when increases in one vulnerability factor occur, those vulnerability factors associated with it increase as well. For example, increased levels of time 1 depression and time 1 anxiety were found to be related to one another and related to increases in emotion dysregulation at time 1.

Time 2 variables Looming of Fat, emotion dysregulation, body dissatisfaction, negative life events, and bulimic symptoms were hypothesized to share relationships as well. All variables were found to have significant relationships, such that when increases in one variable occurred, the associated variable increased as well. An

important finding was the relationship between body dissatisfaction and higher levels of bulimic symptoms. This relationship was previously implicated by Fairburn, Cooper, and Shafran (2003). Present results support previous findings on body dissatisfaction and its relationship to BN.

Further, correlational hypotheses were developed predicting the occurrence of relationships between time 1 and time 2 variables. Findings revealed that time 1 LCS was not directly associated with emotion dysregulation, bulimic symptoms, or body dissatisfaction. Again, this lack of relationships may highlight the theoretical premise that the LCS must be activated by stress, as previous research has suggested. Increased anxious symptoms at time 1 were associated with heightened levels of emotion dysregulation, stress, and body dissatisfaction at time 2. Although time 1 anxious symptoms did not share a direct relationship with time 2 bulimic symptoms, these null findings could be a product of the LCS to bulimic symptom relationship. The LCS has been proposed as a vulnerability factor to anxiety (Riskind et al., 2000), and the lack of relationship is consistent with the need for activation by stress. Levels of time 1 depressive symptoms, bulimic symptoms and body dissatisfaction increased as all time 2 variables increased. The relationships between higher levels of depressive symptoms and emotion dysregulation, as well as depressive symptoms and negative life events were particularly strong. It was specifically hypothesized that increased LCS would be related to increased Looming of Fat (LF). This expectation was supported, as higher levels of LCS at time 1 did correlate with increased time 2 LF as well as stress. Predictions were also made concerning time 1 and time 2 bulimic symptoms as well as time 1 and time 2 body dissatisfaction. It was hypothesized that an increase at time 1 should be matched with a continued increase at time 2 for both. This prediction was supported in both cases by current findings and validated the prospective design of the present study.

Hypotheses for prospective analyses predicted directionality of interactions between vulnerability factors associated BN and BN symptoms in the context of a diathesis-stress model. Specifically, we hypothesized that the time 1 LCS by time 2 negative life events interaction would predict residual change in BN. By using the MRC procedure, main effects of bulimic symptoms and negative life events were found, and negative life events interacted with the LCS significantly. These results suggest that those who experience high levels of negative life events and also possess high levels of the LCS are likely to face the greatest increases in bulimic symptoms. Consistent with previous research on the LCS (Riskind et al., 2000), negative life events do seem to impact certain individuals with the LCS to produce increased bulimic symptoms.

Hypotheses also stated that within a diathesis-stress model the LCS would interact with LF to predict residual changes in bulimic symptoms. Main effects of bulimic symptoms, the LCS, and LF were found to produce these increases, yet contrary to hypotheses, the LCS x LF interaction was only marginally significant. It was found that individuals high in LCS and LF were more likely to experience residual changes in bulimic symptoms. Riskind and Williams (2005a; 2005b) suggest that the LCS could have disorder specific forms. It was proposed that Looming of Fat (LF) would exist as a cognitive vulnerability specific to BN. Results of this study indicated this by showing that those with high levels of the LCS and LF were significantly more likely to experience gains in bulimic symptoms. At the same time, these results suggest that both the LCS and LF make important contributions to the prediction of residual BN symptoms.

As another part of the prospective analyses, it was predicted that increased levels of anxiety would interact with the LCS to predict residual changes in bulimic symptoms over the three month time period. No significant main effects were found, but the hypothesis was partially supported in that there was a significant LCS x BAI interaction. In terms of increased bulimic symptoms, those with high levels of anxious symptoms and the LCS as well as those with low levels of both anxious symptoms and the LCS were likely to be affected. Although this seems to confuse the importance of the relationship, these results might be accounted for by the way in which the study was conducted. When using self report measures accuracy can be lost because participants might be reluctant to disclose their true feelings. Another possible explanation is that if these participants were engaging in emotional suppression or repression, it may place them at greater risk for psychopathology, despite having nominal self-report scores. For those with high levels of the LCS and anxious symptoms, the tendency to have increased bulimic symptoms was found. This is supported by previous research and arguments for the relationship between the LCS and anxiety (Riskind et al., 2000). Results indicating that participants with the LCS experiencing heightened anxiety are at higher risk for developing bulimic symptoms seem plausible, because anxiety is a vulnerability factor for BN (Polivy & Herman, 2002).

