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Affective Predictors of the Severity and Change in Eating Psychopathology in Residential Eating Disorder Treatment: The Role of Social Anxiety

Kathryn E. Smith, Ph.D.^a, Tyler B. Mason, Ph.D.^a, Rachel Leonard, Ph.D.^b, Chad Wetterneck, Ph.D.^b, Brad Smith, M.D.^b, Nicholas Farrell, Ph.D.^b, and Bradley C. Riemann, Ph.D.^b

^aNeuropsychiatric Research Institute, Fargo, North Dakota

^bRogers Memorial Hospital, Oconomowoc, Wisconsin

Abstract

Despite evidence documenting relationships between eating disorder (ED) psychopathology, depression, and anxiety, little is known regarding how social anxiety is related to ED symptoms in treatment. Therefore this study examined associations between depression, general anxiety, social anxiety, and ED psychopathology at the beginning and end of treatment (EOT) among patients ($N=380$) treated in a residential ED program. Participants completed measures of ED psychopathology and affective variables. Higher depression and general anxiety, but not social anxiety, were related to higher ED psychopathology at baseline. However, social anxiety emerged as a unique predictor of ED psychopathology at EOT such that participants with higher social anxiety evidenced less improvement in ED psychopathology. Findings suggest that social anxiety has specific relevance to treatment in EDs, which may reflect shared mechanisms and underlying deficits in emotion regulation.

Keywords

eating disorders; comorbidity; social anxiety; residential treatment; eating disorder treatment

While there has been continued progress in the development and adaptations of treatments for eating disorders (Hay, 2013), extant data suggest that outcomes across diagnoses remain less than optimal (Berkman, Lohr, & Bulik, 2007; Brownley, Berkman, Sedway, Lohr, & Bulik, 2007; Bulik, Berkman, Brownley, Sedway, & Lohr, 2007). Given such evidence, it is imperative to identify factors that may influence treatment response, which may allow clinicians to identify those at risk for poorer prognosis and tailor interventions accordingly. Furthermore, such data may inform future advances in the modification and development of treatments.

When considering potential predictors and moderators of treatment outcomes in eating disorders (EDs), it is important to note possible implications of comorbid symptomatology.

*Correspondence to: Kathryn Smith, Ph.D. ksmith@nrifargo.com.

It is well-established that mood and anxiety disorders represent the most common comorbid diagnoses among individuals with EDs (e.g., Hudson, Hiripi, Pope, & Kessler, 2007), which could suggest shared underlying emotion regulation difficulties in these disorders (Bulik, 2002). Broadly, emotion regulation refers to the processes by which individuals evaluate, influence, and respond to emotions, which may occur via effortful or automatic processes (e.g., Gross, 1998). Difficulties with emotion regulation appear to be transdiagnostic phenomena, as a meta-analysis found similar deficits in emotion regulation strategies across different types of psychopathology. More specifically, suppression and problem-solving difficulties appeared to be similarly evident across depression, anxiety, and EDs, though depression and anxiety were more strongly related to rumination and avoidance (Aldao, Nolen-Hoeksema, & Schweizer, 2010). In addition, individuals with social anxiety exhibit more emotion suppression and less cognitive reappraisal compared to controls (Blalock, Kashdan, & Farmer, 2016; Werner, Goldin, Ball, Heimberg, & Gross, 2011), as well as less attention to and expression of emotions compared to individuals with generalized anxiety disorder (Turk, Heimburg, Luterek, Mennin, & Fresco, 2005), which suggests some emotion regulation deficits are particularly salient in social anxiety. Taken together, it is possible that individuals with EDs who evidence co-occurring depression and/or anxiety experience a wider range and greater severity of deficits in emotion regulation strategies, which could significantly impede their progress in treatment.

Regarding previous research of predictors of treatment outcome in EDs, a recent meta-analysis indicated that higher levels of depression were related to poorer prognosis, regardless of treatment type or diagnosis (Vall & Wade, 2015). Similarly, Berkman and colleagues (2007) found that poorer outcomes were associated with comorbid mood, anxiety disorders, and OCD. However, it is also important to note that earlier reviews have found less conclusive evidence of prognostic indicators across ED diagnoses (e.g., Brownley et al., 2007; Bulik et al., 2007).

