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Mood regulation and quality of life in social anxiety disorder: An examination of generalized expectancies for negative mood regulation

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Abstract

The present study examined negative mood regulation expectancies, anxiety symptom severity, and quality of life in a sample of 167 patients with social anxiety disorder (SAD) and 165 healthy controls with no DSM-IV Axis I disorders. Participants completed the Generalized Expectancies for Negative Mood Regulation Scale (NMR), the Beck Anxiety Inventory, and the Quality of Life Enjoyment and Satisfaction Questionnaire. SAD symptom severity was assessed using the Liebowitz Social Anxiety Scale. Individuals with SAD scored significantly lower than controls on the NMR. Among SAD participants, NMR scores were negatively correlated with anxiety symptoms and SAD severity, and positively correlated with quality of life. NMR expectancies positively predicted quality of life even after controlling for demographic variables, comorbid diagnoses, anxiety symptoms, and SAD severity. Individuals with SAD may be less likely to engage in emotion regulating strategies due to negative beliefs regarding their effectiveness, thereby contributing to poorer quality of life.

Keywords

Social anxiety disorder; Social phobia; Emotion regulation; Generalized expectancies for mood; regulation

1. Introduction

Social anxiety disorder (SAD) is an early-onset, chronic psychiatric condition characterized by intense fear and avoidance of social situations. Recent theories have emphasized a role for deficits in emotion regulation among patients with anxiety disorders, including SAD

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(Aldao, Nolen-Hoeksema, & Schweizer, 2010; Cisler, Olatunji, Feldner, & Forsyth, 2010; Mennin, Heimberg, Turk, & Fresco, 2005; Mennin, Holaway, Fresco, Moore, & Heimberg, 2007), but relatively few studies have examined this issue in clinical samples of patients meeting DSM-IV criteria for the disorder. This literature gap is noteworthy, given that SAD is one of the most common mental health conditions (Ruscio et al., 2008) and is associated with significant disability and quality of life impairment (Barrera & Norton, 2009; Olatunji, Cisler, & Tolin, 2007; Simon et al., 2002; Stein & Kean, 2000; Wittchen, Fuetsch, Sonntag, Muller, & Liebowitz, 2000).

1.1. Emotion regulation and SAD

Broadly defined, emotion regulation is a multidimensional construct that encompasses a number of cognitive and behavioral strategies for recognizing and responding to emotional distress (Gratz & Tull, 2010). Emotion regulatory processes can be automatic or voluntary and may take place in or out of conscious awareness (Gross & Thompson, 2007). Effective emotion regulation involves the ability to identify various emotional states and to flexibly employ appropriate coping strategies for the situational context, whereas emotion dysregulation involves the application of strategies that are inappropriate, rigid, and/or ineffective for managing distress in a given situation (Cisler et al., 2010; Gratz & Roemer, 2004; Gross & Thompson, 2007).

Mennin's (2005) emotion dysregulation model posits that patients with anxiety disorders exhibit (1) heightened intensity of emotions, (2) poor understanding of emotions and difficulty differentiating between them, (3) negative reactivity to their own emotional states, and (4) maladaptive management of emotions. Data supporting this theory have focused primarily on individuals with generalized anxiety disorder (McLaughlin, Mennin, & Farach, 2007; Mennin et al., 2005; Roemer et al., 2009), but preliminary studies suggest that social anxiety is associated with similar difficulties in emotion regulatory processes (Mennin, McLaughlin, & Flanagan, 2009). For example, Turk et al. (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005) found that undergraduates with higher levels of social anxiety symptoms had greater difficulty identifying, describing, and managing emotions when compared to non-anxious controls. There was also some evidence for emotion regulation deficits that were specific to social anxiety. Relative to individuals with generalized anxiety and healthy controls, socially anxious individuals attended less to their emotions, had greater difficulty describing their emotions, were less expressive of positive emotions, and trended towards being less expressive of negative emotions. These results suggest that individuals with SAD are less likely to employ socially-oriented emotion regulation strategies (e.g., describing and revealing one's emotions to others) than those without SAD. However, this study used an analogue measure of social anxiety; whether these results hold for clinical samples remains unclear.