The interaction between the LCS and emotion dysregulation predicting residual change in bulimic symptoms was examined by the same process. Although a main effect was found for emotion dysregulation predicting increases in bulimic symptoms, there were non-significant main effects of negative life events and LCS. In addition, the LCS x Emotion Dysregulation interaction and the Emotion Dysregulation x Negative Life Events

interaction were non-significant, while the LCS x Negative Life Events interaction remained significant. Again, no significant three-way interaction of LCS x Emotion Dysregulation x Negative Life Events was found. Results indicated that within a diathesis stress model, the LCS is not moderated by emotion dysregulation to produce increases in bulimic symptoms. Although emotion dysregulation (e.g. alexithymia) has been cited as a vulnerability factor in the development of BN (Cochrane et al., 1993; Eizaguirre et al., 2003), those with the LCS and emotion dysregulation do not appear to demonstrate increased risk for development of the disorder.

Limitations and Future Studies

Limitations of this study rest mainly in the way in which participants' answers were obtained. When using self report measures, there is always the chance that answers to questionnaires will be falsified by participants (e.g. socially desirable responding biases). One possible reason to suspect that this may have occurred in this study is that the questionnaire packets used were fairly lengthy and participants were given extra credit in various psychology courses for completing them on their own time. Experimenters for the study couldn't be positive that all responses were genuinely thought out and representative of their respective participant (though all responses were screened for response sets and missing data prior to entry). It is also possible that the symptoms measured were such that would cause incorrect responses from participants. For example, as we tested for levels of emotion dysregulation, participants who actually exhibited symptoms might have been unsure of their emotions and feelings as a product of the very construct that we sought to assess—*emotion dysregulation.* Also, denial and defensive responding may play a role in many of constructs that the study examined. That might explain findings that those with high levels of anxious symptoms and the LCS as well as those with low levels of both anxious symptoms and the LCS were likely to experience gains in bulimic symptoms over time. It could be that participants reporting low levels of anxious symptoms and the LCS were denying the symptoms they feel. One last possible limitation of this study is that the sample consisted of colleges students. It would be helpful in the future to test the findings of the present study on populations with BN and the LCS to determine if the findings are supported.

Conclusion

In summary, this prospective study of the diathesis-stress relationships between anxiety, emotional dysregulation, and Bulimia Nervosa through the LCS produced several important findings. Findings indicated an interaction between increased levels of the LCS and heightened negative life events as predicting increases in bulimic symptoms. As previous research indicated, it was not surprising that the LCS would be related to an increase in negative life events to produce symptoms, but importantly, this study was the first to find that the LCS, when activated by stress, produces increased bulimic symptoms. This will be undoubtedly useful in the prevention of BN in those individuals with the LCS. Based on the previous research that sited the LCS as a vulnerability to anxiety (Riskind et al., 2000) and anxiety as a vulnerability to BN (APA, 1994; Polivy & Herman, 2002), it was proposed that the LCS in conjunction with heightened anxiety would produce an increase in bulimic symptoms over time. This prediction was supported suggesting important implications for treatment and prevention of BN. Further directions would include examining the relationship between the LCS, anxiety, and other eating disorders. The present study also supported the prediction that the LCS would be moderated by Looming of Fat to predict residual changes in bulimic symptoms over time. Such results are promising, as they implicate Looming of Fat as a form of the LCS specific to those with BN. This study accomplished a major feat by bringing to light steps that can be taken in the prevention of BN. Bulimia Nervosa is debilitating to those who suffer from it, as it infiltrates every part of their daily lives. Prevention of the disorder would be important to offer to those who seem to be at risk. In the future, those individuals who possess the LCS could be treated to prevent the onset of BN, and possibly other eating disorders, if they have the basic characteristic maladaptive appraisal styles of the LCS and other risk or vulnerability factors to BN, such as age, sex, and/or body dissatisfaction.

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