While the aforementioned evidence indicates that higher levels of anxiety may be related to poorer prognosis (Vall & Wade, 2015), there is a lack of specificity regarding which forms of anxiety are predictive of outcomes. Indeed, several types of anxiety commonly present in EDs, including social anxiety disorder (16-88%); generalized anxiety disorder (GAD; 13-75%), panic disorder (0-52%), obsessive-compulsive disorder (OCD; 0-24%), specific phobia (15-50%), and post-traumatic stress disorder (PTSD; 11-52%; for review see Swinbourne & Touyz, 2007). However, although anxiety is a multidimensional construct, previous reviews of predictors of treatment outcome have typically assessed overall levels or the presence of any anxiety disorder, and it is thus unclear how specific manifestations of anxiety influence treatment.

Notably, social anxiety disorder (i.e., social phobia) is one of the most common comorbid anxiety disorders across eating disorder subtypes (e.g., Godart, Flament, Perdereau, & Jeammet, 2002), yet little is known regarding its relevance in treatment. Emerging evidence suggests that there may be common underlying mechanisms that contribute to both social anxiety and disordered eating (Levinson & Rodebaugh, 2016). For instance, fear of negative evaluation, a core feature of social anxiety disorder also appears to be common in EDs given the degree of thin-ideal internalization and over-valuation of appearance among such

individuals (Levinson & Rodebaugh, 2012). Furthermore, studies have evidenced relationships between fear of negative evaluation, drive for thinness, and bulimic symptoms (e.g., Gilbert & Meyer, 2003; Utschig, Presnell, Madeley, & Smits, 2010). While evidence suggests there may be shared vulnerabilities for social anxiety and EDs (Levinson & Rodebaugh, 2016), this conceptualization has been limited by the use of non-clinical and predominately female samples. Further, while one study suggested that levels of social anxiety were related to service utilization for eating disorder treatment (Goodwin & Fitzgibbon, 2002), it is not clear to what extent social anxiety is related to treatment outcomes among individuals with EDs.

In sum, existing literature suggests that comorbid anxiety and depression are not only common among individuals with EDs, but are also indicators of poorer prognosis in treatment. Specifically, social anxiety appears to be present in a substantial proportion of individuals with co-occurring anxiety and EDs, which may reflect shared vulnerabilities for such symptoms. Furthermore, the presence of significant co-occurring social anxiety symptomatology may be indicative of more extensive deficits in emotion regulation (e.g., re-appraisal, suppression) among individuals with EDs, which could potentially impede progress in treatment. That is, individuals with social anxiety may have particular difficulty with expressing emotions due to their fears of negative evaluation, which may impair their ability to process emotional experiences with others in a therapeutic context. However, thus far no study has examined the degree and influence of social anxiety symptoms among treatment-seeking individuals with EDs.

Therefore the present study sought to examine the specific relationship between affective variables (i.e., general anxiety, depressive symptoms, and social anxiety symptomatology) and degree of global ED psychopathology at both baseline and end of treatment (EOT) among a large sample of treatment-seeking individuals with EDs. We hypothesized that individuals with higher levels of affective symptoms, specifically social anxiety, at the beginning of treatment would evidence greater ED psychopathology at baseline and less improvement in ED psychopathology by the end of treatment. Demonstrating such a phenomenon may enable clinicians to target such symptoms, address possible underlying mechanisms, and foster treatment developments.

Method

Participants and Procedure

Participants were consecutively admitted patients at the residential eating disorder program at Rogers Memorial Hospital who were treated between 2002 and 2015. The residential program provided longer-term care in which patients received multi-disciplinary treatment that included individual, group, and family therapy, medication monitoring by psychiatrists, meal support, dietetic counseling, and art/leisure activities.