A small body of experimental research supports the notion that SAD is associated with distinctive patterns of emotion dysregulation, and that these are specific to situations that represent potential social threat. In a recent study comparing emotional reactivity to potential threat among individuals meeting DSM-IV criteria for SAD and healthy controls, Goldin et al. (Goldin, Manber, Hakimi, Canli, & Gross, 2009) found that those with SAD

experienced higher levels of initial negative emotional reactivity and they were less effective at using cognitive–linguistic strategies for emotion regulation in response to social, but not physical, threat. These data suggest that although patients with SAD are able to employ strategies for general emotion regulation, they are less able to use similar strategies when confronted with situations that are interpreted as socially threatening. It follows then that patients with SAD may need to learn skills that enhance the implementation and effectiveness of emotion regulation strategies.

1.2. Negative mood regulation expectancies

Information processing biases, negative self-beliefs, and behavioral avoidance of anxiety-provoking social situations have been well documented in patients with SAD (e.g., Clark & McManus, 2002; Clark & Wells, 1995; Heinrichs & Hofmann, 2001; Hofmann, 2007), but other factors that may impact the successful use of emotion regulation strategies have been relatively under-studied. One construct that may be particularly relevant to understanding barriers to effective emotion regulation in SAD is that of negative mood regulation (NMR) expectancies. NMR expectancies have been defined as beliefs regarding one's ability to alleviate negative mood states using a variety of emotion regulation strategies (Catanzaro & Mearns, 1990, 1999). According to theories of social learning and affective self-regulation, expectations that a particular behavioral or cognitive strategy will yield a desired outcome are important determinants of both affect and behavior (Catanzaro, 1994; Catanzaro & Greenwood, 1994; Catanzaro & Mearns, 1990). As such, NMR expectancies can function as a sort of self-fulfilling prophecy in which beliefs regarding the effectiveness of a given strategy impact the degree to which it is implemented successfully (Catanzaro & Mearns, 1990).

Data from undergraduate samples suggest that individuals holding low NMR expectancies are at greater risk for emotion dysregulation. The majority of these studies have focused on the relationship of NMR expectancies to dysphoria and/or depressive symptoms, but a small number have examined anxiety symptoms. In a study of 218 college students undergoing a stressful examination, Catanzaro (1996) found that those with low NMR expectancies showed greater state anxiety and poorer exam performance relative to those with higher NMR expectancies. Mood regulation expectancies also appear to independently contribute prospectively to change in anxiety symptoms. In a more recent study of 322 college students, Kassel et al. (Kassel, Bornovalova, & Mehta, 2007) found that lower NMR expectancies were predictive of increased anxiety symptoms over an 8-week period, even after controlling for demographic characteristics, baseline anxiety, and coping styles. Moreover, in a sample of depressed outpatients, NMR expectancies were linked with the number of comorbid mental disorders, supporting the notion that these expectancies may be involved in the etiology or maintenance of disorders (Pfeiffer, Kaemmerer, Mearns, Catanzaro, & Backenstrass, 2011).

1.3. SAD and quality of life

It has been well documented that individuals with social anxiety disorder report significantly lower quality of life compared to healthy controls (Ghaedi, Tavoli, Bakhtiari, Melyani, & Sahragard, 2010; Heiser, Turner, Beidel, & Roberson-Nay, 2009; Simon et al., 2002;

Watson, Swan, & Nathan, 2010). SAD symptom severity, depressive symptoms and psychiatric comorbidity (Watson et al., 2010) have all been shown to be negatively associated with quality of life in this population (Eng, Coles, Heimberg, & Safren, 2005; Lochner et al., 2003; Watson et al., 2010). A recent study by Kashdan et al. (Kashdan, Morina, & Priebe, 2009) suggests that certain aspects of emotion dysregulation, specifically experiential avoidance, may also contribute to poor quality of life among individuals with SAD. Experiential avoidance is defined as the tendency to react negatively to unwanted thoughts and feelings, and the desire to control or avoid these internal experiences and their accompanying distress (Kashdan et al., 2009). The authors found that among a sample of Albanian civilian survivors of the Kosovo War, experiential avoidance partially mediated the effects of SAD and PTSD, but not MDD, on quality of life, thus providing preliminary support for a relationship between social anxiety, emotion dysregulation, and quality of life. However, further study of the relationship between these variables is warranted.