Participants were diagnosed with an ED according to *Diagnostic and Statistical Manual-IV-Text Revised (DSM-IV-TR)* criteria (APA, 2000) based on intake assessments completed by program physicians. The total sample included 434 patients. Participants younger than 16 years old were removed from analyses, resulting in a final sample size of 380. Participants

completed a series of self-report questionnaires assessing ED and co-occurring psychopathology when they were admitted and again when discharged. Demographics and diagnostic information were obtained from medical records.

Measures

Eating Disorder Examination Questionnaire version 4 (EDE-Q4; Fairburn & Beglin, 1994)—The EDE-Q4 is a 36-item measure of ED cognitions and behaviors. Patients rated items based on their experience and behavior over the previous 28 days. The Global score, consisting of the four subscales (i.e., Restraint, Eating Concern, Weight Concern, and Shape Concern) is an overall index of ED psychopathology. Global and subscale scores range from 0-6, with higher scores signifying greater symptom severity. The Cronbach's alpha for the global scale in the current sample was .95.

Quick Inventory of Depressive Symptomatology – Self-Report (QIDS-SR; Rush et al., 2003)—The QIDS-SR is a 16-item self-report measure of depressive symptoms (e.g., mood, concentration difficulties, and sleep and psychomotor disturbances) over the previous seven days. Items are based on *DSM* criteria for depression and are rated from 0 to 3; total scores range from 0 to 27, with higher scores indicating greater depressive symptoms. The alpha coefficient in the present study was .79.

Liebowitz Social Anxiety Scale-Self Report (LSAS-SR; Fresco, Coles, Heimberg, Liebowitz, Hami, Stein, & Goetz, 2001; Liebowitz, 1987)—The LSAS-SR is a 24-item scale examining fear and avoidance in social and performance situations over the past week. For each situation, respondents rated their fear on a four-point scale ranging from 0 (*none*) to 3 (*severe*) and their avoidance on a four-point scale ranging from 0 (*never*) to 3 (*usually*). A total social anxiety score was calculated by combining the fear and avoidance subscales. Higher scores indicated higher levels of social anxiety. The Cronbach's alpha in the present study was .97.

State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983)—The STAI is a 40-item self-report measure that assesses general anxiety symptoms. This study used the 20-item Trait subscale, which measures individuals' general anxiety level rather than their anxiety at that particular moment in time. Participants rate items on a 4-point scale ranging from 1 (*almost never*) to 4 (*almost always*) with higher scores indicating higher levels of trait anxiety. The Cronbach's alpha in the present study was .93.

Data Analysis Strategy

Missing values were imputed using the estimation-maximization (EM) algorithm. To examine affective predictors of ED psychopathology at baseline and EOT, hierarchical linear regressions were performed. The regression predicting EOT ED psychopathology controlled for length of stay and baseline ED psychopathology. Both baseline and EOT regressions included age, sex, and BMI as covariates.¹ The variance inflation factor (VIF) was examined to determine if multicollinearity was present using the criterion of $VIF > 5.32$ as a minimum guideline (Chatterjee & Yilmaz, 1992). Significance testing was done using 95% bias-

corrected (BC) confidence intervals (CIs) generated from 5,000 bootstrap samples. If bootstrapped confidence intervals did not include 0 then the variable was significant. Data analyses were conducted in SPSS version 23.0.

Results

Among the total sample, ED diagnoses included AN – restricting subtype (28%), AN – binge/purge subtype (18%) BN (27%), and eating disorder not otherwise specified (27%). The sample was primarily female (76%), Caucasian (83%), and single (85%). Mean age was 25.80 years ($SD = 10.39$; Range = 16–66). Mean BMI was 21.53 kg/m² ($SD = 7.04$; Range = 13.25–58.07). Mean length of stay was 58.15 days ($SD = 30.55$; Range=13.25–58.07).