1.4. The present study

These preliminary studies suggest a role for NMR expectancies as a contributor to emotion regulation difficulties in SAD, but research in this area has thus far been limited to undergraduate samples with broadly-defined anxiety symptoms. The extent to which outpatients with SAD show decrements in specific types of NMR expectancies (e.g., cognitive, behavioral, social) relative to healthy controls has yet to be examined. The relationship between NMR expectancies, anxiety symptom severity, and quality of life among patients with SAD has similarly received little attention. In order to address these gaps in the literature, the present study examined different types of NMR expectancies as assessed by subscales on the Generalized Expectancy for Negative Mood Regulation Scale (Catanzaro & Mearns, 1990) in a sample of patients with a primary DSM-IV diagnosis of SAD and healthy controls with no current DSM-IV Axis I disorders. We hypothesized that: (1) participants with SAD would be less likely than controls to believe in the effectiveness of cognitive, behavioral, and general strategies to alleviate negative mood states, (2) among those with SAD, lower NMR expectancies would be associated with greater severity of social and general anxiety symptoms and with poorer quality of life, and (3) lower NMR expectancies would uniquely predict poorer quality of life among SAD patients after controlling for demographic characteristics, comorbid psychiatric conditions, and severity of social and general anxiety symptoms.

2. Material and methods

2.1. Participants

The sample consisted of 167 patients with a primary diagnosis of SAD, as well as 165 non-anxious controls with no current DSM-IV Axis I disorders. Excluded conditions included serious acute suicidal risk, lifetime history of psychosis, and alcohol or substance abuse or dependence in the past 6 months. Participants were recruited via advertisement or clinical referral and consented to participate in a questionnaire-based protocol. For patients, this protocol was ancillary to a psychopharmacology, psychotherapy, or combined treatment protocol for SAD at the Center for Anxiety and Traumatic Stress Disorders at Massachusetts General Hospital.

2.2. Measures

The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) is a 21-item, self-report measure of common anxiety symptoms. Participants rate how bothersome each symptom has been in the past week on a Likert scale from 0 (“not at all”) to 3 (“severely – I could barely stand it”). The BAI has demonstrated high discriminant validity, internal consistency ($\alpha = .92$), and test-retest reliability ($r = .75$; Beck et al., 1988). Internal consistency was high in the current sample ($\alpha = .93$). Skewness and kurtosis values were acceptable (Skewness = 1.17, Kurtosis = $-.12$).

The Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) is a 24-item, clinician-rated measure which separately assesses fear and avoidance in a variety of social and performance situations. In each situation, fear and avoidance are rated on a 4-point Likert scale (0 = “none” or “never” to 3 = “severe” or “usually”). Total scores range from 0 to 144, with higher scores representing higher levels of symptom severity. The internal consistency was high in the current sample ($\alpha = .90$). Skewness and kurtosis were acceptable (Skewness = .44, Kurtosis = $-.37$).

The Generalized Expectancy for Negative Mood Regulation Scale (NMR; Catanzaro & Mearns, 1990) is a 30-item, self-rated scale designed to measure the strength of beliefs in one’s ability to alter negative mood states using a variety of emotion regulation strategies. Statements about one’s beliefs when upset are rated on a 5-point Likert scale (1 = “strongly disagree” to 5 = “strongly agree”). The scale yields total scores ranging from 30 to 150, with higher scores representing greater belief in one’s ability to alleviate a negative mood. The NMR is divided into a *cognitive* subscale, with 10 items reflecting cognitive strategies for negative mood regulation (e.g., “I’ll feel ok if I think about more pleasant times”), a *behavioral-alone* subscale, with 4 items reflecting mood-regulating behaviors that are done alone (e.g., “I can feel better by treating myself to something I like”), and a *behavioral-social* subscale, with 6 items reflecting mood-regulating behaviors that are done with others (e.g., “Going out to dinner with friends will help”), and a *general* subscale, with 10 items reflecting mood-regulating strategies that do not fall specifically into cognitive or behavioral domains (e.g., “I can find a way to relax”). The overall scale has high internal consistency ($\alpha = .86-.92$, depending on sample), test-retest reliability ($r = .67-.78$, depending on sample), and discriminant validity from social desirability (Catanzaro & Mearns, 1990). Several studies have also demonstrated its discriminant validity from measures of related concepts such as depression, anxiety, locus of control, and coping (Catanzaro, 1993; Catanzaro & Mearns, 1990; Kirsch, Mearns, & Catanzaro, 1990; Mearns, 1991). In the current sample, internal consistency was excellent for the overall scale ($\alpha = .93$) and good for the general ($\alpha = .84$), cognitive ($\alpha = .82$), and behavioral-social ($\alpha = .86$) subscales. However, internal consistency was low for the behavioral-alone subscale ($\alpha = .43$). Skewness and kurtosis were acceptable (Skewness = $-.55$ to $-.09$, Kurtosis = $-.93$ to $.00$).