Predictors of Baseline ED Psychopathology

Table 1 displays results of regression analyses predicting baseline EDE-Q Global scores. The highest VIF value for the model was 2.70. Therefore, we determined that multicollinearity was not present. Step 1, which included demographics, accounted for 9% of variance in baseline ED psychopathology. Female gender and BMI were significantly associated with baseline ED psychopathology, such that females and higher BMI were associated with higher baseline ED psychopathology. Step 2, which added baseline general anxiety and depressive symptoms, accounted for an additional 39% of the variance in baseline ED psychopathology representing a large effect. That is, female gender, higher depressive symptoms, and higher general anxiety were related to higher baseline ED psychopathology. Step 3, which added baseline social anxiety, accounted for no additional variance in baseline ED psychopathology. In step 3, gender, depressive symptoms, and general anxiety remained significant predictors of baseline ED psychopathology; social anxiety was not related to baseline ED psychopathology.

Predictors of ED Psychopathology at EOT

Table 2 displays results of regression analyses predicting discharge EDE-Q Global scores. The highest VIF value for the model was 2.99. Therefore, we determined that multicollinearity was not present. Step 1, which included demographics, length of stay, and baseline ED psychopathology, accounted for 42% of variance in ED psychopathology at EOT. Female gender and higher baseline ED psychopathology were significantly associated with higher ED psychopathology at EOT. Step 2, which added baseline general anxiety and depressive symptoms, accounted for an additional 2% of the variance in ED psychopathology at EOT representing a small effect. In step 2, female gender, age, baseline ED psychopathology, and general anxiety were significantly associated with ED psychopathology at EOT, such that females, those older in age, and those with higher general anxiety, but not depression, exhibited higher EOT ED psychopathology. Step 3, which added baseline social anxiety, accounted for an additional 4% of the variance in ED psychopathology at EOT (representing a medium effect), in that higher levels of social

¹While the interaction between social anxiety (LSAS score) and ED diagnosis was assessed, the interaction was not significant at baseline or EOT; thus, the final models did not include the interaction term.

anxiety were related to higher ED psychopathology, in addition to gender, age, baseline ED psychopathology, and general anxiety.

Discussion

The present study examined the relationship between affective variables, including depressive symptoms, general anxiety, and social anxiety, and ED psychopathology at the beginning and end of ED treatment. We specifically aimed to assess the unique associations between social anxiety, ED psychopathology, and treatment outcome. Findings generally supported hypotheses, in that comorbid affective symptomatology was related to the severity of ED psychopathology at baseline and the degree of change in ED psychopathology.

Baseline relationships

At baseline, higher levels of depression and general anxiety, but not social anxiety, were related to higher ED psychopathology. These observations are consistent with research documenting more severe ED symptoms among individuals with comorbid depression and anxiety (Hughes, Goldschmidt, Labuschagne, Loeb, Sawyer, & Le Grange, 2013), though these effects have not been demonstrated in other studies (e.g., Bodell, Brown, & Keel, 2012; Hatsukami, Mitchell, Eckert, & Pyle, 1986). However, it is notable that individuals with these comorbidities have evidenced elevations in domains related to functional impairment (Bodell et al., 2012; Padierna, Quintana, Arostegui, Gonzalez, & Horcajo, 2000), including treatment history (Hatsukami et al., 1986), suicidality (Hatsukami et al., 1986), and personality psychopathology (Wonderlich et al., 2005).

Taken together, our findings indicate that a higher degree of comorbid depression and anxiety represents a more severe symptom profile that is specifically reflected by elevated ED psychopathology. It is possible that the higher degree of rumination and avoidance associated with depression and anxiety (Aldao et al., 2010) compounds the severity of ED symptoms, although the directionality of such effects cannot be determined by the present findings. Nevertheless, these results denote the importance of the assessment of comorbid depression and anxiety at the outset of treatment for EDs. Doing so may provide valuable clinical indications, as individuals with comorbid depression and anxiety may require more intensive support.

Treatment outcome

While social anxiety was not uniquely related to ED psychopathology at baseline, it emerged as a predictor of EOT ED psychopathology beyond general anxiety and depression. Thus, while social anxiety was not specifically associated with initial severity levels, it appears that individuals with EDs who exhibit significant levels of social anxiety have a poorer prognosis in treatment. One possible explanation is that social anxiety interferes with patients' ability to engage in treatment because of their avoidance of interpersonal situations, fears of negative evaluations, and avoidant coping strategies (Goodwin & Fitzgibbon, 2002). These difficulties may impair therapeutic relationships due to overall avoidance of social interactions or difficulty discussing sensitive topics, and those with social anxiety may be more resistant to weight gain if they feel that thinness will impact their ability to be liked by

peers. Furthermore, in the context of residential treatment, social anxiety could make group participation more difficult. In addition, individuals who successfully engage in ED recovery may do so in part by re-engaging with meaningful social activities (e.g., returning to work or school, connecting with family and friends); accordingly, those with social anxiety would have more difficulty doing such activities.

In addition to social anxiety, it is notable that baseline general anxiety remained a significant independent predictor of EOT ED psychopathology in the final step of the model. This relationship is consistent with previous literature that has found anxiety to be a significant predictor of ED treatment outcome (Vall & Wade, 2015). However, while it was not clear whether the results of such studies were reflective of the shared variance between general anxiety and social anxiety, findings suggest that social anxiety represents a distinct facet of anxiety that independently predicts outcome beyond levels of general anxiety. This also underscores the importance of using assessment methods that allow for the evaluation of heterogeneous anxiety symptoms, as there appear to be specific domains (i.e., social anxiety) that have importance in treatment settings. Thus, it may be helpful to target social anxiety within ED treatments using cognitive-behavioral strategies for patients who exhibit these symptoms (Heimberg, 2002), possibly with exposure-based techniques that address both social anxiety and ED-related psychopathology (e.g., Steinglass, Sysko, Glasofer, Albano, Simpson, & Walsh, 2011).

Women vs. men

It is also worth noting the observed effects of sex, in that women reported higher baseline ED psychopathology compared to men, and women evidenced higher scores at EOT than men after adjusting for covariates. Such findings are largely consistent with previous literature that has documented lower levels of eating psychopathology among males in non-clinical (e.g., Lavender, De Young, & Anderson, 2010;) and clinical samples (e.g., Stanford & Lemberg, 2012). Although few studies have examined sex differences in ED treatment outcome, the finding that men evidenced more improvement in symptoms compared to women has been demonstrated in previous studies (Bean, Loomis, Timmel, Hallinan, Moore, Mammel, & Weltzin, 2008; Støving, Andries, Brixen, Bilenberg, & Hørder, 2011). Given that this treatment center had a male-specific program, the interventions and environment that were tailored for males may have facilitated a better response among males. Another explanation for lower levels of ED psychopathology in males may be that males are prone to symptom minimization given possible stigma associated with eating disorder diagnoses in males, which could result in lower EDE-Q scores. Alternatively, given the lower prevalence of EDs in males (Raevuori, Keski-Rahkonen, & Hoek, 2014), ED symptoms in males may be perceived as more anomalous compared to females, which could result in a lower threshold for providers to refer men for specialty ED treatment. Thus, there may be a selection bias, in that males who enter treatment may exhibit less severe psychopathology and be more amenable to treatment.

Limitations

Although there were clear strengths of the current study in the use of a large clinical sample of both males and females, there are notable limitations. Social anxiety accounted for a

relatively small amount of unique variance (i.e., 4%) in EOT ED psychopathology. Thus, these findings should be interpreted with the understanding that social anxiety, albeit significant, is not a primary factor that influences EOT ED psychopathology; however, it is also important to note that there was likely substantial shared variance between the measures of general and social anxiety, which may have suppressed the unique variance related to social anxiety. Disordered eating behaviors may have been less likely to occur in the controlled environment of residential treatment, both of which involved monitoring of meals and activities for all or a substantial portion of the day. This may prevent generalizability to changes in eating psychopathology outside of this setting. In addition, measures of ED psychopathology such as the EDE-Q were developed and validated with female samples, and therefore reflect female body ideals and ED behaviors reported by women. Thus, the assessment of ED psychopathology in the present study may have failed to capture male-specific dimensions of ED psychopathology and body dissatisfaction (Cafri & Thompson, 2004; Stanford & Lemberg, 2012; Strother, Lemberg, Stanford, & Turberville, 2012). Our sample was also limited to individuals ages 16 and older, which precludes generalizability to younger ages. Furthermore, participants were patients who were treated in a residential eating disorder program, and thus it is unclear whether such effects would be similar in less intensive treatment settings. Lastly, diagnoses were not based on structured interviews, and the treatment implemented in the present study was not standardized, and therefore we cannot make conclusions regarding the possible influence of treatment components on outcomes.