The Quality of Life Enjoyment and Satisfaction Questionnaire (QLESQ; Endicott, Nee, Harrison, & Blumenthal, 1993) is a self-rated measure which asks participants to rate 16 aspects of quality of life, including physical health, mood, and social relationships, on a scale from 1 (“very poor”) to 5 (“very good”). Scaled scores range from 0 to 100, with

higher scores representing higher perceived quality of life. The QLESQ has been found to have high internal consistency ($\alpha = .88$; Mick, Faraone, Spencer, Zhang, & Biederman, 2008). Internal consistency was good in the current sample ($\alpha = .86$). Skewness and kurtosis were acceptable (Skewness = $-.36$, Kurtosis = $-.32$).

2.3. Procedures

All participants first signed informed consent, and were then assessed by structured clinical interview (either the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995) or the Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) to determine the presence of primary SAD in patients and the absence of current psychiatric disorders in controls, as well as to screen for comorbid and excluded psychiatric disorders. The LSAS was administered to all patients to assess SAD symptom severity, and all participants completed a packet of questionnaires, which included a demographics questionnaire as well as the NMR, BAI, and QLESQ. Clinician-rated assessments were administered by trained psychologists and psychiatrists. The institutional review board at Massachusetts General Hospital approved all study procedures.

2.4. Statistical analyses

One-way repeated measures ANOVA was used to compare differences in mean NMR subscale scores for SAD participants relative to healthy controls. Post hoc Bonferroni-corrected pairwise comparisons were computed to follow up significant main and interaction effects. Bivariate correlational analyses were then calculated to explore associations between NMR subscale scores (general, cognitive, behavioral-social, behavioral-alone), anxiety symptom severity (BAI total score), social phobia severity (LSAS total score), and quality of life (QLESQ total score) in the SAD group. Hierarchical linear regression analysis was then used to examine the unique contribution of generalized expectancies for negative mood regulation (NMR total score) in predicting quality of life (QLESQ total score) among patients with SAD after controlling for demographic characteristics, current psychiatric comorbidity, anxiety symptoms (BAI total score), and social phobia severity (LSAS total score). Age, gender, and race were entered in the first step, followed by current comorbidity, BAI, and LSAS total scores in the second step, and NMR total score in the third step. Finally, the complete model was examined to assess the extent to which NMR expectancies explained significant variance in quality of life while controlling for the other predictors.

3. Results

3.1. Sample description

Sample demographics and rates of psychiatric comorbidity are presented in Table 1. Mean age for participants in the SAD group was slightly lower than for those in the control group (34.5 years vs. 40.2 years). The SAD group also included a greater proportion of men (66.5% vs. 46.1%) and Caucasians (82.0% vs. 46.1%). Participants with SAD reported a mean duration of SAD symptomology of 12.2 years ($SD = 7.2$) and levels of psychiatric comorbidity were high. A higher proportion of participants completing the SCID self-identified as Hispanic (9.2% vs. 2.6% of those completing the MINI), but there were no

other significant differences in demographics, comorbidity, or questionnaire measures based on the type of clinical interview conducted (see Supplemental Table 1).

Supplementary material related to this article found, in the online version, at doi:10.1016/j.janxdis.2012.01.004.

3.2. SAD vs. control NMR comparisons

Table 2 presents mean NMR scores for the total measure, as well as for the four NMR subscales. One-way ANOVA revealed main effects for diagnosis ($F(1, 330) = 156.66, p < .001$, partial $\eta^2 = .322$) and subscale (Wilkes $\lambda = .47, F(3, 328) = 123.07, p < .001$, partial $\eta^2 = .53$), as well as a diagnosis by subscale interaction (Wilkes $\lambda = .83, F(3, 328) = 23.24, p < .001$, partial $\eta^2 = .18$). Individuals with SAD were less likely to believe that their attempts to alter negative mood states would be effective, as demonstrated by significantly lower total NMR scores in this group (see Table 2). They also scored significantly lower than controls on each of the NMR subscales, indicating lower expectancies for negative mood regulation across all four domains. A small effect size was found for the behavioral-alone subscale, whereas large effect sizes were found for the behavioral-social, cognitive, and general subscales.

3.3. Associations between NMR, general anxiety, social phobia severity, and quality of life

Table 3 presents zero-order correlations between the NMR, BAI, LSAS, and QLESQ for participants with SAD. Results indicated significant positive correlations between each of the NMR subscales ($r = .32-.78$). The behavioral-social subscale showed a moderate positive correlation with the QLESQ, whereas the behavioral-alone subscale was not significantly associated with any of the anxiety or quality of life measures. NMR total scores as well as the cognitive and general subscale scores were negatively correlated with BAI and LSAS scores ($r = -.19$ to $-.24$) and positively correlated with QLESQ scores ($r = .32-.42$).

3.4. Hierarchical regression analysis

Table 4 presents summary statistics for the hierarchical regression model. Age, gender, and race entered at Step 1 accounted for approximately 6% of the variance in QLESQ scores ($F = 3.31, p < .05$), with race emerging as the only statistically significant predictor (see Table 4). When current comorbidity, BAI, and LSAS scores were added at Step 2, the proportion of variance accounted for increased to approximately 29% ($F = 10.96, p < .001$). Examination of individual parameter estimates at Step 2 indicated that current comorbidity, BAI, and LSAS were each significant negative predictors of QLESQ scores (see Table 4). When NMR total score was entered at Step 3, the proportion of variance accounted for increased to approximately 34% ($F = 11.83, p < .001$). Race, current comorbidity, and BAI remained significant predictors in the final model, whereas LSAS score was no longer a significant predictor of QLESQ when NMR scores were included in the model. NMR score was a significant positive predictor that uniquely accounted for approximately 5% of the variance, after controlling for demographic variables, comorbid diagnoses, BAI, and LSAS scores.

4. Discussion

Taken together, our results indicate that individuals with SAD are less likely than healthy controls to believe that behavioral and cognitive strategies will be effective in alleviating negative mood states, and this contributes uniquely to poorer quality of life among patients with SAD. As expected, individuals with SAD reported lower expectancies for negative mood regulation in cognitive, behavioral, and general domains. Although patients with SAD had lower NMR scores across the board, they were most similar to controls in their beliefs regarding mood regulation strategies that could be performed alone. These findings add to a growing body of literature showing that SAD is associated with specific deficits in emotion regulation (e.g., Goldin et al., 2009).

SAD participants' lack of confidence in socially-oriented relative to solitary emotion regulation strategies is consistent with the core features of the disorder (i.e., persistent fear and avoidance of social situations). Patients with SAD are often uncomfortable revealing their feelings to others for fear of embarrassment. They may therefore have very little experience relying on friends or family to help them manage emotional distress. In addition, longstanding patterns of avoidance may mean that patients with SAD do not trust themselves to implement socially-oriented emotion regulation strategies successfully. Furthermore, when individuals with SAD do attempt to enlist the help of others, they may do so less effectively or interpret the other person's response more negatively, thereby reinforcing the belief that social strategies are not as effective in alleviating negative mood states.

The present study also expands upon previous studies by examining the relationships between emotion regulation expectancies and the types of functional outcomes (i.e., quality of life impairments) that have been well documented in SAD. We found a moderate positive association between SAD patients' beliefs regarding their abilities to use socially-oriented emotion regulation strategies and their self-reported quality of life. A similar pattern of results was found for cognitive strategies and for more general emotion regulation strategies. Interestingly, beliefs regarding emotion-regulating behaviors performed alone were not significantly related to quality of life. Also noteworthy is the fact that higher emotion regulation expectancies were predictive of greater quality of life, even after controlling for anxiety symptom severity, sociodemographic factors, and psychiatric comorbidity. These findings are in line with Cisler et al.'s (2010) model of the long-term consequences of maladaptive emotion regulation among anxiety-disordered individuals. They propose that over time, chronic patterns of maladaptive emotion regulation (e.g., over-reliance on suppression and negative reappraisal) result in increased fear, which leads to avoidance and serves to maintain high levels of anxiety. Continued inflexible reliance on these emotion regulation strategies is hypothesized to lead to the sorts of impairments in school, work, and relationships that are typically seen among patients with SAD.