Conclusions

Despite the aforementioned limitations, the present study provides valuable information regarding the relationship between affective variables, particularly social anxiety, and ED psychopathology in a treatment context. Depression and anxiety appear to be markers of greater ED severity, and social anxiety is specifically related to poorer outcomes, which supports the possibility that there are magnified deficits in emotion regulation in the presence of these comorbidities. Thus, the assessment of these constructs at the outset of treatment may enable clinicians to identify and target these domains more effectively. Furthermore, while findings are generally consistent with previous research regarding social anxiety and EDs, there remains a dearth of literature assessing this relationship, especially in treatment-seeking samples. Further study in this area could lead to future treatment developments that improve the effectiveness of ED interventions.

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Table 1

Predictors of EDE-Q Global at Baseline with Bias Corrected Confidence Intervals

	B	β	95% BC CI	R	R ²	F	p
Step 1				.30	.09	13.73	<.001
Age	.01	.07	[-.01, .03]				
Gender	1.01*	.28	[.66, 1.36]				
BMI	.02*	.10	[.01, .04]				
Step 2				.69	.48	78.45	<.001
Age	.002	.01	[-.01, .01]				
Gender	.57*	.16	[.30, .85]				
BMI	.00	.00	[-.02, .02]				
General Anxiety	.03*	.23	[.01, .04]				
Depressive Symptoms	1.58*	.46	[1.12, 2.03]				
Step 3				.69	.48	65.79	<.001
Age	.001	.01	[-.01, .01]				
Gender	.55*	.15	[.27, .83]				
BMI	-.001	-.003	[-.02, .02]				
General Anxiety	.03*	.21	[.01, .04]				
Depressive Symptoms	1.52*	.44	[1.06, 1.97]				
Social Anxiety	.12	.06	[-.06, .31]				

Note.

* $p < .05$. BC CI = bias corrected confidence intervals.

Table 2
Predictors of EDE-Q Global at Discharge with Bias Corrected Confidence Intervals

	B	β	95% BC CI	R	R ²	F	p
Step 1				.65	.42	60.91	<.001
Age	-.01	-.07	[-.02, .002]				
Gender	.63*	.19	[.40, .88]				
BMI	.01	.04	[-.01, .03]				
Length of Stay	.001	.01	[-.003, .004]				
Baseline EDE-Q Global	.53*	.57	[.46, .59]				
Step 2				.66	.44	46.93	<.001
Age	-.01*	-.09	[-.02, -.001]				
Gender	.59*	.18	[.35, .84]				
BMI	.01	.04	[-.01, .02]				
Length of Stay	-.00	-.002	[-.004, .003]				
Baseline EDE-Q Global	.45*	.49	[.36, .55]				
General Anxiety	.02*	.21	[.01, .04]				
Depressive Symptoms	-.22	-.07	[-.66, .22]				
Step 3				.69	.48	48.21	<.001
Age	-.01*	-.10	[-.02, -.002]				
Gender	.51*	.16	[.29, .73]				
BMI	.004	.02	[-.01, .02]				
Length of Stay	-.001	-.01	[-.004, .003]				
Baseline EDE-Q Global	.44*	.47	[.34, .53]				
General Anxiety	.02*	.14	[.003, .03]				
Depressive Symptoms	-.43	-.14	[-.88, .01]				
Social Anxiety	.50*	.26	[.32, .67]				

Note.

* $p < .05$. BC CI = bias corrected confidence intervals.