4.1. Clinical implications

Our results may also have some implications for treating patients with SAD. There is evidence that both cognitive behavioral therapy (CBT) and mindfulness based stress reduction result in acute improvements in symptom severity, emotion regulation, and quality

of life among patients with SAD (Eng et al., 2005; Goldin & Gross, 2010; Koszycki, Benger, Shlik, & Bradwejn, 2007; Suveg, Sood, Comer, & Kendall, 2009). However, a recent 12-month follow-up study of patients who completed a course of group CBT for SAD found that despite improvements in social anxiety symptoms, improvements in social functioning were modest and did not last beyond the acute treatment phase (Watanabe et al., 2010). Similarly, Eng et al. (Eng et al., 2005) found that patients who completed a course of group CBT for SAD showed some improvements, but failed to attain normative levels of satisfaction in the domains of achievement and social functioning.

These results suggest that while the aforementioned interventions provided training in CBT skills, they may not have sufficiently addressed patients' deeply held negative beliefs regarding the effectiveness of self-regulation strategies, thus precluding long-term improvements in social functioning. Patients holding negative expectations regarding skill use may be unlikely to employ adaptive emotion regulation strategies following completion of treatment, thereby limiting the scope of their treatment gains. Altering perceived deficits in emotion regulation strategies (particularly those that are socially-oriented) may therefore be an important consideration for those seeking to successfully treat SAD. In the context of CBT, this could be achieved by employing exposure and cognitive restructuring tactics specifically designed to address negative self-perceptions about emotion regulation. Additional interventions targeting distress intolerance may also be effective in promoting this type of cognitive change. Distress intolerance is itself linked to measures of mood regulation efficacy as well as multiple domains of dysfunctional behavior (Weitzman, McHugh, & Otto, 2011). Distress intolerance is also richly modifiable with focused treatment (Otto, 2008; Smits, Berry, Tart, & Powers, 2008).

4.2. Strengths and limitations

Strengths of this study are recruitment of a relatively large sample and the use of structured diagnostic interviews to determine presence of Axis I disorders. Limitations include the use of self-report measures to assess NMR expectancies, anxiety symptoms, and quality of life. By relying on a self-report measure of NMR expectancies, rather than direct observations of emotion regulating behaviors, we are limited to examining participants' beliefs regarding the effectiveness of various emotion regulation strategies, rather than their actual use of such strategies when faced with emotional distress. Additionally, the overall sample was primarily Caucasian and nearly two-thirds of those in the SAD group were men. Therefore, our results may not generalize to the entire population of patients with SAD. Finally, the cross sectional nature of our study limits the extent to which causal conclusions can be made. Nevertheless, this study contributes to a growing body of research showing that individuals with SAD show unique patterns of perceived emotion dysregulation that contribute to quality of life impairments.

5. Conclusions

We found lower self-reported mood regulation expectancies among patients with SAD relative to healthy controls. NMR expectancies were unique predictors of quality of life even after controlling for demographic and clinical covariates. These results suggest that individuals with SAD may be less likely to engage in helpful emotion regulating strategies

due to negative beliefs regarding their effectiveness, thereby contributing to poorer quality of life in this population. Interventions that increase expectancies for emotion regulation may be warranted.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Sample demographics and comorbidity for SAD ($n = 167$) vs. control ($n = 165$) groups.

Demographic characteristic	Group		Test statistic		Effect size	
	SAD	Control	df	t/χ^2	r/ϕ	r/ϕ
Age (years), mean (SD) ^c	34.5 (12.7)	40.2 (13.5)	330	$t = 3.90$	$r = .21$	$r = .21$
Gender, % (n) ^c			1	$\chi^2 = 14.05$	$\phi = -.21$	$\phi = -.21$
Female	33.5 (56)	53.9 (89)				
Male	66.5 (111)	46.1 (76)				
Ethnicity, % (n)			1	$\chi^2 = 2.38$	$\phi = -.09$	$\phi = -.09$
Hispanic or Latino	4.8 (8)	9.1 (15)				
Not Hispanic or Latino	95.2 (159)	90.9 (150)				
Race, % (n) ^a			4	$\chi^2 = 10.35$	$\phi = .17$	$\phi = .17$
Asian	6.6 (11)	4.8 (8)				
Black or African American	5.4 (9)	13.9 (23)				
Caucasian	82.0 (137)	72.1 (119)				
Native American or Alaska Native	0.0 (0)	0.0 (0)				
Native Hawaiian or Pacific Islander	0.6 (1)	0.0 (0)				
Other	5.4 (9)	9.1 (15)				
Diagnosis, % (n)						
At least 1 comorbid disorder	29.3 (49)	N/A	-	-	-	-
Generalized anxiety disorder	16.3 (27)	N/A	-	-	-	-
Posttraumatic stress disorder	1.8 (3)	N/A	-	-	-	-
Panic disorder	5.4 (9)	N/A	-	-	-	-
Agoraphobia	3.0 (5)	N/A	-	-	-	-
Obsessive compulsive disorder	1.2 (2)	N/A	-	-	-	-
Major depressive disorder	18.0 (30)	N/A	-	-	-	-
Bipolar disorder	0.0 (0)	N/A	-	-	-	-

Note. N/A: not applicable, inclusion criterion for control group was lack of Axis I comorbidity.

^a $p < .05$.

^c $p < .001$.

Table 2

NMR total and subscale scores for SAD ($n = 167$) vs. control ($n = 165$) groups.

	SAD		Control		Test statistic		Effect size	
	Mean	SD	Mean	SD	t	df	t	r
NMR total score ^c	92.89	17.42	117.70	14.33	330	330	14.16	0.61
NMR subscale scores								
Behavioral social ^c	14.31	4.65	20.36	6.12	330	330	10.16	0.49
Behavioral alone ^b	12.57	3.05	13.62	3.07	330	330	3.11	0.17
Cognitive ^c	30.34	6.67	38.06	5.45	330	330	11.55	0.54
General ^c	32.28	7.44	42.28	5.89	330	330	13.52	0.60

Note. NMR: Generalized Expectancy for Negative Mood Regulation Scale; SAD: social anxiety disorder.

^b $p < .01$.

^c $p < .001$.

Table 3

Zero-order correlations between emotion regulation, anxiety, and quality of life measures in SAD group ($n = 167$).

	BAI total score	LSAS total score	QLESQ total score
NMR total score	-.22 ^b	-.21 ^b	.40 ^b
NMR behavioral social	-.04	-.13	.30 ^b
NMR behavioral alone	-.13	-.03	.07
NMR cognitive	-.19 ^a	-.21 ^b	.32 ^b
NMR general	-.24 ^b	-.20 ^b	.42 ^b

Note. NMR: Generalized Expectancy for Negative Mood Regulation Scale; LSAS: Liebowitz Social Anxiety Scale; QLESQ: Quality of Life Enjoyment and Satisfaction Questionnaire.

^a $p < .05$.

^b $p < .01$.

Table 4
 Hierarchical regression estimating quality of life scores in participants with SAD ($N = 167$).

Variable	Model 1		Model 2		Model 3	
	<i>B</i>	<i>SE B</i>	<i>B</i>	<i>SE B</i>	<i>B</i>	<i>SE B</i>
Age	.11	.05	.15	.11 ^a	.15	.07
Gender	1.5	1.49	.08	1.31	.10	1.26
Race	3.82 ^d	1.82	.17	3.60 ^d	.16	3.95 ^a
Comorbidity	-	-	-	-5.64 ^c	1.35	-29
BAI total score	-	-	-	-.26 ^c	.07	-.26
LSAS total score	-	-	-	-.08 ^a	.04	-.16
NMR total score	-	-	-	-	-	.13 ^b
R^2	.06	.29	.34			
<i>F</i> for R^2 change	3.31 ^a	17.60 ^c	12.39 ^c			

Note. Gender was coded as 0,1 with male as the reference group; race was coded as 0,1 with Caucasian as the reference group; comorbidity was coded as 0, with no current comorbidity as the reference group. SAD: social anxiety disorder; BAI: Beck Anxiety Inventory; LSAS: Liebowitz Social Anxiety Scale; and NMR: Generalized Expectancy for Negative Mood Regulation Scale.

^a $p < .05$.

^b $p < .01$.

^c $p < .001$.